Racism in psychiatry

Professor Tyrer’s editorial (2005) is welcome and long overdue. He highlights a serious inequality between the contributions of authors from the industrialised and non-industrialised world. What Professor Tyrer failed to discuss is a more deeply imbedded problem of the institutionalised racism that lies at the heart of the conceptual systems we use in psychiatry. This is, of course, an understandably even harder and more painful issue for our profession to face; it is, however, necessary that we examine the potential for the concepts that we use to be inherently discriminatory.

For example, is the consistently higher rate of diagnosis of schizophrenia in second-generation British–Caribbean people a result of incorrect diagnosis, or the potential for a reductionist biomedical model of mental health to label whole communities as ‘mad’ with the resulting stigma these communities then suffer (as well as masking from us the impact of social issues such as immigration and racism)? Another example relates to the concept of depression, which is meaningless in some cultures. What impact does imposing a meaningless diagnosis have on someone’s willingness and motivation to engage with services? This obviously has the potential to discriminate in a subtle way against whole communities on their ability to develop meaningful relationships with their treating psychiatrists.

We should not be surprised that there are inherently racist concepts embedded in our ‘institutionalised’ ways of thinking about mental health problems, how to conceptualise them, what to do about them, and what value system we take into our daily practice. Mental health ideology and technology have developed not as the result of the discovery of testable physical pathology, but through a system of consensus resulting from powerful psychiatrists’ interpretation of the existing evidence.

These psychiatrists have carried their own cultural assumptions (derived from the dominant Western culture) without apparently being aware of this, and developed a system thinking and acting for psychiatrists based on these Western cultural ideals. Thus, from its conception, modern psychiatry has been imposing these Western, culturally constructed ideas on communities who have very different models for understanding mental health problems and what to do about them.

Sadly, I am not sure how ready our profession is to engage in some self-reflection and a thorough re-examination of these issues. I guess that may be just too painful; however, if we do not do this, we will not get rid of the scourge of institutional racism from our profession.


S. B. Timimi Lincolnshire Partnership NHS Trust, Ash Villa, Sleaford NG34 8QA, UK. E-mail: stimimi@talk21.com

Author’s reply: Dr Timimi raises the much larger issue of scientific racism in psychiatry in his letter. How much of this is institutional is difficult to determine but I contend that this is not the primary responsibility of journal editors to correct. The duty of an editor is to inform, to promulgate and explicate rather than to direct and legislate, and if this is done successfully it can help, together with many other influences, in changing minds and opinions. So we carry this out using the approach of Harriet Beecher Stowe rather than that of Abraham Lincoln and, if we change public opinion through the written word, we can also influence the climate of psychiatric practice favourably.

I hope that the Journal is helping to change opinion more in Dr Timimi’s direction in the spirit of my editorial (Tyrer, 2005). So we accept that our definitions of psychiatric illness are indeed too centred on the developed world and point out, for example, that the ICD–10 and DSM–IV diagnostic classification descriptions of anorexia nervosa are deficient in Ghana as those with the condition there ‘would not be classed as having anorexia nervosa, as they had neither a morbid fear of fatness nor a pervasive need to be slim. Rather, they reported a desire to exert self-control through deliberate self-starvation’ (Bennett et al, 2004). Similarly, in changing our attitudes towards British–Caribbean people who have schizophrenia, if we appreciate that stigma is likely to be a consequence of delayed presentation and compulsory admission (Morgan et al, 2003), then we are able to both give an explanation and possibly gain from the experience of other countries in getting services provided early to a stigmatised group (Chatterjee et al, 2003). I therefore do not share Dr Timimi’s pessimism; by opening up the debate we have moved from ‘powerful psychiatrists interpreting the existing evidence’ in their favour, to powerful evidence from around the world influencing the responses of all psychiatrists, irrespective of their status. Long may this process continue.


Peter Tyrer Editor. British Journal of Psychiatry. 17 Belgrave Square, London SW1X 8JP, UK. E-mail: bjp@rcpsych.ac.uk

Phenomenology of psychosis

I read with interest the title of the editorial by Harland by Harland (2004), which promised a fascinating synthesis of phenomenology, anthropology and the psychology of the self to formulate a new model for the aetiology of psychosis. Sadly, this was not achieved by the authors and I was left wondering how this had been lost on the Editor.
My response to the authors’ striking claim that they propose a new model (even if only ‘an outline sketch of a potential model’) is: what model? Unfortunately, careful reading of the paper in search of this model resulted in little that is new and certainly nothing approaching a testable hypothesis.

Is it a new model to state that the environment affects the configuration of the self and that this can somehow lead to psychosis? The authors cite the example of migration and its association with increased risk of psychosis to illustrate their point, but it does not seem that they have anything new to say about this fascinating area.

The authors call for greater rigour in future conceptual models that integrate the biological and the social in the aetiology of psychosis. It is, therefore, doubly surprising that they advocate the integration of hermeneutically oriented social sciences into such future models. Given that hermeneutics is disconnected from the rigours of the laws of causation that govern the material world, one wonders how this would reduce the ‘vagueness’ that the authors warn us against. It is also of interest that the two other concepts central to the authors’ model are the ‘self’ – a concept that has numerous competing definitions (one article cited 21 competing concepts of the self; see Zahavi, 2003) – and ‘social capital’, which has no operational definition (see McKenzie et al., 2002). It seems to me that the authors should have followed their own admonition against vagueness or else produced their own clear definitions of these concepts.

Also, the authors commit an elementary error by confusing the concept of ‘biological’ with ‘genetic’ or ‘genomic’ in their critique of current theories on schizophrenia, citing the work of Eisenberg (2004). When we consider the role of a given environmental factor in shaping a particular trait, we are most certainly dealing with a biological process. Can we discuss the effect of sunlight on tanning of the skin without considering melanocytes and melanin (see Gaulin & McBurney, 2001)? Similarly, if the human brain/mind has the propensity, under certain environmental conditions and given a particular genetic make-up and early-life experience, to produce the clinical picture we call psychosis, this cannot be understood outside of biology. Phenotypes, we should remember, are not simply the obligate expression of genes but the complex outcome of the interaction of the genome with the environment. In other words, the identification of an environmental risk factor for a particular disorder is not the end of the story. To achieve a real understanding of how the phenotypic trait was shaped, we still need to understand the intra-organismic process that led to the said trait.


R. Abed
Rotherham District General Hospital, Moorgate Road, Rotherham S60 2UD, UK. E-mail: abedr1@btinternet.com

Authors’ reply: In suggesting that the environment acts to produce biologically based phenotypes depending on genetic propensity, through ‘intra-organismic’ processes, this letter points to one mantra of modern psychiatry. But in looking for how our editorial contributes to the incremental nature of this important science it misunderstands our intent.

By ‘a new model’ we are describing an alternative way of seeing the problem, as opposed to the box and line model we have become more familiar with when considering a novel testable hypothesis. Phenomenology, conceived by Husserl, developed through Heidegger, Ricoeur and others, is notoriously difficult. On these terms there may be those who feel that what we have attempted is misconceived. However, it is a tradition that influenced many of the early scientific thinkers in our field, including Jaspers, Schneider, Minkowski and Lewis, and it continues to influence today (e.g. Cutting, 1997; Sass, 2004; Stanghellini, 2004).

To precis Dan Zahavi (2003: p. 59) we cannot ask what it is like to be a bar of soap or a rock. However, we can ask what it is like to be a mouse, a human or, indeed, to experience schizophrenia. This ‘what it is like’ has an internal structure that phenomenology attempts to capture. Likewise, in our editorial we suggest that migration can provoke changes in ‘what it is like to be’ on a similar level and that the field of anthropology (which draws on phenomenology) offers insights here. We then link these changes to the increased rates of psychotic illness in some groups and suggest that this fits well with the current psychiatric thesis that the brain is the product of its own historical trajectory.

We remain open to whether biological or environmental correlates with identified phenomenological structures can meaningfully be found. But a reinvigoration of phenomenology is perhaps just what psychiatry needs at this time. We would do well to bring to mind that despite our best efforts we have yet to find aetiological factors in environmental or biological terms that take us beyond the group effect to the individual.

Above all the purpose of writing the editorial was to stimulate debate.


R. Harland, C. Morgan
Division of Psychological Medicine, Institute of Psychiatry, London, UK. E-mail: r.harland@kcl.ac.uk

G. Hutchinson
The University of the West Indies, Department of Psychiatry, Mount Hope, Trinidad

Personality and attachment in adolescence

It was encouraging to see the study by Western et al. (2005) published in a mainstream journal such as the British Journal of Psychiatry. For a variety of reasons, there is a reluctance among many British adolescent mental health clinicians to diagnose personality disorders in their patients, despite the clear presence often of the requisite diagnostic features. This study shows that personality disorders in adolescents can be validly diagnosed, whether using an established framework such as the DSM–IV or a new, empirically derived taxonomy.
I was struck by the extent to which certain factors derived from the Q-factor analysis appear to map onto current conceptualisation of attachment categories, a point borne out partially by the same research group (Nakash-Eisikovits et al, 2002) using Bartholomew’s attachment classification (Bartholomew & Horowitz, 1991). Specifically, the ‘psychological health’ factor shows strong correspondence to features of a secure internal working model, while the ‘histrionic sexualisation’ and ‘emotional dysregulation’ factors contain items integral to the conceptualisation of ambivalent/preoccupied attachment. It is interesting that in their 2002 study, the group found that attachment avoidance was correlated with their ‘avoidant’ Q-factor but not with DSM-IV avoidant personality disorder; on this basis, they questioned the prevailing conceptualisation of avoidant personality disorder. It is unclear how attachment disorganisation is related to the SWAP-200-A factors, as it is still uncertain to what extent to which Bartholomew’s ‘fearful’ category corresponds to disorganised/unresolved attachment.

Therefore, it is perhaps logical to hypothesise that some personality traits constellations (the most maladaptive of which may constitute personality disorders) are indeed disorders of attachment. This hypothesis, which is supported theoretically (Nakash-Eisikovits et al, 2002) and which makes intuitive sense to many adolescent mental health professionals, needs to be tested with longitudinal research. In addition to other empirical work, the above research shows the continuing clinical importance of attachment theory. However, there is still no easily administered validated measure of adolescent attachment in widespread clinical use currently in the UK. Surely, this is a deficit that needs to be addressed.

**Dementia prevalence**

Shaji et al (2005) have estimated the prevalence of dementia in an urban population in Kerala, India and have provided a glimpse into the various factors associated with dementia in their study. A few methodological issues of the study need further clarification.

Although a cut-off score of 23 on the Mini-Mental State Examination (MMSE) was used for all the participants, a different cut-off score would have been appropriate among those who were illiterate (11.2%) as educational status has been shown to affect MMSE scores.

With no objective evidence to suggest hypertension in the participants other than the verbal account of the caregivers, the very high odds ratio for hypertension is misleading. Furthermore, with such a small number of individuals, the selection of the controls should have been more stringent. It would be advisable to take a larger number of controls for such a small sample of individuals with vascular dementia (n=22).

Although age has been shown to be a risk factor for dementia in many studies, how this conclusion was reached in this study is not clear.

There is a discrepancy in the number of patients reported as receiving treatment for Alzheimer’s dementia (21 out of 31) compared with the total number of individuals with Alzheimer’s dementia detected in the study (n=30).

Despite a few limitations, this study adds to the growing literature of the epidemiology of dementia in developing countries and would be helpful for healthcare planners for adequate resource allocation for preventive and curative services.

**Involuntary placement in Italy**

Salize & Dressing (2004) show figures for various indicators for involuntary placements in psychiatric facilities in the European Union countries. According to the authors, Italy was unable to provide nationwide data from the 1990s. Therefore, in Table 1 they showed data from the Lombardy region, concerning only the rate of in-patient episodes that were involuntary placements for an ‘unknown year’. Data from Lombardy on number and rate of involuntary placements per 100,000 population are indicated as ‘not available’.

Actually, nationwide data have been published up to 1997, and data from the Lombardy region are available for the period 1995–2001 (see Table). The Italian data can be found on the website of the National Institute of Statistics (http://www.istat.it/Societa-Sanita-e-P/Storico) and those of Lombardy on the website of the Regional Directorate of Health (http://www.sanita.regione.lombardia.it/documenti).

Also, Salize & Dressing report a rate of 11 involuntary placements per 100,000 for France. This is not correct, as can be seen looking at absolute numbers of 61,063 involuntary admissions in France. Given the French population of about 59.3

---

**Table: Involuntary placement in Italy**

<table>
<thead>
<tr>
<th>Year</th>
<th>Lombardy</th>
<th>Italy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>Percentage of all in-patient episodes</td>
</tr>
<tr>
<td>1996</td>
<td>2832</td>
<td>12.3</td>
</tr>
<tr>
<td>1997</td>
<td>2818</td>
<td>11.6</td>
</tr>
<tr>
<td>1998</td>
<td>2803</td>
<td>12.1</td>
</tr>
<tr>
<td>1999</td>
<td>2792</td>
<td>12.6</td>
</tr>
<tr>
<td>2000</td>
<td>2794</td>
<td>11.8</td>
</tr>
<tr>
<td>2001</td>
<td>2487</td>
<td>10.8</td>
</tr>
</tbody>
</table>

One hundred years ago

Pyromania, a psychosis of puberty

In the Archives de Neurologie for December, 1904, Dr. Raoul Leroy, assistant physician at the Evreux Asylum, refers to the medico-legal important subject of pyromania in young persons of both sexes, a form of mental disorder which leads to acts of incendiarism. "Whenever reported fires occur," he says, "in a village or in the country suspicion generally falls on persons of incomplete mental or physical development – weak-minded youths or girls – among the inhabitants and it generally proves to be well-founded." The mental state of such incendiaries, says Dr. Leroy, is peculiar and characteristic. They are weak-minded and are often members of families in which epilepsy, insanity, or alcoholism occurs. Reference is made to the fact that among the peasant population of Normandy, where alcoholism prevails to a high degree, juvenile crimes of the nature of incendiarism are common. These feeble-minded delinquents are prone to set fire to buildings or other objects in revenge against their owners or in some cases merely to amuse themselves with the spectacle. A few cases, says Dr. Leroy, suffer from the influence of an obsession which irresistibly impels them to such acts, such cases forming a special form of insanity to which the term "pyromania" is applied. True cases of pyromania manifest themselves for the first time at the period of puberty. The following typical case is given in illustration of this affection. The patient or culprit in this case was a girl, aged 15 years, a domestic servant, who on three separate occasions had set fire to the house of her master. She was the child of respectable parents and at first no suspicion was entertained of her but on being questioned before the police at the third outbreak of fire she showed much agitation and finally confessed her guilt. "When she had stated the facts fully and was asked if she realised the wickedness of her crime," she replied, "Something supernatural urged me to set the place on fire." Although she was reasoned with and her wrong-doing demonstrated in the clearest manner "she remained unshakable and invariably repeated the same words in justification." She had no reason to hate her master, there was no motive whatever of vengeance that impelled her, only a presumably morbid impulse. The medico-legal inquiry revealed the fact that a highly neurotic hereditary taint existed in the family; the grandfather was a man of excessively violent disposition, a first cousin was liable to periodic attacks of insanity during which he wandered about, the grandmother committed suicide at the age of 63 years, and the patient’s mother was a very nervous, emotional, weak-willed woman afflicted with coxalgia. The patient herself was a child of but little intelligence and never could read and write correctly. Her character was excitable, violent, and impulsive, with a total lack of good judgment. At the age...
of 13 years she developed nocturnal somnambulism, walking all over the house in her sleep and having no recollection of it in the morning. The menstrual periods, which set in at the age of 15 years, were attended with headache, insomnia, and great nervous prostration. On one such occasion she had a hallucination that her bed was surrounded by flames and this was the starting point of her obsessions. Almost daily after this she felt the sudden morbid impulse to set something on fire. It grew stronger though she struggled against it, suffering great mental and physical distress in the process. The morbid obsession occupied her mind to the exclusion of all other ideas and caused such distress and agony that she could resist no longer. Taking a lighted taper she set fire to a packet of waste paper. This was followed by instant relief of distress and an agreeable feeling of satisfaction. These obsessions occurred from time to time and on three such occasions she set fire to outbuildings and parts of her master’s house. The medical evidence taken before the magistrate in the trial of this patient showed the occurrence of insane impulses the patient was removed to the asylum. Dr. Leroy concludes that the morbid heredity on both paternal and maternal sides resulted in a brain liable to disorder and readily provoked to morbid impulse (in this case pyromania) on the occurrence of the stresses of puberty, a critical time in mental development.

REFERENCE


Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Epsom, Surrey

**Corrigendum**

Personality and attachment in adolescence
K. Ma
Access the most recent version at DOI: 10.1192/bjp.186.6.541

References
This article cites 3 articles, 1 of which you can access for free at:
http://bjp.rcpsych.org/content/186/6/541#BIBL

Reprints/permissions
To obtain reprints or permission to reproduce material from this paper, please write to permissions@rcpsych.ac.uk

You can respond to this article at
/letters/submit/bjprcpsych;186/6/541

Downloaded from
http://bjp.rcpsych.org/ on July 9, 2017
Published by The Royal College of Psychiatrists