

Correspondence

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Contents

- Salience dysregulation syndrome: a patient's view
- Immigration and borderline personality disorder
- Clozapine and risk of pneumonia
- Dual diagnosis quandries

Salience dysregulation syndrome: a patient's view

Jim van Os has done us a service in bringing to attention the unsatisfactory nature of the concept of schizophrenia.¹ He argues that the scientific evidence for the category is weak and that the present label is highly stigmatised. He suggests that a new concept – salience dysregulation syndrome – be assessed with regard to its clinical utility and patient acceptability. (Compare with Sato.²)

The term 'syndrome' is understandable as a constellation of symptoms rather than just one symptom. For example, I am susceptible to schizophrenia but have never heard voices and never hallucinated. That does not mean I cannot be diagnosed as having schizophrenia.³ My problem as a patient is that the terms 'salience' and 'dysregulation' are unfamiliar medical jargon.

If an alternative concept is to replace the construct 'schizophrenia', it needs to be acceptable to patients; that, van Os and I agree on. It needs to be understandable, neutral in tone, and without any misleading negative associations. Salience dysregulation syndrome meets the latter two criteria, but not the first. To me and other patients with whom I have discussed van Os's proposal, the suggested terminology is obscure.

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doi: 10.1192/bjp.194.5.467

Author's reply: I agree that the term 'salience' may appear obscure at first glance but let us analyse the issue in more detail. The term 'schizophrenia' is stigma-inducing because it confusingly and mystifyingly refers to a disease that is characterised by a 'split mind' – a psychological state that the public cannot personally relate to. This is different from, for example, depression, as virtually every member of the public knows that depression is about a negative emotional state that they themselves may also experience on a daily basis, albeit to a lesser degree. Say we were to call schizophrenia 'reality distortion syndrome' or 'integration dysregulation syndrome'. Although the meaning of the words would certainly be clear to the general public, the problem is that these names may paradoxically also result in stigma because the people cannot relate to a universal psychological function of 'reality' or 'integration'. How long will people talk to somebody

at a party who 'cannot see reality' or is 'not integrated'? In other words, I do not think that it is the degree of immediate and easy recognition that is important for a new name for schizophrenia, but (a) the potential of the new name to teach the general public about the experiences we call psychotic, based on (b) a scientifically valid model and (c) an aspect of psychological experience that everybody can relate to. The reality is that this is never going to be easy and cannot be solved by an appealing name alone. Salience is about how internal or external stimuli can become attention-grabbing and how this, if it is not willed, can lead to perplexing experiences that result in a search for an explanation that we subsequently call delusions. There may be some explaining to do, but maybe not an impossible message to convey.

In conclusion, I feel it is not so much important whether or not a new name is immediately clear to everybody, but whether it has got potential to make people recognise it as relating to an aspect of psychological experience that is universal. Salience may be a vehicle to teach the general public about the experiences we call psychotic. The second issue is that it may be important to move on from criticising the term schizophrenia to systematically proposing alternatives. The reason that the cogent scientific reasoning by people such as Herman van Praag,¹ Mary Boyle,² Richard Bentall³ and Ian Brockington,⁴ and many others did not have an impact on DSM–IV and ICD–10 may be because an alternative was never proposed. This is why I started with an alternative, not just a criticism of the term schizophrenia.

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doi: 10.1192/bjp.194.5.467a

Immigration and borderline personality disorder

The study by Pascual *et al*¹ is interesting and shows a lot of effort by the researchers, who reviewed thousands of cases despite the limitations of research methodology. However, I wonder what prompted the authors to think that immigration could be a risk factor for borderline personality disorder?

Unlike functional illnesses such as depression and schizophrenia, which can develop at any age and can have lots of predisposing factors, personality disorders develop during the early years of childhood and adolescence with most of the personality traits well established by adulthood.

Most of the immigrant groups in this study¹ are from low- and middle-income countries and it is not surprising that fewer people from this group were diagnosed with borderline personality disorder as compared with the indigenous population. We know that the prevalence of personality disorders is greater in high-income/Western countries.²

If we look at the features and diagnostic criteria for personality disorders, using either DSM–IV or ICD–10, we broadly see two main factors at the base of most of the symptoms: poor coping mechanisms and maladaptive behaviours. Factors commonly seen in Western/ high-income countries which contribute to such traits

and learned behaviours are the breakdown of community norms³ such as lack of family cohesion, lack of a social support network, dysfunctional families and child abuse. Also, in high-income countries as people enjoy more privileges, they tend to take less responsibility for their actions and expect more and more from the state. We increasingly see more pressure on social services, rather than on parents, to account for the welfare of children.

This does not mean that borderline personality disorder is exclusive to the West, but in the social context we do see more reasons for people in the West to have such traits.

Given the aetiological factors that we are aware of, and the crucial age factor for borderline personality disorder, it is no surprise that immigration is not a risk factor for borderline personality disorder.

This is an interesting study that confirms what was earlier suggested by Tyrer *et al*⁴ and Baleyrier *et al*,⁵ however, I am not sure whether a similar study in future would be useful, given that it is unlikely that immigration can be a risk factor for developing borderline personality disorder.

I do, however, agree with the authors that future studies in younger immigrants and second generations who will be more influenced by the Western way of life are likely to be interesting and helpful, especially in terms of clinical management.

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doi: 10.1192/bjp.194.5.467b

Authors' reply: We thank Dr Mushtaq for his comments on our article.¹ Although we agree with his comment that it is unlikely that immigration could be a risk factor for developing borderline personality disorder, we think that this issue is still open to debate.

First, other authors such as Paris² have suggested that the process of migration from traditional societies to Western countries could result in the development of borderline personality disorder in individuals who did not present any symptoms in their country of origin. Paris considered that although individuals could have a biological predisposition to this disorder, such as an innate affective instability, the structure of traditional societies tends to suppress the kind of psychopathology seen in borderline personality disorder. Once these patients emigrate to Western countries, this sociocultural suppression disappears.² In contrast, Tyrer *et al*³ and Baleyrier *et al*⁴ observed a lower incidence of personality disorders in immigrant patients admitted to psychiatric emergency services. Likewise, in a previous study that was not centred on an immigrant population, we found that patients with borderline personality disorder were less likely to be immigrants.⁵ For this reason, we performed an exploratory study (i.e. without an initial hypothesis) to examine whether there

really was an association between immigration and borderline personality disorder, where immigration could either be a risk factor or have a 'protective' effect.¹ Despite the fact that, in our opinion, we observed a 'protective' association for immigration on the development of borderline personality disorder, our results do not invalidate Paris's hypothesis. In Spain, immigration is a relatively new phenomenon, and the majority of patients we evaluated were adults from poorer countries who were not yet totally immersed in Western culture. It is possible that in younger immigrants (whose personality has not yet been totally consolidated) or in second-generation immigrants, a higher prevalence of borderline personality disorder could eventually be observed, as suggested by Paris.²

Second, another important point of our study is that the immigrant sample must not be considered as a homogeneous group, since important differences exist between the subgroups of immigrants according to their geographical origin. For instance, patients from sub-Saharan Africa and Asian countries were more than seven times less likely than other immigrants to be diagnosed with borderline personality disorder. Therefore, it could be suggested that certain cultural differences in these regions, for example a greater tolerance of suffering, could be useful factors to prevent the development of this disorder. The identification and analysis of these 'protective' cultural factors could offer future tools to prevent the appearance of borderline personality disorder in Western societies.

We would also like to highlight that although we share Dr Mushtaq's opinion that it is unlikely that immigration may be a risk factor for borderline personality disorder, the empirical evidence so far is not only scarce but also somewhat contradictory and with important methodological limitations. In fact, our own study presents some of these limitations. To confirm our findings, more methodologically rigorous studies would be necessary.

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doi: 10.1192/bjp.194.5.468

Clozapine and risk of pneumonia

Taylor *et al* showed that among the 'reasons for discontinuing' clozapine is the unfortunate outcome of death.¹ Out of the 21 deaths reported, five patients died from pneumonia (~24%). Interestingly, 'there was no evidence of neutropenia or agranulocytosis in any patients at the time of death'.¹

The relationship between clozapine and infection is indeed complex. Links between clozapine agranulocytosis, and between agranulocytosis and the increased risk of infection are well