

Correspondence

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Ethnic effects – a view from West London

I shared a critical review of Das-Munshi *et al*'s paper¹ with colleagues at the Ealing Journal Club, West London Mental Health Trust, highlighting issues of considerable relevance. My observations below have therefore been enriched by the reflections of a group of doctors working in psychiatry.

The first observation is that clinical applicability is limited by the study's use of a screening test and its cross-sectional survey design. A screening test for psychotic experiences rather than a clinical assessment tool for psychotic illness distances the findings from the clinical domain. As Das-Munshi *et al* indicate, there is reason to believe that psychotic experiences and illness are correlated; and the statistical properties of Bebbington's Psychosis Screening Questionnaire (PSQ)² *v.* a gold standard³ look impressive. However, especially with the symptom domain of delusions, there seems room to doubt how effectively a member of the general public will self-assess for presence/absence of symptoms; and, with the symptom domain of hypomania, how well this taps into the core concept of psychosis (construct validity).

As the authors acknowledge, the cross-sectional design brings broader limitations, precluding inferences of temporality. Even if an association between psychosis and ethnic density is granted, it remains unclear which way the arrow is pointing. It may even be that ethnic density is just another factor in a nexus of contributors to overall risk of psychosis (multifactorial model).

A second observation is that, although the associations between psychosis and psychosocial factors (racism/discrimination, chronic strains and social support) are quite convincing, those between psychosis and ethnic density are less robust. Indeed, statistical significance is only reached for the Indian group and the combined minority sample. From here, it seems a substantial jump to claim that 'the general trend was supportive of similar associations for all minority groups'.

This connects to the third observation: that, in grouping together the different ethnic groups (thereby generating sufficient power for a significant effect), the authors may have fallen foul of the same 'over-homogenising' they rightly deem a 'limitation of previous work'.

Finally, we were intrigued to note the 'cross-over' effects in the interactional models (Figs 3 and 4). It appears that, at very low ethnic densities ('ethnic isolation'), practical/confiding support from the nominated closest person may actually increase risk of psychotic experiences. A chance finding cannot be excluded, but there are other possibilities. First, more supportive relationships may have elements of enmeshment, possibly related to high expressed emotion. Second, a more unwell person might elicit higher levels of support from those closest to them. Third, in the context of ethnic isolation, a close and supportive relationship may actually limit chances of encountering disconfirmatory evidence from outside the 'relationship bubble' for any psychotic-spectrum symptoms emerging.

We would like to thank Das-Munshi and colleagues for their thought-provoking paper, and would welcome their own thoughts on the above observations.

- 1 Das-Munshi J, Bécaries L, Boydell JE, Dewey ME, Morgan C, Stansfeld SA, et al. Ethnic density as a buffer for psychotic experiences: findings from a national survey (EMPIRIC). *Br J Psychiatry* 2012; **201**: 282–90.
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Authors' reply: We are pleased that our study has been discussed among a group of practising psychiatrists and appreciate Dr Yates' interest.

We disagree, however, that the clinical applicability of the study findings are limited. As van Os highlighted in the accompanying editorial to our paper,¹ psychotic experiences elicited through the Psychosis Screening Questionnaire (PSQ) are probably indicative of a dimensional phenotype which is frequently present in common mental disorders, as well as present in psychotic disorders. Although a proportion of people screening positive on the PSQ may later convert to clinical psychosis (cited in van Os¹ and Das-Munshi *et al*)², for common mental disorders the presence of these symptoms usually suggests a poorer prognosis.¹ The clinical applicability of our findings is that there may be important risk factors in the environment which are associated with an increased risk of mental disorders in ethnic minority groups. From both a public health perspective and a clinical perspective, the findings suggest ways in which one may intervene to modify these social risk factors. As van Os suggests,¹ the advantage of using the PSQ as an outcome measure also meant that the findings of the study cut across traditional diagnostic boundaries. This may be why we found similar associations in a related analysis on common mental disorders.³

We wholeheartedly agree that associations of psychotic experiences with ethnic density should be seen within a multifactorial model where individual-level factors are understood from within a contextual framework. The inference of causality in a complex condition such as psychosis (or any other mental disorder) should be multifactorial, and should also assess possible interactions between causes,⁴ as we believe our study has done.

The assertion that 'the general trend was supportive' of density associations for all of the ethnic minority groups was a reference to Fig. 1 in the paper, which suggests that only the White British group were not party to protective density effects. Focusing on an arbitrary significance cut-off of $P=0.05$ masks an understanding of the study, which we feel must also be informed by looking at effect sizes as well as associated confidence intervals.⁵

An advantage of our analysis was the ability to examine contextual and individual-level experiences by ethnic group. We did assess and present data relating to the 'combined' ethnic minority sample as well (Figs 2–4).² Although we assert that research should refrain from 'lumping' minority groups together, some experiences (especially those relating to discrimination, social support and adversity) may have salience and cut across cultural or ethnic differences, and so we felt justified in presenting this for the full sample.

Finally, Dr Yates' suggestion for some of the 'cross-over effects' shown in the interaction models are fully justified as this is a cross-sectional study and so temporality cannot be assumed. We are pleased that the study lends speculation as well as an understanding to this area, and hope that future research may help address some of the issues highlighted by our paper.

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Identifying treatment-resistant schizophrenia

I was surprised to read the paper of Howes *et al.*¹ No control group was investigated. This paper implies that any patient for whom a third antipsychotic is considered ought to be considered for clozapine, and if they are not started on clozapine, this constitutes an unacceptable delay.

Such a sweeping assumption cannot be verified without the inclusion of a group of preferably contemporaneous control patients of approximately the same age, gender, diagnosis and treatment duration, who were treated with a third antipsychotic drug, but who did not go on to receive clozapine. Merely the existence of such a group verifies that the delay is indeed theoretical. How are those who will go on to need clozapine be identified? One cannot predict the future, stating with confidence that all patients on a third antipsychotic will require clozapine eventually. The paper is silent on these consequential issues.

This obvious limitation is not mentioned by the authors in their critique of their methodology. Neither does Patel's editorial commentary remark on it.²

I emphasise that I agree with the authors, that delay in initiating clozapine does many patients a great disservice. I have been one of the UK's major users of and proponents of clozapine since 1990, and recently published on clinical use of clozapine in *Advances in Psychiatric Treatment*.³ I still, regularly, encounter patients who should have been started on clozapine much longer than 4 years ago. The trouble is, the longer this treatment decision is ignored or put off, the more reluctance there seems to be. Nevertheless, there remain a number of alternative strategies to address those contributors to unsatisfactory response to antipsychotic drugs, such as poor adherence, substance misuse and intolerance, which may not necessarily succumb to clozapine treatment.

- 1 Howes OD, Vergunst F, Gee S, McGuire P, Kapur S, Taylor D. Adherence to treatment guidelines in clinical practice: study of antipsychotic treatment prior to clozapine initiation. *Br J Psychiatry* 2012; **201**: 481–5.
- 2 Patel MX. Clinician hesitation prior to clozapine initiation: is it justifiable? *Br J Psychiatry* 2012; **201**: 425–7.
- 3 Mortimer AM. Using clozapine in clinical practice. *Adv Psychiatr Treat* 2011; **17**: 256–65.

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Authors' reply: We thank Professor Mortimer for raising some important issues. The first is about the absence of a control group. Our study was observational, designed to determine clinical practice in antipsychotic prescribing prior to clozapine relative to treatment guidelines, which is why there is no control group. Prescribing in patients who have treatment-refractory illnesses but have never received clozapine is an important issue but outside the scope of our study. The second issue was that the paper implies that any patient for whom a third antipsychotic is considered ought to be considered for clozapine. Our study specifically selected patients who went on to be prescribed clozapine, predominantly because the illness was treatment refractory (see p. 481).¹ As such, it is relevant to patients with treatment-refractory illness, and in no way should be taken to imply that all patients who are being considered for a third antipsychotic should automatically be considered for clozapine. It is important to note that our design could not confirm the illness was treatment refractory at the point at which two antipsychotics had been used and, although treatment resistance can be evident from the first episode,² it may potentially emerge later in some patients (this is discussed further in the paper, p. 483¹). For this reason, the delay is described as the maximum theoretical delay (see p. 482).¹

We agree with Professor Mortimer that it is currently not possible to predict which patients will have treatment-refractory illness or will respond to clozapine, although the development of pathophysiologically specific markers may enable this.^{3–6} Likewise, we agree with Professor Mortimer that clozapine is not the only option for treatment resistance, although it has the best evidence base and this is reflected in its pre-eminence in treatment guidelines. However, we found that over a third of patients had received antipsychotic polypharmacy, and a third had received high-dose antipsychotic treatment prior to clozapine treatment.¹ This indicates that poorly evidenced strategies are commonly used in preference to clozapine and, to echo Professor Mortimer, this is a disservice to our patients.

Declaration of interest

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