

Correspondence

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Contents

- Is there a vulnerability paradox in PTSD? Pitfalls in cross-national comparisons of epidemiological data
- Ethnic density – meaning and implications

Is there a vulnerability paradox in PTSD? Pitfalls in cross-national comparisons of epidemiological data

Dückers *et al*¹ analyse the relationship between prevalence estimates of trauma exposure and post-traumatic stress disorder (PTSD) in published data-sets from 24 countries, and between PTSD and vulnerability (based on a country vulnerability index developed in the 2013 World Risk report). The findings are substantially counterintuitive; countries with low vulnerability have higher lifetime rates of PTSD, meaning that countries with low vulnerability and high trauma exposure have the highest rates of lifetime PTSD. The authors do emphasise a number of limitations of their work, and yet they conclude that a ‘vulnerability paradox’ exists for both PTSD and depression, with rates higher in countries with more resources and better healthcare systems.

This conclusion would seem inconsistent with a great deal of work in global mental health, which emphasises the considerable treatment gap in mental health services, with under-diagnosis and under-treatment particularly high in low- and middle-income countries.² It raises the question of what precisely is being measured by epidemiological studies of common mental disorders in general, and by studies of trauma exposure and PTSD in particular.³ There has been no shortage of critics of psychiatric nosology, including the construct of PTSD:⁴ are counterintuitive findings such as those of Dücker *et al* valid in some way, or do they underscore the limitations of our current classification systems, and the epidemiological surveys which employ related measures?

Consider, for example, the findings cited by Dücker *et al* that in South Africa and Lebanon, 73.8% and 68.85% of the population reported exposure to trauma, lower rates than in The Netherlands or Canada. In our view, given the multiple influences that determine self-reported rates of trauma exposure (including those noted by Dücker *et al*), comparing such rates across surveys is a matter of ‘comparing oranges and apples’. Other data from other sources may legitimately allow comparison of prevalence estimates: for example, the death rate from motor vehicle accidents in South Africa is 25.1 per 100 000 compared with 3.4 in The Netherlands, and there were 35.7 *v.* 8.9 murders per 100 000 in South Africa *v.* The Netherlands.⁵ Furthermore, rigorous examination of raw data across surveys (which Dücker *et al* note that they did not undertake) allows valid conclusions about trauma exposure: for example, that a small number of traumatic events account for a larger proportion of all traumatic event exposure across the world.⁵

When it comes to PTSD, Dücker *et al* note a prevalence of PTSD of 0.0% in Nigeria, 3.4% in Lebanon, and 9.2% in Canada; they emphasise a range of methodological issues that may have contributed to such findings, but nevertheless proceed to their analysis. In our view, the 0.0% prevalence estimate of PTSD in Nigeria should be considered as a single sampling, prone to any

number of measurement errors.⁶ While many sociocultural factors may affect the prevalence of PTSD, given the many universal findings about the phenomenology and psychobiology of PTSD,⁷ drawing strong inferences from this single data-point is not a scientifically sound approach. Again, however, other data and other analytic approaches do allow rigorous conclusions regarding the cross-national epidemiology of PTSD. For example, rigorous analysis of raw data from the World Mental Health Surveys has indicated that dissociative symptoms indicate a particularly severe and impairing subtype of PTSD.⁸

In view of these considerations, we wish to express our scepticism about the construct of a ‘vulnerability paradox’. This is not to criticise all of the fascinating literature on health paradoxes; it may well be the case that well and well-informed populations complain more about health problems than ill but ill-informed populations.⁹ And it is not to ignore the considerable methodological issues facing psychiatric classification and epidemiology in general, as well as particular issues relevant to trauma such as the causal relationship between trauma exposure and a range of disorders other than PTSD.¹⁰ Instead, our argument is that given these issues, certain kinds of analyses (such as those undertaken by Dücker *et al*) are fundamentally flawed, and the field should instead focus on those analyses which allow rigorous conclusions about trauma exposure and PTSD.

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Authors' reply: Vermetten *et al* repeat several of the limitations addressed in our original discussion.¹ We agree that the comparison of prevalence data is complicated and requires caution. We are also aware that current scientific evidence does not allow us to address all potential issues, despite the checks we reported in the initial paper. However, we do not agree that the points raised by Vermetten *et al* in any way contradict or detract from our analysis.

Their first suggestion is that our analysis is contradicted by known facts about the under-recognition and under-treatment

of PTSD in low- and middle-income countries. Under-recognition and under-treatment of PTSD, as well as problems with the PTSD construct, may well exist in certain countries; however, these issues are logically distinct and cannot explain the specific pattern of results we obtained. For example, under-treatment cannot explain why prevalence rates based on standardised population surveys are relatively high or low.

Their second concern is about the measurement of exposure to trauma. It is true that we could not distinguish between different exposure types, which we continue to see as an important limitation. But in our analyses – and also when the exposure rates by Benjet *et al*² are used – higher rates of trauma exposure were associated with higher prevalence in the expected way. It is not the exposure data but the country vulnerability data that generate the paradox. Vermetten *et al* do not raise concerns about the measurement of vulnerability.

We disagree with their suggestion that ‘drawing strong inferences from this single data-point is not a scientifically sound approach’. Table 2 and Figure 2 in our paper clearly show patterns in the data as a whole that are not reliant on one country. Vermetten *et al* suggest that ‘other analytic approaches do allow rigorous conclusions regarding the cross-national epidemiology of PTSD’. However, the example they give does not involve country-level variables, which are the focus of our analysis. It is not clear to us how their example is relevant to our quite different research question.

So far, we found indications that, regardless of exposure, PTSD and other mental health problems are more often observed in less vulnerable, more affluent countries.^{1,3} The analyses we have used are appropriate to the question asked. Rather than ignoring challenging findings, we believe it is scientifically responsible to explore them further. If reliable, they have potentially far-reaching implications from an international mental health perspective.

- 1 Dückers MLA, Alisic E, Brewin CR. A vulnerability paradox in the cross-national prevalence of post-traumatic stress disorder. *Br J Psychiatry* 2016; **209**: 300–5.
- 2 Benjet C, Bromet E, Karam EG, Kessler RC, McLaughlin KA, Ruscio AM, et al. The epidemiology of traumatic event exposure worldwide: results from the World Mental Health Survey Consortium. *Psychol Med* 2016; **46**: 327–43.
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Ethnic density – meaning and implications

The ecological study by Keown *et al*¹ is undoubtedly of value, both methodologically and in relation to the further exposition as to how ‘structural and social issues can shape mental health’, as Burns and Rugkåsa² (p. 97) note in their related editorial. However, some clarification of the authors’ use and operationalisation of the term ‘ethnic density’ is required to more fully understand the study’s implications and limitations.

The study documents ‘a positive association between ethnicity and compulsory in-patient treatment’ in urban areas (p. 158), but as the denominator of population analysis is relatively large (divided by primary care trusts (PCTs) with an average population of 350 000), it is unclear whether ‘ethnic density’ is defined in their study according to the overall prevalence of different

ethnic groups within these relatively large unit PCT populations under study, or whether smaller and more relevant unit neighbourhood-level measures of ethnic density have been used.

An important earlier study using such neighbourhood-level measures, by Das-Munshi *et al*,³ demonstrated that ‘people resident in neighbourhoods of higher own-group density experience “buffering” effects from the social risk factors for psychosis’ (p. 282). As psychotic presentations are more likely to result in compulsory admission, Das-Munshi *et al*’s findings would be expected to predict, when controlling for other variables highlighted by Keown *et al* – in particular, age and deprivation indices – that higher ethnic density, through ‘buffering effects’, would lead to lower levels of compulsory admission. Although it is possible that the findings of Das-Munshi *et al* and Keown *et al* are therefore in contradiction, it seems more likely that the Keown *et al* study did not measure ethnic density at the more relevant neighbourhood level in which buffering effects are manifest, and therefore that their measure of ‘ethnic density’ is less meaningful.

Ecological studies, by definition, attempt to attend to these more proximal influences on the immediate living environment.⁴ Although the data-set used by Keown *et al* no doubt precluded this, the contingent limitations of such data, if this was the case, are therefore important to further acknowledge. Neighbourhood-level ethnic density data would also be needed to confirm the significance of Keown *et al*’s unexpected finding of a lack of association between ethnicity and compulsion in rural areas, where genuine neighbourhood-level ethnic density might be expected to be low, at least in some areas. Nonetheless, Keown *et al*’s study alerts us to the importance of attending to both social and cultural factors influencing the genesis, precipitation and maintenance of mental illness, including psychosis, which may be variously protective or risk-amplifying, and which interact in complex – sometimes counterintuitive – ways, influencing prognosis,⁵ hospital admission and compulsion.

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Authors’ reply: We thank Rodger for his interest in our study and for the opportunity to clarify our measure of ethnic density. The measure used was the percentage of the total adult PCT population from Black and minority ethnic (BME) groups. However, the original work which preceded this ecological analysis¹ was a multilevel model to estimate the risk of compulsory admission, which involved simultaneous consideration of both individual ethnicity and ethnic density calculated as the proportion of adults reporting White British ethnicity for lower-layer super output areas (LSOAs; average population ~1500), which we loosely regarded as ‘neighbourhoods’. In that study, neighbourhood ethnic density was associated with an increased overall risk of compulsory