Missing ethnic density data

To investigate the concept of ethnic density one would have to draw a sample representative of the rich diversity of ethnicities in England. Modood & Berthoud[1] define ethnic group as:

'a community whose heritage offers important characteristics in common between its members and which makes them distinct from other communities. There is a boundary, which separates ''us'' from ''them'', and the distinction would probably be recognised on both sides of that boundary. Ethnicity is a multi-faceted phenomenon based on physical appearance, subjective identification, cultural and religious affiliation, stereotyping, and social exclusion.'

The study by Das-Munshi *et al*[2] included White British, Irish, Black Caribbean, Bangladeshi, Indian and Pakistani respondents. In the EMPIRIC study, ethnicity was defined by self-assessment using the same categories as the 1991 Census.[3] The 1991 Census collected data on nine ethnicity groups: White, Black Caribbean, Black African, Black other, Indian, Pakistani, Bangladeshi, Chinese and any other ethnic group.[4] Das-Munshi *et al*'s study has not looked at four of the nine ethnic subgroups listed and is therefore not representative of the ethnicities in England.

Das-Munshi *et al* have not explained the significant non-participation of candidates (37.8%) in the study, making it difficult to draw conclusions from the data. Owing to various factors – including that of participants having moved out of the survey area or to an unknown new address, or the participant having died or reached the age of 75 after the sample was drawn – 11% of the sample was no longer eligible for interview.[5]

Of eligible individuals, refusals were received in person directly from the selected participant in 18% of cases, a further 2% refusing by post and 2% being proxy refusals. Most non-contacts resulted from the interviewer being unable to make contact with the participant, although there were also cases where no contact was made with anyone at the household after four or more telephone call attempts. The most common reason for any other unproductive outcome was that the participant was away on holiday or in hospital throughout the survey period.[5]

1 Modood T, Berthoud R. *Ethnic Minorities in Britain: Diversity and Disadvantage – The Fourth National Survey of Ethnic Minorities*. Policy Studies Institute, 1998.

2 Das-Munshi J, Bécares 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.

3 Weich S, Nazroo J, Sproston K, McManus S, Blanchard M, Erens B, et al. Common mental disorders and ethnicity in England: the EMPIRIC study. *Psychol Med* 2004; **34**: 1543–51.

4 Office of Population Censuses and Surveys. *1991 Census: Definitions, Great Britain*. HMSO, 1992.

5 UK Data Archive. *Ethnic Minority Psychiatric Illness Rates In The Community (EMPIRIC): User Guide for UK Data Archive*. UKDA, no date (http://www.esds.ac.uk/doc/4685/mrdoc/pdf/4685userguide.pdf).

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Authors' reply: Dr Topiwala raises two additional points which we have discussed in our paper. The original EMPIRIC survey omitted a number of ethnic minority groups.[1] We were therefore unable to analyse associations for these groups as we did not have the data. As we mention in the 'Limitations' section of our paper,[2] since this survey (2002) there have been a number of other new migrant groups to Britain, for example from Eastern Europe. Therefore we caution that the findings should not be generalised outside of the ethnic minority groups surveyed in our study.

The issue of non-response has been discussed in the Method under 'Survey design'.[2] Weights to account for non-response bias were derived using stepwise logistic regression techniques using data which were available from previous Health Surveys for England.[1] A number of important demographic predictors of non-response were included in the models (including individual and household predictors as well as primary sampling units).[1] Interactions by ethnicity were also included in the models, in order to account for the differing probabilities of response by ethnic group.[1] As mentioned in our paper, wherever possible we used these survey weights to correct for non-response in our analyses.[2]

1 Sproston J, Nazroo J. *Ethnic Minority Psychiatric Illness Rates in the Community (EMPIRIC): Quantitative Report*. TSO (The Stationery Office), 2002.

2 Das-Munshi J, Bécares 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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Cardiovascular fitness and serious depression in adulthood

The report by Åberg *et al*[1] of an association between lower cardiovascular fitness at age 18 and serious depression in adulthood is interesting but their conclusion that the results 'strengthen the theory of a cardiovascular contribution to the aetiology of depression' is questionable. The authors recognise that although they controlled for psychiatric symptoms and disorders prior to and at the time of baseline assessment of cardiovascular fitness, they did not obtain information 'which could help in defining subsyndromal affective problems', nor details of 'other possible confounders such as personality, smoking and low self-esteem' that may increase risk for both poor fitness and depression. Seemingly lower cardiovascular fitness is not a risk factor for bipolar depression (or mania) and may not be a risk factor for females.[2] It is not stated whether consideration was given to psychiatric disorders apart from depression (e.g. anxiety disorders) and this leaves uncertainty as to whether lower cardiovascular fitness at age 18 is only a risk factor for non-bipolar depression in males. Considering that all study participants were sufficiently fit for recruitment into national service, it would be difficult to explain how a mere difference in cardiovascular fitness could (as suggested by the authors) contribute to cause 'severe' depression even before the age of 30. Given a recognised relationship between adverse developmental experiences, personality traits and depression,[3,4] it would be important to obtain information about such potentially confounding variables as well as 'subsyndromal problems' at the time of assessing cardiovascular fitness. It is suggested that in light of such missing information, the reported finding does not as yet 'strengthen the theory of a cardiovascular contribution to the aetiology of depression'. It is more an interesting association that warrants further investigation.

1 Åberg MAI, Waern M, Nyberg J, Pedersen NL, Bergh Y, Åberg DN, et al. Cardiovascular fitness in males at age 18 and risk of serious depression in adulthood: Swedish prospective population-based study. *Br J Psychiatry* 2012; **201**: 352–9.

2 Sund AM, Larsson B, Wichstrom L. Role of physical and sedentary activities in the development of depressive symptoms in early adolescence. *Soc Psychiatry Psychiatr Epidemiol* 2011; **46**: 431–41.

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3 Heim C, Newport DJ, Mletzko T, Miller AH, Nemeroff CB. The link between childhood trauma and depression: insights from HPA axis studies in humans. *Psychoneuroendocrinology* 2008; **33**: 693–710.

4 Boyce P, Mason C. An overview of depression-prone personality traits and the role of interpersonal sensitivity. *Aust N Z J Psychiatry* 1996; **30**: 90–103.

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Authors' reply: We would like to clarify a few points regarding our conclusion that our results 'strengthen the theory of a cardiovascular contribution to the aetiology of depression'.[1]

First, we talk about a 'contribution' which does not necessarily imply a direct causal pathway, stating that 'our findings are not explanatory with respect to causal chains leading to the onset of depression'. In line with this, we did include a careful discussion about other possible confounding mechanisms, i.e. factors that may increase the risk for both poor fitness and depression – for example, childhood factors, personality, self-esteem and subsyndromal affective problems. By including parental educational level as a confounder and by performing subanalyses within full brother pairs, many of the early childhood risk factors could be accounted for.

Second, as the conscription routines included extensive questions regarding every possible previous and present mental health problem in combination with separate examinations by professional psychologists and physicians, we believe that subsyndromal affective problems were not often overlooked. Also, to further reduce baseline misclassification, we did perform separate analyses excluding incident cases in the first year.

Third, we would like to stress that not all study participants were fit for recruitment into national service, but that the conscription test was used to select suitable recruits. Participation in the conscription tests was compulsory according to Swedish law and exemptions were granted only for incarcerated males and severe chronic medical disabilities (approximately 2–3% of the yearly male population). We can therefore consider our study a population study. After conscription, about 40 000 individuals were considered 'unfit' due to a cardiovascular fitness stanine score 1–3. All these 'unfit' young men were included in our study.

Fourth, the question of whether cardiovascular fitness may be related to increased risk for other types of psychiatric disorders in adulthood is one that we will continue to pursue in future analyses of the national conscription data.

Taken together, we still argue that the data 'strengthen the theory of a cardiovascular contribution to the aetiology of depression', which in our paper stands in direct connection with the sentence: 'although the results in the present population-based prospective study are compelling, a number of confounders could not be measured and intervention studies are needed to determine whether physical exercise in young adulthood can prevent future onset of depression.' We fully agree with de Jonge & Roest in their editorial[2] that a greater understanding of the mechanisms underlying these associations, including complex bidirectional models, may provide opportunities and strategies for prevention.

1 Åberg MAI, Waern M, Nyberg J, Pedersen NL, Bergh Y, Åberg DN, et al. Cardiovascular fitness in males at age 18 and risk of serious depression in adulthood: Swedish prospective population-based study. *Br J Psychiatry* 2012; **201**: 352–9.

2 de Jonge P, Roest AM. Depression and cardiovascular disease: the end of simple models. *Br J Psychiatry* 2012; **201**: 337–8.

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New paradigm: developmental psychopathology

Strong on diagnosis, but weak on prescription, Bracken *et al*'s[1] critique of contemporary psychiatry suffers from the very difficulty which they decry. They rightly complain that current paradigms ignore the psychosocial, fail to combat stigma, and that academic psychiatry has little impact on clinical practice. They cogently argue that the relational aspects of treatment, whether avowedly psychotherapeutic or pharmacological, outweigh any supposed specificity in their effectiveness.

Sadly, their remedies are vague and anodyne: encouraging service user involvement, acknowledgement of complexity, taking account of 'systems of meaning'. Motherhood and apple pie anyone? This anti-psychiatry rehash sounds the retreat rather well, but as a call to arms is feeble; it knows what it is 'anti', but lacks a convincing 'pro'.

Yet there is in fact an exciting way forward, one where academic psychiatry and psychology convincingly combine to enhance work in the clinic. Developmental psychopathology is the current cutting edge, drawing on attachment theory, neuro-imagining and epigenetics.[2] We are beginning to see how developmental experience inscribes itself on the brain, and sometimes on the genome; how the interaction of adverse developmental processes within the social milieu sows the seeds for psychiatric disorder. This provides the intellectual and evidential underpinning for effective psychotherapeutic treatments, which enhance resilience through fostering mentalising and mindfulness skills, promoting a sense of agency, and validating appropriate help-seeking. Psychiatrists-of-the-future's enthusiasm needs to be fanned by this flowering of environmental neuroscience, rather than doused with thin foam of post-modern angst.

1 Bracken P, Thomas P, Timimi S, Asen E, Behr G, Beuster C, et al. Psychiatry beyond the current paradigm. *Br J Psychiatry* 2012; **201**: 430–4.

2 Holmes J. Psychodynamic psychiatry's green shoots. *Br J Psychiatry* 2012: **200**: 439–41.

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Territorial disputes are a zero sum game: if one side gains ground, it can only be at the expense of the other. As clinical psychologists, it was therefore with a wry smile that we read the recent paper by Bracken and colleagues,[1] which calls for psychiatry 'to move beyond the dominance of the current, technological paradigm' and towards an understanding of mental health problems not as