

Correspondence

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Prisoners with mental disorders in Europe

The prevalence of psychiatric disorders in prisoners is substantially higher than in the general population.¹ Additionally, there is scientific evidence that the number of prison inmates with mental disorders is rising. As a consequence, the World Psychiatric Association has repeatedly voiced concern about the increasing number of mentally ill individuals who are being placed in correctional facilities.²

In European Union (EU) member states, forensic legal provision governing the diversion of offenders with mental disorders is diversely included in penal codes, general and mental health legislation, and it is difficult to establish whether member states place emphasis more on medical or punitive parameters in judicial deliberations.³ From a human rights perspective, depriving such prisoners of any state-of-the-art treatment cannot be accepted.⁴ However, there is a serious shortage of information and data in the field. Therefore, the European Commission funded the research project EUPRIS, which aimed to collect structured information on concepts, models and routine practices in prison mental healthcare in 24 EU member states and other European countries.⁵ The results of the study are alarming and should give rise to public policy and research activities. Even the most rudimentary health-reporting standards for mental healthcare in prison are lacking almost everywhere in Europe. Almost none of the included countries provided regular national statistics on the frequency of mental disorders of prisoners or on the availability or frequency of psychiatric treatments. A major reason for the lack of data on the prevalence of mental disorders in prisons is the deficient implementation of standardised psychiatric screening and assessment procedures at prison entry and during imprisonment. In many countries, the appointment of inadequately trained staff to perform such screenings increases considerably the risk that mental disorders or psychiatric needs of the inmates will remain undetected. Furthermore, the pathways to care in case of an acute psychotic episode differ significantly, since referral to prison hospitals, medical prison wards, forensic hospitals or general psychiatric hospital are used in various combinations depending on different national legal regulations, the availability of services and other regional circumstances. Therefore, the equivalence of care for prisoners with mental disorders has been questioned by the cooperating national experts. As a basic prerequisite for any action taken, more awareness of the deficiencies and problems must be raised by responsible authorities and decision makers. The implementation of some basic indicators used in general psychiatric research seems to be overdue.

Declaration of interest

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One ace and three faults don't win the set

The well-written paper by Álvarez-Jiménez *et al*¹ attempts to address a major concern in the management of psychosis, namely weight gain with antipsychotic medication, which has an overarching impact on the management of psychosis. The question it purports to answer is clearly focused and the search strategy thorough and systematic.

However, from the description of the conduct of included trials and assessment of the risk of bias presented in the online Table DS2, it becomes clear that several poor-quality trials were included, with only 2/10 having used an intention-to-treat analysis and 1/10 disclosing allocation concealment.

Proper randomisation is particularly important in small trials as it is relied upon to produce groups with similar baseline characteristics. Poor-quality randomisation would instead produce unequal groups with questionable validity of the results.²

The attrition rate is particularly high (up to 50%) for the control group in this case. Empirical evidence suggests that participants who adhere to medication tend to do better than those who do not, even after adjustment for all known prognostic factors and irrespective of assignment to active treatment or placebo.

In the absence of an intention-to-treat analysis, the results are biased in an unpredictable manner, compounded by the small size of the trials. Similar problems extend to the subgroup analysis. The authors confirm that the effect size is reduced in the better-quality studies. Three out of four trials in the nutritional therapy subgroup analysis were of poor quality; similarly, four out of five studies in the comparison of individual *v.* group therapy. Hence, by including poor-quality trials with larger treatment effects in the analysis, the beneficial effect of the intervention has been overestimated.

The choice of mean weight change as an outcome measure is an interesting one as it actually masks the heterogeneity between individuals in small trials. In simple terms, if one person in the intervention arm of the trial, loses 20 kg it skews the results in favour of the intervention even if the other five individuals gained 2 kg each, giving a group mean weight loss of 10 kg. It would perhaps have been more appropriate to have chosen a dichotomous definition of significant weight change (say 5%), so that it would be clear how many individuals actually benefited from the intervention.

The reviewers could have chosen to request the raw data from individual trials, to allow them the opportunity to account for

those who withdrew, redo the intention-to-treat analysis and calculate dichotomous weight change outcomes. This, however, would still not resolve the basic problem with regard to quality in individual studies. Well-designed, large-scale pragmatic trials with longer periods of follow-up are needed before undertaking further review in this area, an implication which has been acknowledged by the authors.

- 1 Álvarez-Jiménez M, Hetrick SE, González-Blanch C, Gleeson JF, McGorry PD. Non-pharmacological management of antipsychotic-induced weight gain: systematic review and meta-analysis of randomised controlled trials. *Br J Psychiatry* 2008; **193**: 101–7.
- 2 Hollis S, Campbell F. What is meant by intention to treat analysis? Survey of published randomised controlled trials. *BMJ* 1999; **319**: 670–4.

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Authors' reply: We would like to thank George *et al* for their comments. However, we believe that some clarification is needed regarding the outcomes and procedures of our meta-analysis.

First, we agree that percentage of weight gain is a more appropriate measure to assess weight gain compared with body weight change. In fact, somewhere else we have pointed to 'the importance of reporting percentage of weight gain, as absolute body weight changes may be deceptive, concealing the real extent of this side effect on those who experience weight gain'.¹ To put it more simply, research shows that up to 80% of individuals being treated with antipsychotics suffer significant gain in body weight. As a result, patients taking antipsychotics are more likely to gain 20 kg than they are to lose 20 kg. Indeed, weight-management interventions do not usually produce weight loss but they attenuate antipsychotic-induced weight gain.² For these reasons, data on weight change is unlikely to overestimate the effectiveness of weight management interventions as George *et al* contend. To illustrate this further, in a previous randomised controlled trial (RCT) of weight-management interventions we assessed the proportion of patients that gained more than 7% of their baseline body weight. Patients in the control group gained 6.9 kg compared with 3.9 kg in the intervention group. These absolute gains were translated into 78.8% in the control group increasing their baseline weight by more than 7% *v.* 39.9% in the intervention group.³

George *et al* also commented on the quality of the included trials as a potential threat to the reliability of the results. First, it should be pointed out that only RCTs were included – in three of them we were able to pool relevant data with the help of the authors. Second, we performed several sensitivity analyses to determine the robustness of our findings to the exclusion of low-quality trials and exclusion of small trials.⁴ The exclusion of these studies affected the overall effect size and confidence intervals only marginally. Importantly, there was notable consistency across all study estimates, which was reflected in the robustness of the findings across analytic methods. Thus, our findings are unlikely to be biased by these issues.

After examining all the available evidence, it is now possible to conclude that large-scale pragmatic trials with longer follow-up are needed to make further progress in this area as George *et al* state.

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gain in chronic and first-episode psychotic disorders: a systematic critical reappraisal. *CNS Drugs* 2008; **22**: 547–62.

- 2 Álvarez-Jiménez M, Hetrick SE, González-Blanch C, Gleeson JF, McGorry PD. Non-pharmacological management of antipsychotic-induced weight gain: systematic review and meta-analysis of randomised controlled trials. *Br J Psychiatry* 2008; **193**: 101–7.
- 3 Álvarez-Jiménez M, González-Blanch C, Vázquez-Barquero JL, Pérez-Iglesias R, Martínez-García O, Pérez-Pardal T, et al. Attenuation of antipsychotic-induced weight gain with early behavioral intervention in drug-naïve first-episode psychosis patients. A randomized controlled trial. *J Clin Psychiatry* 2006; **67**: 1253–60.
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Aetiological significance of middle-ear disease in schizophrenia

We read the study by Mason *et al*¹ with great interest. The authors conclude that there is an association between middle-ear disease and schizophrenia which may have aetiological significance. However, the authors have based their conclusions on a case-control study, which is susceptible to biases and effects of confounding factors; we would like to raise concerns about these conclusions.

First, we would like to highlight the strong possibility of selection bias as this study design is particularly prone to it. In this case, at the sample selection stage, no precautions were taken to ensure that the person selecting the patients was masked to the study hypothesis. This could lead to bias towards selecting patients with middle-ear disease and schizophrenia.

Case-control studies are more susceptible to bias and confounding factors than are cohort studies. In order to establish the association, it is recommended that we should have an odds ratio > 4 ,² because the higher the odds ratio, the stronger the association. However, Mason *et al* have concluded about the association when the odds ratio is < 4 , which could be as a result of bias alone. This raises strong doubts about the validity of the authors' conclusions.

We would request that the authors clarify these issues.

- 1 Mason P, Rimmer M, Richman A, Garg G, Johnson J, et al. Middle-ear disease and schizophrenia: case-control study. *Br J Psychiatry* 2008; **193**: 192–6.
- 2 Sackett DL, Straus SE, Richardson WS, Rosenberg W, Haynes RB. *Evidence-Based Medicine. How to Practice and Teach EBM*. Churchill Livingstone, 2000.

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Author's reply: Jainer & Shivanandaswamy's comments about the problems of bias in case-control studies are well made. However, our study¹ was designed to avoid such problems by recruiting all patients with a likely diagnosis of schizophrenia in contact with general practitioners in a defined catchment area. There was no possibility of influencing the selection of individuals

since they were all patients with a diagnosis of schizophrenia on a community mental health team's case-load.

The community mental health team concerned looked after an area of high socio-economic deprivation and the study included patients who had drifted down the social scale from more affluent rural areas where one would expect a lower prevalence of middle-ear disease. If there is any bias in this study it is likely to favour the null hypothesis rather than that suggested by Jainer & Shivanandaswamy.

In addition, perhaps the most striking finding in this study was the excess of left-sided middle-ear disease. In this case, the odds ratio of 4.15 meets the recommendation of Sackett *et al*² that an odds ratio of greater than 4 should be used to establish an association in case-control studies.

- 1 Mason P, Rimmer M, Richman A, Garg G, Johnson J, et al. Middle-ear disease and schizophrenia: case-control study. *Br J Psychiatry* 2008; **193**: 192–6.
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DSM-V: should PTSD be in a class of its own?

Principles of diagnostic taxonomy suggest that disorders of a specific class, or spectrum, should aggregate more with each other than with disorders from another class. Results of recent comorbidity studies raise questions about whether this is true for post-traumatic stress disorder (PTSD) – which has been classified as an anxiety disorder since DSM-III – and the implications for where the diagnosis should be located in DSM-V.

Several factor analyses of diagnostic data from epidemiological and clinical samples suggest that PTSD covaries more strongly with disorders defined by anhedonia, worry and rumination (i.e. the unipolar mood disorders and generalised anxiety disorder) than with ones characterised by pathological fear and avoidance (e.g. the phobias, panic/agoraphobia and obsessive-compulsive disorder).¹ However, classifying PTSD among these 'anxious-misery' disorders provides a poor fit to the data because PTSD is conditional on trauma exposure and, in new-onset cases, typically develops before its comorbid conditions. For example, when PTSD and major depression co-occur following trauma exposure, PTSD usually precedes or develops concurrently with the depression. New-onset major depression that develops in the wake of trauma rarely precedes or develops in the absence of PTSD.² This implies a causal influence of PTSD on comorbid psychopathology and suggests a distinct phenomenology which should be reflected in its diagnostic class membership within DSM.

Developmental studies have shown that adult psychopathology is often foreshadowed by childhood and/or adolescent problems in the same domain. Along these lines, many adults with anxiety disorders report histories of juvenile anxiety disorders but they do not typically report juvenile externalising disorders. The exception to this is found among samples of individuals with PTSD where adult patients frequently have histories of childhood externalising disorders.³ Twin studies align with this finding and have shown that PTSD shares genetic influences with both internalising- and externalising-spectrum diagnoses.⁴ Other work suggests that many adults with PTSD exhibit a predominantly externalising pattern of comorbidity characterised by problems in the domain of impulse-control, antisociality and substance misuse.⁵ These findings raise concern about conceptualising PTSD simply as the manifestation of a vulnerability to anxiety-related psychopathology.

Since its third edition, the DSM has taken a largely descriptive, as opposed to aetiological, approach to defining and classifying disorders. The most notable exception to this is the PTSD diagnosis, which specifies a causal relationship between trauma exposure and symptom development. We believe that the most appropriate location for PTSD in DSM-V would be among a class of disorders precipitated by serious adverse life events, i.e. a spectrum of traumatic stress disorders. Candidates for inclusion would include PTSD, acute stress disorder, adjustment disorder, a traumatic grief or bereavement-related diagnosis, and possibly complex PTSD. These disorders are the product of an environmental pathogen (i.e. a traumatic stressor) operating on individual diatheses that span the spectrum of human variation in vulnerability to psychopathology. This diathesis-stress interaction can result in extensive heterogeneity in the phenotypic expression of psychopathology – pathological anxiety being just one manifestation of the process.

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Correction

Interventions for people bereaved through suicide: systematic review. *BJP*; **193**: 438–43. Reference 5 should read: Hawton K, Simkin S. Helping people bereaved by suicide. Their needs require special attention. *BMJ* 2003; **327**: 177–8.

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