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.


3 The inclusion of weight management interventions as George et al. contended would lead to bias towards selecting patients with greater weight change. 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. 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.

4 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 > 4.7 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.


Mario Álvarez-Jiménez, ORYGEN Research Centre, University of Melbourne, Australia, and University Hospital ‘Marqués de Valdecilla’, Department of Psychiatry, Santander, Spain. Email: mahavez@unibalmc.edu.au; Sarah E. Hetrick, ORYGEN Research Centre, University of Melbourne, Australia; César González-Blanch, University Hospital ‘Marqués de Valdecilla’, Department of Psychiatry, University of Cantabria School of Medicine, Santander, Spain; John F. Gleeson, ORYGEN Research Centre, and Department of Psychiatry, University of Melbourne, and NorthWestern Mental Health Programme, Melbourne, Australia; Patrick D. McGorry, ORYGEN Research Centre, University of Melbourne, Australia.

doi: 10.1192/bjp.194.1.89a

Aetiologic 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 > 4.7 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.


Ashok K. Jainer, Coventry & Warwickshire Partnership Trust, Caludon Centre, Coventry CV2 4TE, UK. Email: ashokjainer@hotmail.com; Supriya M. Shivanandaswamy, ORYGEN Research Centre, University of Melbourne, and NorthWestern Mental Health Programme, Melbourne, Australia; Patrick D. McGorry, ORYGEN Research Centre, University of Melbourne, Australia.

doi: 10.1192/bjp.194.1.89a

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...