BJPsych | The British Journal of Psychiatry (2013) 202, 152–156

Correspondence

Edited by Kiriakos Xenitidis and Colin Campbell

Contents

Psychotic experience: things to consider

Kelleher *et al*'s study is very interesting and raises some important questions,[1] but we think that it also has some confounding factors that need to be addressed before conclusions are made. In addition, there are some methodological issues which we would like to be clarified. The response rate in study 1 is 52%, which might not be enough to support the conclusion of this kind of study. Second, owing to the different inclusion criteria in studies 1 and 2, there is a strong case for non-response bias. The way in which the first interview sample (study 3) was assembled seems unclear. Also, the way in which the second interview sample (study 4) was composed raises questions as to whether it can truly be considered a sample that represents the general population as claimed in the article. As far as confounding factors go, there is no mention of psychoactive substance misuse. With the potential of drugs to produce hallucinogenic effects, and the known link between conduct disorder, depression and attention-deficit hyperactivity disorder with substance misuse comorbidity,[2] there is a chance that this could lead to results that do not reflect the true nature of the link between psychotic symptoms and non-psychotic disorders.

Another thing that could possibly be of interest and could affect the overall conclusions of the study is whether the study made any kind of differentiation between hypnagogic, hypnopompic and daytime hallucinations.[3] Last, there is no mention on the effects of the hallucinations on the children and adolescents, whether they have perceived them as positive, negative or neutral, and whether they have sought any help or counselling because of them. There is also no mention of help-seeking or school and family problems among the children and adolescents who were classified as having a diagnosable non-psychotic disorder, which might have been a more precise way to link the severity of childhood and adolescent problems than the simple use of the number of comorbid diagnoses assessed in one interview in a non-clinical setting.

1 Kelleher I, Keeley H, Corcoran P, Lynch F, Fitzpatrick C, Devlin N, et al. Clinicopathological significance of psychotic experiences in non-psychotic young people: evidence from four population-based studies. *Br J Psychiatry* 2012; **201**: 26–32.

2 Zeitlin H. Psychiatric comorbidity with substance misuse in children and teenagers. *Drug Alcohol Depend* 1999; **55**: 225–34.

3 Ohayon MM, Priest RG, Caulet M, Guilleminault C. Hypnagogic and hypnopompic hallucinations: pathological phenomena? *Br J Psychiatry* 1996; **169**: 459–67.

Milutin V. Kostic, Psychiatry Trainee, Institute of Mental Health in Belgrade, Palmoticeva 37, 11000 Belgrade, Serbia. Email: milutin.kostic@imh.org.rs; **Nikola Jovanovic**, Psychiatry Trainee, **Ana Munjiza**, Psychiatry Trainee, **Danilo Pesic**, Psychiatry Trainee, **Aleksandar Repac**, Psychiatry Trainee, Institute of Mental Health, Belgrade, Serbia.

doi: 10.1192/bjp.202.2.152

Authors' reply: There are a number of misunderstandings put forward by Kostic *et al* that we should clarify. First, it is important to correct the authors with regard to their understanding of the issue of confounding: a confound is a variable of relevance in epidemiological models of causation. To be clear, we did not suggest in our report that psychotic symptoms somehow cause psychiatric disorder. Symptoms and signs of course cannot cause pathology; rather, they act as clinical risk markers for disease. Using an analogy from respiratory medicine, the authors' suggestion that we should control for substance misuse (which is a potential cause of psychotic symptoms) makes no more sense than suggesting that respiratory researchers should control for cigarette smoking when looking at haemoptysis as a risk marker for lung pathology. That is, haemoptysis alerts the clinician to the likely presence of pathology (i.e. it is a risk marker); the cause of the pathology remains to be determined. Similarly, we showed that psychotic symptoms act as risk markers for a broader range of psychopathology than has generally been recognised (and, in particular, for multimorbid psychopathology). In the same way that there are multiple mechanistic causes for the occurrence of haemoptysis in lung pathology (e.g. cigarette smoking, infection, trauma), there are also likely multiple mechanistic causes for the occurrence of psychotic symptoms in psychopathology. In this regard, we would direct the authors to paragraph three of the Discussion, in which we put forward a number of suggestions for such causes.

Kostic and colleagues also wonder whether the response rate in study 1 or the fact that study 4 specifically overselected for psychopathology may have affected the validity of these findings. Unfortunately, we do not have space to provide a comprehensive explanation of the epidemiological impact of response rates on findings; however, it is important to clarify that, although response rates can introduce bias with regard to reported incidences or prevalences, they usually have little effect on statistical measures of association. With regard to study 4, which purposely overselected for psychopathology, this is, in fact, the very methodological basis of a case–control study. A statistical weight must be applied to determine population prevalences from such an approach but, as evidenced by the many thousands of case–control studies in the medical literature, this does not create problems for identifying associations that can be generalised to the population. Quite aside from this, we would remind the authors that the best way to address the possibility that sampling and other biases are responsible for a set of results is independent replication; our findings were replicated across multiple independent studies, led by multiple independent teams in multiple independent centres. With regard to symptom inclusion, in accordance with the guidelines of the interview instrument (the Schedule for Affective Disorders and Schizophrenia for School-Aged Children),[1] hypnopompic, hypnagogic and drug-induced hallucinations were excluded, as were symptoms experienced only in the context of febrile illness.

Last, Kostic and colleagues state that there was no mention of the potential role of 'school and family problems' in our findings, although we specifically suggested this as an important issue in our discussion. In fact, we have already published results from study 4 (in this journal, in fact) on the relationship between