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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.

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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

psychotic symptoms and a number of measures of school and family problems, including bullying, interparental domestic violence and physical and sexual abuse.[2] We cited this in the paper. Furthermore, Kostic *et al* will be glad to know that a report on the relationship between childhood trauma and psychotic symptoms in another of the samples (study 2) is currently under review (details available from the authors on request). However, it is important to recognise that, again, the authors are raising an issue of causality in the relationship between psychotic symptoms and psychopathology; the point of the current paper, on the other hand, was to highlight new developments in our understanding of the importance of psychotic symptoms as clinical risk markers for psychopathology.

We appreciate that Kostic and colleagues are certainly not the only individuals who may have had conceptual misunderstandings about the above epidemiological points and we thank them for the opportunity to clarify some of these issues for the benefit of other readers with similar questions. We are also pleased to find that the *Journal*'s readers are actively discussing the importance of assessing psychotic symptoms in the context of non-psychotic psychopathology. As well as recognising that psychotic symptoms are risk markers for a range of non-psychotic Axis I disorders in general, and for multimorbidity in particular,[3] we would also especially encourage discussion about findings on the importance of these symptoms as risk markers for suicidal behaviour in young people with psychopathology.[4] Considering the serious implications of these findings, an improved awareness of the significance of these symptoms among clinicians is urgently needed.

1 Kaufman J, Birmaher B, Brent D, Rao U, Ryan N. *The Schedule for Affective Disorders and Schizophrenia for School Aged Children: Present and Lifetime Version*. University of Pittsburgh, Western Psychiatric Institute and Clinic, 1996.

2 Kelleher I, Harley M, Lynch F, Arseneault L, Fitzpatrick C, Cannon M. Associations between childhood trauma, bullying and psychotic symptoms among a school-based adolescent sample. *Br J Psychiatry* 2008; **193**: 378–82.

3 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.

4 Kelleher I, Lynch F, Harley M, Molloy C, Roddy S, Fitzpatrick C, et al. Psychotic symptoms in adolescence index risk for suicidal behavior: findings from two population-based case-control clinical interview studies. *Arch Gen Psychiatry* 2012; doi: 10.1001/archgenpsychiatry.2012.164. (Epub ahead of print.)

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The need for inclusion of concepts of recovery in clinical trials

The study by Tohen and colleagues addresses a field of clinical practice that has traditionally posed a great deal of therapeutic challenge.[1] Evidence of potential therapeutic response in initial trials are therefore welcome and the authors are right to call for further research to assess the efficacy of olanzapine, while cautioning in relation to the high non-adherence rates observed with this medication.

The authors also attempt to explore the degree of recovery experienced by individuals within their trial. It is correct that this concept is addressed, even in early trials such as this. By considering concepts such as recovery, clinical trials can provide information that allows clinicians and service users to make truly informed decisions in relation to treatment options. Calls for the inclusion of recovery-oriented outcomes in clinical trials into various disorders have been made.[2,3]

However, in this study the authors appear to make the mistake of conflating the concepts of recovery and symptom remission. The concept of recovery is generally recognised as being more than simple remission of symptoms, instead involving a deeper acceptance of disorder and personal adaptation to experience. In this journal, a narrative review by Leamy *et al* described five main themes of recovery that are representative of this concept; they are the sense of: connectedness, hope, identity, meaning and empowerment.[4]

Measures such as the Montgomery–Åsberg Depression Rating Scale (MADRS) are valuable in their sensitive detection of change in the symptoms of depressive disorders but they do not address the core concepts of recovery.[5] Simple definition of recovery as a sustained period of symptom remission (MADRS $\geqslant 12$ for $\leqslant 4$ weeks) as in this paper is therefore inadequate.

The development of suitable recovery-oriented outcome measures for inclusion in clinical trials is urgently required to allow us to develop an evidence base that considers all aspects of treatment and allows us to provide service users with the information they require to make informed treatment decisions.

1 Tohen M, McDonnell DP, Case M, Kanba S, Ha K, Fang YR, et al. Randomised, double-blind, placebo-controlled study of olanzapine in patients with bipolar I depression. *Br J Psychiatry* 2012; **201**: 376–82.

2 Slade M, Hayward M. Recovery, psychosis and psychiatry: research is better than rhetoric. *Acta Psychiatr Scand* 2007; **116**: 81–3.

3 Bateman AW. Treating borderline personality disorder in clinical practice. *Am J Psychiatry* 2012; **169**: 560–3.

4 Leamy M, Bird V, LeBoutillier C, Williams J, Slade M. Conceptual framework for personal recovery in mental health: systematic review and narrative synthesis. *Br J Psychiatry* 2011; **199**: 445–52.

5 Montgomery SA, Åsberg M. A new depression scale designed to be sensitive to change. *Br J Psychiatry* 1979; **134**: 382–89.

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Author's reply: I agree with Dr Shepherd that there is a need to better define outcomes in clinical trials. It is correct that we defined recovery as a sustained remission of psychiatric symptoms. Indeed, we followed the definition recommended by the International Society for Bipolar Disorders (ISBD).[1] The term recovery in the ISBD consensus guidelines is based on sustained absence of or low-severity symptomatology without considering functional outcomes.

Observational studies in bipolar disorder, however, have in fact shown that symptomatic remission is not always accompanied by functional recovery,[2,3] which supports Dr Shepherd's point that symptom resolution is not always followed by improved functional outcomes such as adaptation to the experience.

I agree with Dr Shepherd that functional outcomes allow clinicians to make better treatment decisions that are more patient-centred. Furthermore, in the consideration of regulatory approval around the globe, symptom improvement is the main criterion for a new treatment to get approved. Including functional outcomes in the regulatory approval of pharmacological treatments would be beneficial to patients.

1 Tohen M, Frank E, Bowden CL, Colom F, Ghaemi NS, Yatham LN, et al. The International Society for Bipolar Disorders (ISBD) Task Force report on the