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Authors' reply: We thank Khandaker *et al* for their response to our paper, and are in broad agreement that psychotic symptoms are more common than currently recognised in mood disorders. Indeed our previous work has highlighted this and likewise the importance of mood symptoms in psychosis.^{1,2} In our present study the presence of hallucination but not delusion was most frequent in those with a history of child sexual abuse.³ Hallucinations in particular may occur in non-psychotic diagnoses in the presence of childhood or later trauma.³ Thus hallucinations may have less specificity for psychosis than one might expect given the weight positive symptoms have in diagnostic terms.^{4,5} Khandaker *et al*'s proposal that diagnostic systems should acknowledge the presence of these experiences in non-psychotic disorders is important. This recognition would allow clinicians to better accept diagnostic uncertainty, allay worries provoked by an exclusive association of hallucinations with psychosis, and enable further investigation as to underlying causality within a symptom-specific approach.

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Childhood non-affective psychoses: data analysis

The epidemiology of psychosis in children is a story of even smaller numbers than for psychosis in grown-ups. But how much smaller? The short report of Tiffin & Kitchen¹ on incident cases of schizophrenia in children in 2010–2011 is a well-needed addition to a limited descriptive literature on psychosis in children. I have a couple of points about the analysis, which I feel might clarify the report.

The authors point out at the outset the heuristic that earlier onset cases of a disorder might have a 'greater loading of causal factors'. This does not seem to be correct – all cases of a given

disorder represent the interrelation of a number of risk factors on a causal pathway – it is therefore unclear why earlier cases would represent the culmination of pathways with a greater number of causal factors. The authors may mean that earlier onset psychoses could represent individuals with stronger risk factors for the disorder. However, although this is usually correct for diseases in general, this point rests on the assumption that there is a single underlying outcome under study, which has long been contested.² Assuming this heterogeneity is related in some way to the clinical heterogeneity of schizophrenia across people of different ages and with different risk factor patterns, this may explain the large number of false positives identified in Tiffin & Kitchen's study.

Second, although the authors present confidence intervals (presumably based on Poisson standard errors), it is important to point out what the intervals are referring to – this study attempts to capture all cases within a surveillance approach, rather than to sample the target population. In this case, the confidence intervals refer to the underlying randomness of the disease process, rather than to the design of the study.³ Confidence intervals used in this way are not meaningless, but they may not be strictly necessary here – it seems that reporting the crude rate in this type of study is an almost equally informative approach for the purposes of this research. Furthermore, uncertainty about the true rate of the disorder due to random variability ('error') in the disease process likely pales in comparison with the systematic error introduced by biases in this design.

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Authors' reply: To clarify, we did mean to state that earlier onset psychoses could represent individuals with stronger risk factors for the disorder. We agree that there is likely to be some heterogeneity in the outcomes, even if the loading of putative causal factors is similar for any set of given individuals. However, there is evidence that cases of childhood-onset psychosis spectrum disorders in general, when carefully defined, tend to be more severe and more homogeneous, with stronger family histories of schizophrenia spectrum disorders than adult-onset cases.^{1–3} Therefore, there may still be much to be learned about causality, even if the assumption of homogeneous clinical outcomes does not hold strictly true. Moreover, we believe the high rate of false-positive reports is likely to be due, at least in most instances, to clinicians initially wrongly attributing perceptual disturbance in children to an underlying psychotic illness. In most cases, it is likely that voice experiences and other potentially psychotic phenomena may result from processes that may be conceptualised as more psychologically driven, such as dissociation. Such experiences are commonly reported in community samples of children and adolescents, who are likely to share few, if any, of the risk factors associated with the development of early-onset schizophrenia spectrum disorders.⁴