did not follow-up the help-seeking individuals who underwent the clinical assessment at the prodromal services but were not considered at risk for psychosis (HR–). Consequently, it is completely obscure how the authors may have estimated the correct prevalence of false negatives (HR–, who developed psychosis over time) in their analysis. Given all the above concerns, I feel the results of this meta-analysis should be considered carefully as pilot data strongly undermined by significant methodological biases.

1 Chuma J, Mahadun P. Predicting the development of schizophrenia in high-risk populations: systematic review of the predictive validity of prodromal criteria. *Br J Psychiatry* 2011; **199**: 361–6.

2 Yung AR, Phillips LJ, Yuen HP, Francey SM, McFarlane CA, Hallgren M, et al. Psychosis prediction: 12 months follow up of at risk (prodromal) group. *Schizophr Res* 2003; **60**: 21–32.

3 Yung AR, Phillips LJ, Yuen HP, McGorry PD. Risk factors for psychosis in an ultra high-risk group: psychological and clinical features. *Schizophr Res* 2004; **67**: 131–42.

4 Yung AR, Phillips LJ, Yuen HP, McGorry PD, Kelly D, Dell'olio M, et al. Mapping the onset of psychosis: the comprehensive assessment of at risk mental states. *Aust NZ J Psychiatry* 2005; **39**: 964–71.

5 Yung AR, Nelson B, Stanford C, Simmons MB, Cosgrave EM, Killackey E, et al. Validation of 'prodromal' criteria to detect individuals at ultra high risk of psychosis: 2 year follow up. *Schizophr Res* 2008; **105**: 10–7.

6 Woods SW, Addington J, Cadenhead KS, Cannon TD, Cornblatt BA, Heinssen R, et al. Validity of the prodromal risk syndrome for first psychosis: findings from the North American Prodrome Longitudinal Study. *Schizophr Bull* 2009; **35**: 894–908.

7 Cannon TD, Cadenhead K, Cornblatt B, Woods SW, Addington J, Walker E, et al. Prediction of psychosis in youth at high clinical risk: a multisite longitudinal study in North America. *Arch Gen Psychiatry* 2008; **65**: 28–37.

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Authors' reply: Dr Fusar-Poli identified a number of studies reporting follow-up transition rates, which is not the same as predictive the validity of the tests or criteria. Most, if not all, of the studies he identified did not have information on predictive attributes of the tests or criteria, such as sensitivity and specificity. However, they had useful information on transition rates. From these it is impossible to know how good the tests/criteria were in ruling in or out the risk of developing schizophrenia from prodromal symptoms, since these studies were not systematically following up those who tested negative to the test.

Dr Fusar-Poli raised another important issue regarding overlapping of samples. We checked for double publication, but not necessarily overlapping of samples. We were interested in knowing how good the test is in predicting schizophrenia in high-risk populations. We therefore were interested in diagnostic attributes of a test in each study/subsample. The values for sensitivity and specificity for Yung *et al* (2003)[1] and Yung *et al* (2004)[2] were not identical. For the purposes of predictive validity of a test, these are two different studies. Yung *et al* (2005)[3] had a follow-up of 6 months ($n = 105$) and Yung *et al* (2008)[4] had a follow-up of 24 months ($n = 292$). Again, these are different studies, we are not sure whether there was overlapping of samples in these two but we don't see how this would affect how good the test is at ruling in or out the risk of developing schizophrenia. The same can be said with studies by Cannon *et al*[5] and Woods *et al*,[6] the diagnostic attributes of the Cannon study were not identical to Woods' study.

Dr Mitchell raises important points regarding the predictive validity of prodromal criteria. In particular, Dr Mitchell is right to suggest that the positive predictive value and negative predictive value statistics are more intuitively informative than sensitivity and specificity, and so their reporting would have been beneficial. We also agree that assessing the clinical usefulness of prodromal criteria requires further consideration. We plan to further examine this important question in a subsequent paper. We welcome Dr Mitchell's proposal for a randomised study where high-risk patients are randomised to predicting psychosis with or without formal tests for prodromal criteria.

1 Yung AR, Phillips LJ, Yuen HP, Francey SM, McFarlane CA, Hallgren M, et al. Psychosis prediction: 12 months follow up of at risk (prodromal) group. *Schizophr Res* 2003; **60**: 21–32.

2 Yung AR, Phillips LJ, Yuen HP, McGorry PD. Risk factors for psychosis in an ultra high-risk group: psychological and clinical features. *Schizophr Res* 2004; **67**: 131–42.

3 Yung AR, Phillips LJ, Yuen HP, McGorry PD, Kelly D, Dell'olio M, et al. Mapping the onset of psychosis: the comprehensive assessment of at risk mental states. *Aust NZ J Psychiatry* 2005; **39**: 964–71.

4 Yung AR, Nelson B, Stanford C, Simmons MB, Cosgrave EM, Killackey E, et al. Validation of 'prodromal' criteria to detect individuals at ultra high risk of psychosis: 2 year follow up. *Schizophr Res* 2008; **105**: 10–7.

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6 Woods SW, Addington J, Cadenhead KS, Cannon TD, Cornblatt BA, Heinssen R, et al. Validity of the prodromal risk syndrome for first psychosis: findings from the North American Prodrome Longitudinal Study. *Schizophr Bull* 2009; **35**: 894–908.

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Abortion, mental health and charges of guilt by association

Coleman's meta-analysis of abortion and mental health studies[1] was harshly criticised in three letters by five authors (Robinson, Stotland, Nadelson, Coyne, and Littell) who all cited an *Ethics & Medicine* article[2] I wrote (not Coleman) as evidence that Coleman's study cannot be trusted. My full response[3] is summarised as follows.

First, Robinson's[4] assertion that I am Coleman's 'leader' is nonsense. We have no institutional, financial or personal entanglements. Second, I gathered data that required the analysis of research psychologists. I am thankful that Coleman agreed to analyse it and help present it in a scientifically accurate and impartial manner. As a biomedical ethicist, I explore the intersections of medicine, science, philosophy, theology, ethics and the law. When writing papers intended for each of these fields, I seek to use the language and tools appropriate to each field.

Third, the cited article was a response to a pro-life philosopher who argued that any evidence of emotional suffering of women following abortion is essentially irrelevant to the moral argument against abortion and counterproductive to pro-life efforts.[5] The core of my response was that Christians have an obligation to 'consistently demonstrate as much concern for women as for their unborn children', and that 'our advocacy for women must be consistent and unconditional both for those who are facing crisis pregnancies and for those who have had abortions'. I further argued that 'the harm abortion does to women is just as real as that done to the human fetus'.[2]

Fourth, it also reflected my sincere belief that abortion involves substantial dangers to specific subgroups of women. Unfortunately, critics have distorted this into the charge that I seek to scare women with exaggerated risks.[6] That is untrue. There are real risks, especially for certain higher-risk groups.[7] Women