known facts about psychosis, including the clear dimensionality of the risk of illness and the likely form of the heritability underpinning this, coupled with the notion of discontinuity to recognise the break in behaviour and psychological state that occurs when vulnerability translates into clinical symptoms. Importantly, the model also recognises something that Lawrie *et al* entirely ignore – the fact that psychotic traits can have a healthy expression that takes the individual outside the domain of psychiatric judgement.

Of course, many questions remain, such as how to deal with the overlap between schizophrenic and affective expressions of psychosis, explain the underlying biological mechanisms of these disorders, and incorporate into our thinking how expressions of vulnerability can vary from sick to benign. However, answers to these questions will not make dimensionality go away, for it is part of the essence of human variability (of which psychosis is one form).

On the practical front, these ideas admittedly make for a messy picture that is inconvenient for clinicians seeking a neat solution to diagnostic issues. But psychiatry does itself no favours by ignoring them and retreating (yet again) behind the ramparts of its traditional mode of thinking. Fortunately, as Lawrie *et al* will be aware, their profession actually has moved forward in recent years towards an attempt to find ways of integrating both dimensional and categorical perspectives into its future diagnostic systems. Our plea is that, in doing so, it becomes an even more 'psychologically informed' psychiatry.

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Authors' reply: We thank Drs Gordon and Shoesmith for their interest in our editorial, their complimentary remarks and their considered responses to what we said. Dr Gordon repeats our call to avoid prematurely abandoning categories or dimensions, and highlights the lack of known diagnostic biomarkers for psychosis, either as a whole or for current subtypes. Tandon *et al*[1] did not really consider this, quite reasonably, as their review focuses on what is known about the aetiology and pathogenesis of schizophrenia. As we have clarified in a forthcoming review,[2] the lack of known biomarkers for psychosis (whether as categories or continua) is at least partly because the right sort of studies to find them have only rarely been done and reported in this light. The relevant populations need to be studied and then the results analysed according to the principles of clinical epidemiology (or evidence-based medicine), to extract the potential clinical significance for individuals of statistically significant abnormalities evident in groups of patients. Thus, for example, if one wished to identify specific diagnostic markers of schizophrenia that have clinical utility, a (preferably large) representative population of people in their first episode would need to be assembled, and predictive values and/or likelihood ratios calculated for the value of potential markers of schizophrenia as opposed to, say, bipolar disorder. Despite the paucity of studies, there are already a few well-replicated large differences between people with schizophrenia and healthy controls, which may also distinguish them from those with bipolar disorder.[2] Not all of these require high-tech investigations. Simple clinical measures of neurodevelopmental aberration such as neurological soft signs, and even historical measures such as early social difficulties, are common in people who go on to develop schizophrenia but may not be in those with bipolar disorder. These already influence clinical decision-making but in an informal and rather haphazard fashion. The optimal method of eliciting and using such information needs further investigation, as outlined above and in our review.[2]

Dr Shoesmith is absolutely right to remind us that any resource-intensive diagnostic procedure is going to be much less practical in less well-developed health services. This is of course an immediate and quite possibly fatal problem for any system requiring multiple ratings on continua and could be even more so if, for example, magnetic resonance imaging of the brain/mind turns out to be diagnostically valuable – as we suspect it might.[2] In the long run, whatever turns out to be the best conceptual approach to psychosis for the maximal benefit of patients, and whether or not this has to be pioneered in leading clinical research centres, the process of formalising our diagnostic and therapeutic judgements will bring a much-needed and long-overdue re-engagement of psychiatry with the rest of medicine.

We are also grateful for the opportunity to respond to the letter from Professors Claridge and Barrantes-Vidal, especially those of us who after more than four decades still remember Professor Claridge's excellent and provocative teaching on, and seminal contributions to, the field of schizotypal cognitions, beginning as they did more than 30 years before this area became fashionable. We cite Paul Meehl as he is one of the very few commentators on diagnosis in psychiatry, whether psychologists or psychiatrists, to have offered a testable hypothesis that would allow one to make an informed decision about whether a categorical or continuous approach might be more valid. We recognise that there have been several alternative proposals to handling the complexity of psychosis, but very few of these have been tested in practice. To clarify our position, we are not opposed to continuous measures, be they psychological trait or cognitive test scores or brain imaging variables, nor are we particularly in favour of the *status quo* or hybrid models. We are simply arguing that any proposals to change our diagnostic approach to psychosis, which has survived to this day for some quite good reasons, should be based on data and therefore built on evidence rather than fashion or because something looks good on paper. We would very enthusiastically support, for example, a trial that tested the efficacy of one or more treatments on one or more continua of psychosis severity. Having said that, however, even if that trial generated informative results for clinical practice, any resulting practical system would of necessity have to include thresholds for treatment and would thereby create categories. As we said, continua may or may not be more valid than categories of psychosis, but clinical decisions require choices between alternative courses of action.

1 Tandon R, Keshavan MS, Nasrallah HA. Schizophrenia, 'Just the facts': what we know in 2008. Part 1: Overview. *Schizophr Res* 2008; **100**: 4–19.

2 Lawrie SM, Olabi B, Hall J, McIntosh AM. Do we have any solid evidence of clinical utility about the pathophysiology of schizophrenia? *World Psychiatry* 2011; in press.

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An unjust review

In his review of my book *Fiction's Madness*,[1] Beveridge comments on my omission of Laurence Sterne's *Tristram Shandy* in discussing the history of the novel form.[2] On fictional development in the 1950s, Hawthorn[3] pointedly excludes *Tristram*

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