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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Shandy as anticipating the novel and I made plain that the (postmodern) changes I observed 'came into common usage in Europe and the Unites States in the last three decades or so' (Hawthorn: p. 62). To negate (my) differentiating modernist fiction from the 1950s postmodernist 'shift' might make good criticism if not merely advanced as opinion.

On my text choices being idiosyncratic, I acknowledged this inevitability (p. vi) before providing choices of others as a balance, including David Goldberg. But this was ignored and readers left with assumptions of my eccentricity.

I did not identify psychoanalysis as a dominant force in the 1930s. I asserted its significance as an interest in Freudianism, in the 1920s, with 'think-tanks' involving John Rickman, Lionel Penrose, A. G. Tansley and John Bowlby, who qualified medically in the 1930s. This interest persisted into the 1950s, some medical superintendents being conversant with psychoanalysis whose emergent tensions, in psychiatry, I addressed in my chapter on Pat Barker's *Regeneration*.[4]

On Kafka's *Metamorphosis* being a short story: I quote acclaimed literary critic Harold Bloom:[5] 'Considering the origins of this great short novel, *The Metamorphosis*' (p. 65).

In effect, your reviewer ignored most of my book, opting for points of little intellectual interest. As for my (perceived) disparaging remarks about psychiatry 'throughout the book', my critical take on psychiatrists Dr Yealland (Chapter 3) and Dr Weir-Mitchell (Chapter 5) stemmed from fiction. My 'disparaging comments' were exceptionally sporadic but their effect clearly outweighed the rest of my text.

It is false that I 'dismiss' Nietzsche, Socrates and Foucault. I critically quoted Foucault thus: 'Shall we try reason: to my mind nothing could be more futile' (p. 66). I attributed only to Socrates that he was Plato's mouthpiece and placed my take on Nietzsche within Hesse's *Steppenwolf* and *Richard III*.

In general, the review was ill-considered, selectively dismissive and factually inaccurate.

1 Clarke L. *Fiction's Madness*. PCCS Books, 2010.

2 Beveridge A. Fiction's Madness. *Br J Psychiatry* 2010; **197**: 337–8.

3 Hawthorn J. *Studying the Novel (4th edn)*. Bloomsbury Academic, 2001.

4 Barker P. *Regeneration*. Viking Press, 1991.

5 Bloom H. *Bloom's Guides: The Metamorphosis*. Chelsea House, 2007.

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Author's reply: I would like to make the following points. First, in referring to Laurence Sterne's *Tristram Shandy*, which is regarded by most commentators as a novel, I was challenging the author's contention that: 'From the eighteenth century through to the nineteenth, novels were realist by nature [. . .] from the 1950s, however, novels began to move in mysterious ways. Suddenly "Multivoiced" narratives, unreliable narrators, allegories, genre dodging, satire, and allusiveness [. . .] became the order of the day' (Clarke,[1] pp. 11–12). Sterne's *Tristram Shandy*, written in the 18th century, and James Hoggs' *The Private Memoirs and Confessions of a Justified Sinner*, written in 1824, experiment with the genre and with the notion of the unreliable narrator. Indeed, Clarke himself (p. 17) cites Ford Madox Ford's 1915 novel *The Good Soldier* as representing a good example of an unreliable narrator.

Second, in his letter the author states that he did not identify psychoanalysis as a dominant force in the 1930s, but in his book he writes: 'Psychoanalysis was a major force in English psychiatry during the 1930s' (p. 150).

Third, as regards disparaging remarks about psychiatry, the quote about the smugness of male psychiatrists comes directly from the author, not from a novel. Elsewhere we find other critical remarks. Commenting on psychiatric training the author states: 'three years of preparation for membership of the Royal College of Psychiatrists [. . .] requires not a whit of training in interpersonal relations, little of self-reflection, or what it means to be human. Such diversions might inhibit the self-assuredness provided by a medical model of madness. Alternatively, of course, the hyped confidence may simply compensate for the psychiatrists' self-perceived fragility compared with the knowledge basis and status of other medical specialities' (p. 147).

Finally, with reference to a dismissive approach to major thinkers, the author discusses what he calls 'Socrates' infamous claim that no one can knowingly do wrong', and concludes: 'Perhaps Socrates got it wrong' (p. 156). He writes that 'Although Nietzsche's Superman (*Ubermensch*) was realised most horrifically, in our own time, by the Nazis, the impulse to stomp on others continues' (p. 136). He also observes: 'Foucault foolishly suggests abandoning rationality itself' (p. 186).

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Theories on the evolutionary persistence of psychosis

We note that the Darwinian models of psychosis reviewed by Kelleher *et al*[1] in their editorial were all variants of the 'costly by-product' evolutionary model whereby an adaptive neurobiological system that enhances fitness in the vast majority of the population generates the risk of error in a small minority, resulting in psychosis (including schizophrenia). Burns[2] identified the frontotemporal and frontoparietal cortical connections of the social brain, whereas Crow[3] proposed that the dysregulation occurs in the language centres.

We wish to propose a different and entirely environmental Darwinian formulation for the non-affective psychoses based on an 'environmental mismatch' model. We have explained elsewhere[4] that, although we agree with Burns' proposal regarding locating the dysregulation and dysconnectivity within the social brain, we contend that the aetiology of the dysregulation relates to the effects of the novel post-Neolithic social environment. Although the susceptibility to non-affective psychosis, including schizophrenia, is likely to be ancient, the schizophrenic and the non-affective psychosis phenotype did not manifest itself until very recently in our species' history. In other words, the risk of these disorders lay dormant and did not become evident until the post-Neolithic period.

Hence, we have proposed a reformulation of the social brain theory of schizophrenia and contend that schizophrenia (and the non-affective psychoses) are novel human phenomena that arose following the establishment of large permanent human settlements that accompanied the advent of agriculture and the abandonment of the hunter–gatherer way of life. We have contended that the blurring of the demarcation between in-group and out-group membership and living in close proximity to strangers is a stressor that can lead to perturbation in the development of the social brain in vulnerable individuals, resulting in the syndrome of schizophrenia. Hence, according to our formulation, schizophrenia is the result of a mismatch between the post-Neolithic human social environment and the