The classification of psychosis

Lawrie et al’s editorial on the ‘continuum of psychosis’ is timely and welcome.1 I see this debate two ways: as a doctor needing order to help ease suffering, I agree that it is better, for the time being, to keep existing diagnostic categories of psychiatric disorder, however imperfect they may be. As a patient, I of course want care, but I also want to be understood. Many psychiatrists now consider that too much of life is branded ‘disorder’: in this, none of us diminishes the reality of suffering, but we do look for better ways of explaining it. Certain scientists may hate this – but people’s lives do have narrative. I think we underestimate humankind if we say that we cannot accept symptom-based descriptions of suffering. I hope I am not wrong to suggest that most of the treatments used today to improve mental health are not disease specific, but rather act on either mood, thought or both.

Nevertheless, I agree that the cry for a spectrum approach to psychosis is premature and it does not fit with my experience of so many troubled lives encountered. Peter Tyrer is correct to raise the potential problems, both clinical and pragmatic, of premature abandonment of current diagnostic classifications.2 However, there remains a need to reconsider the neo-Kraepelinian model, if only to bring greater alignment with the technology that Lawrie et al hope will be to our greater mental good. It is my belief that, under the present classification system, neurobiological research cannot fully address complexity. My own view is that we have given too much attention to what Steven Rose3 has termed ‘neurogenetic determinism’ rather than applying biological research to life (we should not risk losing the baby with the bath water, however dirty).

I would contest the presentation of the neurobiology literature as presented by Lawrie et al in the opening paragraph of their editorial. I would also contest the claim, attributed to a paper by Tandon et al,4 that ‘advances in our understanding of aetiology and pathogenesis [of psychosis are] based on highly replicable neurological differences.’ I have read that paper several times, but found, for all the studies and indeed all the words, neither one simple biomarker of any utility nor indeed anything even approaching specificity. Perhaps we should ask why this may be? Could it be that categories, clinically practicable, and needed for now, do not match the complex epigenesis of psychosis?

In concluding, I would suggest that we do not forget history. James Clerk Maxwell was bold enough to stop looking for matter and to consider the energy fields that now govern our lives and, indeed, technology that has been to our collective good. Do we need another Maxwell moment, scientifically brilliant, religion free, willing to see matters as simple as possible, but not simpler?

Lawrie and colleagues urge us not to reject the current categorical classification system prematurely.1 I wish to add to the argument that a categorical system is more likely to be internationally useful. More than 80% of mental illness occurs in middle- and low-income countries.2 Much of the world’s mental illness is seen in overstretched clinics, by practitioners who treat up to 100 patients a day and often have had no training in psychiatry since medical or nursing school. Administering the rating scales necessary for a dimensional system may be possible in high-income countries, but is difficult or impossible elsewhere. The categorical classification system can be used quickly by someone with relatively little training. There is also the problem of translating and validating the rating scales into hundreds of languages. Most published research currently uses the same categorical system, which means that it is useful to doctors all over the world. If the research were to refer only to a dimensional system, then it would not be useful in settings where it is impossible to administer the rating scales.

The categorical system gives more people access to evidence-based treatment than any dimensional system would. A classification system that is going to be used all over the world needs to be simple and robust across healthcare systems, languages and cultures, and this is just as important as how closely it resembles the truth.


As psychologists who have long researched and argued for a dimensional view of psychosis, we would like to comment on Lawrie et al’s editorial.5 We are surprised that the authors pay no attention – with one exception – to the psychological literature. If they had done so they would know that considerable evidence supporting the continuum view has accrued over many decades. The one psychologist they do cite – the late Paul Meehl – is an unfortunate choice. Quite apart from the fact that it is unclear to us how Meehl’s taxonomic (categorical) approach actually helps their case, the authors ought to be aware that the theory is now on the wane. A more viable alternative is what we have termed a ‘fully dimensional’ theory that is capable of encompassing more of the
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 and 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.

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 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 neurocognitive 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.

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.

An unkind 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, Hawthorn3 pointedly excludes Tristram

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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.
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.


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.

Theories on the evolutionary persistence of psychosis

We note that the Darwinian models of psychosis reviewed by Kelleher et al. in their editorial were all variants of the ‘costly by-product’ evolutionary model whereby an adaptive neuro-biological 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 identified the frontotemporal and frontoparietal cortical connections of the biological 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 identified the frontotemporal and frontoparietal cortical connections of the social brain, whereas Crow 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 that, although we agree with Burns’ proposal regarding an ‘environmental mismatch’ model whereby an adaptive neuro-biological 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 identified the frontotemporal and frontoparietal cortical connections of the social brain, whereas Crow 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 that, although we agree with Burns’ proposal regarding 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.

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).
Kelleher et al\(^1\) note the significant prevalence of non-clinical psychotic symptoms in the general population and discuss some hypotheses regarding its evolutionary survival. One theory not mentioned by them or those who have so far responded is a trait known as schizotypy. While accepting that to some degree the whole topic is rich with speculation, I suggest that schizotypy may be the missing piece in the puzzle. What follows is necessarily a brief summary of some of the relevant literature.

Differing from both schizotypal and schizoid personality disorders, schizotypy\(^2\) is a heritable trait associated with an increased likelihood of creativity and of religious or mystical experiences. Importantly for this discussion, schizotypy also appears to be necessary but not sufficient for the development of schizophrenia, although not all those with schizotypy develop psychotic illnesses.

The four key dimensions of schizotypy are unusual experiences (which may be considered to be related to positive symptoms), cognitive disorganisation (related to thought disorder), introverted anhedonia (related to social withdrawal and depression) and impulsive non-conformity. This last is related to some of the disturbed behaviour, such as aggression and self-harm, seen in a range of psychiatric illnesses, including psychosis.

Regarding creativity, additional research by Nettle\(^3\) suggests that different dimensions of schizotypy are associated with different types of creativity. Nettle & Clegg further find that schizotypy is associated with increased ‘evolutionary fitness’ due to a greater number of sexual partners (and therefore offspring) in those with the unusual experience and impulsive non-conformity of the trait.\(^4\) In those with the former but not the latter dimension, the relationship with mating success is mediated by creativity. Nettle & Clegg have proposed that schizotypal traits, which in this case may be a proxy for some non-clinical psychotic symptoms, have therefore persisted because their potential negative effects are offset by enhanced mating success.

Regardless of the outcome of the search to understand the persistence of psychotic symptoms in human beings and of possible future research involving those who have the non-clinical psychosis phenotype, it is important for people working in mental health services to remember that not all those they encounter with symptoms are ill. For those that are unwell, there will be other aspects of their existence that are positive and that may be life-enhancing for them and those around them. They should be encouraged to develop these aspects of themselves as part of their long-term recovery, in addition to the treatment and support they receive from health services, carers and friends.

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By the end, Sancho Panza’s descent into these fantastic delusions is complete. So much so that at his death bed, when Don Quixote regains a measure of lucidity and tries to persuade Sancho to see reason, Sancho Panza is completely insightless and unamenable.

We are not witness to the effect of the separation of Sancho Panza from Don Quixote, as the story ends before it. But apart from that, the description of folie à deux is complete in this wonderfully told tale.


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The benefits of an active control arm

Lesem et al highlight the importance of rapid and safe treatment of agitation, indicating the delayed onset of action associated with intramuscular injection. They make no reference to the time from oral medication administration to onset of effect. However, the combination of oral atypical antipsychotics, with or without benzodiazepines, is well described. Small trials have compared the efficacy of oral atypical antipsychotics with that of intramuscular typicals and produced mean changes in rating scale scores similar to those in Lesem et al’s paper, on similar timescales.

When alternative treatments exist, placebo-controlled trials are appropriate if the target condition is characterised by a high placebo-response rate or a high relapse, remission or spontaneous resolution rate, or if existing therapies are partially effective or have high side-effect rates. Inclusion of an active control arm to the trial would have added to the number of patients required in each arm, but would have provided valuable information on the tolerability and efficacy of the inhaled or oral medication.


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Corrections

Mental disorders and termination of education in high-income and low- and middle-income countries: epidemiological study. BJP, 194, 411–417. The following funding source was omitted from the start of the list on p. 416: US National Institute of Mental Health – Mental Health Burden Study (contract number HHSN271200700030C).

The man behind Philippe Pinel: Jean-Baptiste Pussin (1746–1811). BJP, 198, 241. The DOI for this item is: 10.1192/bjp.198.3.241a. The online version has been corrected in deviation from print and in accordance with this correction.

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