Correspondence

Edited by Kiriakos Xenitidis and Colin Campbell

Contents
- The classification of psychosis
- An unjust review
- Theories on the evolutionary persistence of psychosis
- Don Quixote and Sancho Panza: folie à deux?
- The benefits of an active control arm

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 behaviour.

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-Kraepelien 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 bathwater, 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 neurobiological 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.1 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


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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.

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, we know in 2008. Part 1: Overview. Schizophr Res 2008; 100: 4–19.

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.

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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, Hawthorn3 pointedly excludes Tristram
Shandy as anticipating the novel and I made plain that the (postmodern) changes I observed ‘came into common usage in Europe and the United 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.¹


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.


Author’s reply: I would like to make the following points. First, in referring to Laurence Sterne’s Tristram Shandy, this 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 [. . . ] emerged tensions, in psychiatry, I addressed in my chapter on Pat Barker’s Regeneration.’¹

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 specialties’ (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 (Übermensch) 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).

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 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). Burns² identified the frontotemporal and frontoparietal cortical connections of the social brain, whereas Crow³ 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 elsewhere⁴ 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
Correspondence

Abed R, Abbas M. A reformulation of the social brain theory for non-conformity dimensions of the trait. In those with the unusual experiences (which may be considered to be related to positive schizotypy is associated with increased ‘evolutionary fitness’ different types of creativity. Nettle & Clegg further find that different dimensions of schizotypy are associated with success. Non-clinical psychotic symptoms, have therefore persisted because is mediated by creativity. Nettle & Clegg have proposed that but not the latter dimension, the relationship with mating success psychosis illnesses.

Kelleher et al 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, schizotypy2 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 Nettle3 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 dimensions 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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Don Quixote and Sancho Panza: folie à deux?

Martins de Barro & Busatto Filho date the first report in fiction of folie à deux to the Brazilian author Machado de Assis in 1879.1 I submit that the first fictional account of ‘shared delusions’ was by Miguel de Cervantes over 250 years before. Cervantes wrote Don Quixote de la Mancha in or around 1604, publishing the first part in 1605 and the second, a decade later.

In Don Quixote, the eponymous hero, we have a domineering and voluble fantasist driven ‘out of his wits’ by the undue influence of books of chivalry: ‘He so buried himself in his books that he spent the nights reading from twilight till day break and the days from dawn till dark; and so from little sleep and much reading, his brain dried up and he lost his wits.’2 His character is steeped in rich descriptions of grandiloquent and persecutory delusions, polymorphic hallucinations and cognitive blunting. Sancho Panza, his squire, whom he enlists as his companion for his travels, is described as ‘an honest man – if a poor man can be called honest – but without much salt in his brain-pan.’2 So we have a dominant Don Quixote, who has lost his reason, and a submissive, not so bright Sancho Panza, thrown together through much of their travels, creating a situation ripe for the development of folie à deux. And indeed we see a slow erosion of reason in Sancho Panza. He initially displays some resistance and skepticism to Don Quixote’s delusions about windmills being monstrous giants or St Benedict’s monks being a crew of wicked and diabolical ‘perfidious scoundrels’. But he increasingly becomes convinced of the veracity of Don Quixote’s beliefs. One example should suffice, the example of the balsam of Fierabras. This is a concoction that Don Quixote claims he can make on the cheap. He tells Sancho Panza, ‘If ever you see me cut through the middle in some battle [ . . . ] you have only to take the part of my body that has fallen to the ground and place it neatly and cunningly, before the blood congeals, on to the half that is still in the saddle, taking special care to make them fit exactly. Then you must give me just two drops of this balsam to drink and, you will see, I shall be as sound as an apple.’2 Sancho replies, ‘If that is so, from now on I renounce the governorship of the promised isle, and all I want in payment for all my good services is for your worship to give me the recipe for that marvelous liquor.’2

1 Don Quixote de la Mancha in or around 1604, publishing the first part in 1605 and the second, a decade later.
2 Martins de Barro & Busatto Filho date the first report in fiction of folie à deux to the Brazilian author Machado de Assis in 1879.

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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.


**The benefits of an active control arm**

Lesem *et al*1 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.2 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.3

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.4 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.

The classification of psychosis
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