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In their detailed and well-argued exposition, van Os and Reininghaus¹ identify and substantiate major problems in the intellectual structures that underpin psychiatry. In particular, by reviewing the recent great advances in our knowledge of psychotic conditions, they raise important questions about the relationship between psychiatric phenomena and defined diagnostic categories. They propose a solution that involves a radical remodelling of this relationship. I have considerable sympathy with their position and their arguments, so this commentary is by way of providing additional conceptual context and setting out the implications for advances in research strategies.

The lay concept of madness is common to virtually every society and language group. Thus, certain individuals may be identified by consensus as being in consistent, persistent and idiosyncratic error, often linked to actions perceived as incomprehensible or deeply inappropriate. The recognition that such people required help rather than exorcism or punishment meant that the phenomena of madness gradually came to be seen as the province of physicians, leading to important and enduring changes in the way these phenomena were studied. Specific aspects of madness became codified as the key symptoms of delusions (erroneous thinking) and hallucinations (erroneous perceptions), and these came to be seen as signs of one or more diseases.

Because it encapsulates the idea of disease, diagnostic classification is the central feature of the medical approach. As a branch of medicine, psychiatry was similarly built around the formulation of diagnostic categories. The division of ill-health into categories is based on the belief that this will ultimately enable the rational allotment of treatments. Disease classes (syndromes) are constructed when diligent observation identifies groups of people whose ill-health is associated with consistent and distinguishable features, that is, specific symptoms and signs. In this view, disease classes are theoretical constructs which then provide the basis for testing theories of aetiology, pathology, treatment, course and outcome². When the theories based on them are corroborated (as they often have been in general medicine), the aetiology or pathology associated with the syndromes may consequently take over as classifiers.

The construction of a disease category creates a conceptual shift. The category is thereby held to reflect an underlying disease process and so comes to be accorded an implicit causal function: it becomes the cause of the symptoms by which it is recognized. The disease process in turn is held to be the result of some fundamental cause, which may be extraneous (e.g., microbial, toxic or other physical factors) or constitutional (genetic, or genetic-environmental). This transposition in scientific focus is seen equally in physical and mental disorders. While it is a rational strategy, its success is not guaranteed.

However, disease classes are hostage to empirical evidence: their acceptance should therefore always be tentative, and they may be revised or abandoned in the light of new information (for this reason it is dangerous to accord them an intrinsic reality^{3,4}). The revision of disease categories has been a particular characteristic of psychiatric classification. Indeed, the emergence of psychosis as a preferred term in research over the last 20 years reflects dissatisfaction with narrower categorizations: affective psychosis, schizoaffective disorder and schizophrenia. This was particularly driven by the realization that virtually all psychotic disorder involves affective changes and, quite probably, similar affective mechanisms. While to purists the term psychosis may appear like an imprecise catchall, the flexibility it allows has certainly contributed to an increased knowledge of the conditions covered.

Once categories are agreed, the process of diagnosis depends on categorical judgements that individuals meet or fail to meet the requirements for membership. In psychiatry, our continuing ignorance of any causal correlates sufficient to justify an aetiology-based classification means that we are left defining classes in terms of symptoms. This is what creates the situation addressed by van Os and Reininghaus¹. In particular, a hierarchical element has traditionally been central to psychiatric classification. Thus, schizophrenic disorders are defined in terms of the presence, at some stage, of psychotic symptoms. In their absence, the diagnosis cannot be made; in their presence, the diagnosis will be made irrespective of other psychological symptoms. Schneider's first rank symptoms of schizophrenia are much vaunted in clinical psychiatry, but their significance lies in the fact that they are regarded as prima facie

indicators of schizophrenia, whatever the other psychiatric symptoms individuals might have. We choose to place schizophrenia at the apex of the psychiatric diagnostic hierarchy, for the perfectly good reason that it corresponds to the layperson's idea of madness, the psychiatric problem associated with most distress and dysfunction.

although it is However, reasonably straightforward to identify key symptoms like delusions and hallucinations, problems do arise. In particular, there are dimensional issues even with categorically defined symptoms. Thus, there is a (rational) reticence to diagnose a psychotic disorder if the psychotic symptoms are only experienced rarely, or occur singly, especially if the person is undisturbed by them and has insight. Thus, psychotic symptoms may sometimes be identified in people who fall below diagnostic thresholds, what van Os and Reininghaus call the extended phenotype. In practice, many people have a few symptoms, while only a few have many⁵.

As van Os and Reininghaus demonstrate in their review, a minor degree of psychotic symptomatology may be present in a range of other disorders, most notably affective disorder. In their terminology, these psychotic symptoms are transdiagnostic. To some extent, transdiagnostic symptomatology is an inevitable consequence of the rules placing psychosis high in the diagnostic hierarchy. It is well established that affective symptoms are widespread in the general population⁶, thus they are at least equally likely to appear in people with a diagnosis of psychosis. However, the interesting point, well substantiated by van Os and Reininghaus, is that the level of affective disturbance in people with psychosis is far higher than in the general population.

All in all, the evidence therefore suggests that there is no such thing as an event horizon in psychosis, and this must be taken into account in attempts to determine its causation. In fact, it encourages a productive paradigm shift, away from the idea that disorders cause symptoms. It fosters a view of transdiagnostic symptoms and associated psychological attributes as elements in potential causal chains, possibly linking external experience with the emergence of particular psychotic symptoms⁷. It then becomes possible to examine the interrelationship of social environmental factors and the internal features of psychosis. This endeavour is furthered by analysing symptoms in terms of correlates that are likely to influence them in distinctive ways. There is good evidence of this sort of multiple influence in paranoia, which is characteristically associated with a worry thinking style, negative thoughts about the self, increased interpersonal sensitivity, anomalous internal experiences, insomnia, and various anomalous styles of reasoning⁸⁻¹⁰. As a consequence, transdiagnostic symptoms provide rational targets for psychological therapy in psychosis.

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- 1. Van Os J, Reininghaus U. World Psychiatry 2016;15: .
- 2. Bebbington PE. Soc Psychiatry Psychiatr Epidemiol 2011;46:443-6.
- 3. Kendler KS. Psychol Med 2015; 45:1115-8.
- 4. Bebbington PE. Psychol Med 2015;45:1119-20.
- 5. Bebbington PE, McBride O, Steel C et al. Br J Psychiatry 2013;202:419-27.
- Melzer D, Tom BD, Brugha TS et al. Psychol Med 2002;32:1195-201.
- 7. Bebbington PE. Shanghai Arch Psychiatry 2015;27:70-81.
- 8. Beards S, Fisher HL. Soc Psychiatry Psychiatr Epidemiol 2014;49:1541-4.
- Bentall RP, de Sousa P, Varese F et al. Soc Psychiatry Psychiatr Epidemiol 2014;49:1011-22.
- 10. Freeman D, Garety P. Soc Psychiatry Psychiatr Epidemiol 2014;49:1179-89.