

## Jaspers was right after all – delusions are distinct from normal beliefs

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### INTRODUCTION

Jaspers' textbook *General Psychopathology* has in the past been described as the 'most important single book on the aims and logic of psychological medicine' (Shepherd, 1990). In recent years it seems to have rather fallen off the psychiatric syllabus. Its diminished importance is exemplified by changing opinions about delusions. Nowadays, delusions are considered by many to lie on a continuum with normal beliefs, and this model underpins modern cognitive therapy for psychosis (Kingdon & Turkington, 1994). Jaspers, in contrast, considered that delusions were distinct from normal beliefs. This required him to formulate a distinct mechanism of delusions that resembles, to large extent, the concepts of 'modularity' proposed by Fodor (Fodor, 1983) (who has not yet entered the psychiatric syllabus!). The continuum model suggests that delusions ought to be treatable. However, trials of cognitive-behavioural therapy for psychosis suggest significant benefit in only about half of patients treated (see Turkington & McKenna, 2003). What prevents therapeutic change along the dimensions of belief in non-responsive patients? One possibility is that Jaspers was right after all. In this month's debate the modular view of delusions is taken by Dr Hugh Jones and the case for a continuum model is argued by Professors Philippe Delespaul and Jim van Os.

### FOR

In his classic text *General Psychopathology*, Jaspers (1963) argues for a distinct mechanism for the formation and fixation of delusions – a rationalist view whereby delusions involve a change in the mechanism of belief (Campbell, 2002). Thus, in delusion, 'the awareness of meaning undergoes a transformation', described as a 'direct experience of meaning' or a 'seeing of meaning' (Jaspers, 1963). Delusions are often 'impervious to counterargument' because of a change in belief fixation that Jaspers called 'an alteration of the personality'. It is this distinct mechanism rather than any individual characteristic that distinguishes delusions from normal beliefs. This rationalist explanation, currently rather out of favour in psychiatry, may help guide our future approach to the understanding of psychosis.

Rationalist philosophy has recently become interested in cognitive modularity, and in assessing the relevance of modules to different aspects of brain function (Fodor, 1983). A module is a term for a particular cognitive mechanism and has been suggested to account for certain aspects of sensory perception. Modules are 'fast but dumb'. As with perception, their action is fast and involuntary, but 'dumb' in not being able to use all available information in performing their action. This 'informational encapsulation' may be illustrated by the well-known Muller-Lyre illusion whereby two lines with arrowheads pointing in opposite directions appear of different lengths even after they are measured and found to be equal. The modular mechanism of perceptual inference has no access to information provided by this measurement. Modules may be 'hard wired' so as to allow only certain inputs to influence their activity, thus facilitating their speed of action.

Modularity may provide a basis for understanding the suggested distinct mechanisms of delusions and normal beliefs.

Fodor (1983) has argued that normal belief formation is not modular. Normal beliefs need to be reliable and need access to all relevant information in a manner that informational encapsulation would prohibit. This reliability is not easily achieved. A computer model might exhaustively examine sense and memory data in a way that is incompatible with the normal speed of thought. Indeed, it is these requirements for feasibility and reliability, known as the 'frame problem', that have made normal belief fixation such an intractable problem in artificial intelligence. In contrast, delusions seem to have the features of a module. The fast and involuntary mechanism of a module is seen in 'the direct experience of meaning' of delusional belief formation. That delusions are 'impervious to counterargument' may reflect their informational encapsulation.

The continuum model of beliefs assumes an abnormality, not in the mechanism of belief, but in the experiences (internal or external) that give rise to beliefs. Such a view may be philosophically unsound (Campbell, 2002). For example, delusional misidentification or Capgras syndrome might result from reduced access to the emotional memories associated with this person. But while this may explain the failure to recognise the known person it cannot account for the novel belief of replacement by an impostor (Campbell, 2002).

Adopting a dimensional approach to beliefs has been used to identify psychotic-like symptoms in non-psychiatric populations. But, as Jaspers (1963) emphasised, it is not their 'oddness' or falsity that characterises delusions. People may 'believe' in ghosts but such 'cherished beliefs' do not share the same mechanism as delusions.

The idea of a 'psychosis continuum' has undoubtedly been influenced by the relationship between excess dopaminergic activity and positive psychotic symptoms. But clozapine demonstrates that this relationship is not linear. Dopamine function is not set like a 'thermostat'. One needs to consider both tonic and phasic dopamine activity and how these might alter (Moore *et al*, 1999). It is not clear that changes in such a complex system will be straightforwardly quantitative. The rationalist approach exemplified by Jaspers offers a new way of thinking about the neurobiology of psychosis.

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## AGAINST

Many people in the general population express beliefs that resemble the delusions of patients with a diagnosis of psychotic disorder. This resemblance includes similarity in not only content and factor structure, but also in demographic, psychopathological and risk factor associations (Johns & van Os, 2001). For example, non-patient individuals in the general population expressing delusional beliefs are more likely to be young and urban, report coexistent hallucinatory experiences, be consumers of cannabis and have higher levels of familial liability for psychosis.

To the extent that psychotic-like beliefs are prevalent in the non-patient population, they can be labelled as normal. The question then becomes to what degree the normal beliefs expressed by the non-patients are truly discontinuous with the abnormal beliefs expressed by patients (i.e. to what degree the differences are qualitative rather than quantitative). To assume that they are qualitatively different is to invoke some factor that allows for delusions expressed by patients to be qualified as truly different phenomena. The classic and often invoked Jasperian arguments to this effect revolve around the irreducibility of primary delusions and their subsequent incorrigibility. However, the validity of the concept of primary delusions is not compatible with the available empirical evidence (Garety & Hemsley, 1994; Bentall *et al*, 2001), and the notion has all but disappeared from the diagnostic process and assessment instruments. Similarly, a body of research has demonstrated that the degree of conviction associated with 'fixed' delusions fluctuates not only between persons, but also within persons from day to day and from environment to environment (Myin-Germeys *et al*, 2001). Even the glossary of DSM-IV-TR now states that 'delusional conviction occurs on a continuum and can sometimes be inferred from an individual's behaviour' (American Psychiatric Association, 2000: p. 821). Although delusions commonly persist for many years, research has demonstrated that it is quite common also for the non-patient population to irrationally hold onto fixed beliefs for extended periods of time. This evidence, together with the emerging effect of psychotherapeutic approaches such as cognitive-behavioural therapy, has resulted in a change in reasoning about delusions, the notion that

delusions are not modifiable by experience being replaced by one that allows for variation along dimensions.

The empirical evidence suggests that delusions can be best thought of as a multi-dimensional characteristic which varies along dimensions of conviction, distress, preoccupation and possibly bizarreness. Dramatic changes in one or several dimensions may occur over time even within the same person. In the Dunedin study, for example, a substantial proportion of non-patient children in the general population with psychotic-like ideas at age 11 years displayed true delusions in the context of a psychotic disorder at age 26 years (Poulton *et al*, 2000). It seems difficult to find valid arguments to defend the position that the psychotic-like ideas at age 10 years were qualitatively different from the true delusions at age 26 – a shift along a continuum makes more sense. Bak *et al* (2003) demonstrated that clinicians who did not recognise the quantitative aspects of delusions rejected a number of non-clinical delusional beliefs that 3 years later had evolved into a psychotic disorder. The process of delusion formation is not irreducible but instead can be traced to a multi-factorial aetiology involving an interaction, over the course of development, between a range of cognitive and emotional vulnerabilities, social circumstances and somatic factors, all of which are distributed in the general population and all of which may impact independently on one or more belief dimensions. The prediction of this model is that delusional beliefs of patients are continuous with the beliefs of non-patients, a notion that is increasingly supported by empirical evidence.

The discussion on whether or not delusions are continuous with normal beliefs relates to whether having a diagnosis of psychotic disorder is a condition that is qualitatively different from normal human experience. Evidence has recently been presented suggesting that disorders such as autism and depression are continuous phenotypes with a distribution in the general population (e.g. Constantino & Todd, 2003). The empirical, agnostic approach towards the study of delusions and hallucinations is yielding comparable findings, paving the way for a more balanced concept of the experience of psychosis. The biased and selective focus on the extremes of the psychosis distribution continues to portray schizophrenia as a chronic, incurable condition, drawing away

hope from patients and carers alike. Neither science nor clinical practice seems to be served by these qualitative distinctions.

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