

Models of misbelief: Integrating motivational and deficit theories of delusions

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Abstract

The impact of our desires and preferences upon our ordinary, everyday beliefs is well-documented [Gilovich, T. (1991). *How we know what isn't so: The fallibility of human reason in everyday life*. New York: The Free Press.]. The influence of such motivational factors on delusions, which are instances of pathological *misbelief*, has tended however to be neglected by certain prevailing models of delusion formation and maintenance. This paper explores a distinction between two general classes of theoretical explanation for delusions; the *motivational* and the *deficit*. Motivational approaches view delusions as extreme instances of self-deception; as defensive attempts to relieve pain and distress. Deficit approaches, in contrast, view delusions as the consequence of defects in the normal functioning of belief mechanisms, underpinned by neuroanatomical or neurophysiological abnormalities. It is argued that although there are good reasons to be sceptical of motivational theories (particularly in their more floridly psychodynamic manifestations), recent experiments confirm that motives are important causal forces where delusions are concerned. It is therefore concluded that the most comprehensive account of delusions will involve a theoretical unification of both motivational and deficit approaches.

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1. What are delusions?

If illusions involve low-level misperceptions of reality, then delusions involve cases of high-level *misbelief*—instances where the avowed contents of an individual's beliefs run counter to a generally accepted reality. The prevailing diagnostic view of delusions is that they are rationally untenable beliefs that are clung to regardless of counter-evidence and despite the efforts of family, friends and clinicians to dissuade the deluded individual (American Psychiatric Association, 1995).

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Delusions are observed in an array of psychiatric and neurological conditions. They have been referred to as “the *sine qua non* of psychosis” (Peters, 2001, p. 193); together with hallucinations, delusions constitute first-rank symptoms of psychotic disorders such as schizophrenia, schizophreniform disorder, schizoaffective disorder and delusional disorder. Such disorders affect around one percent of the population and have devastating consequences in terms of suffering and loss of functioning. Delusions also occur in association with dementia, temporal lobe epilepsy, Huntington’s disease, Parkinson’s disease, multiple sclerosis and traumatic brain injury.

Delusions can vary both thematically and in degree of circumscription. Thematically speaking, delusions range from the bizarre and exotic (e.g. the delusion that one’s head has been replaced by a pumpkin or that one has been raped by the devil) to the *relatively* humdrum (e.g. an unjustified conviction regarding the infidelity of a spouse, or an overwhelming suspicion of persecution by one’s neighbours). This is a nosologically important distinction, as the presence of bizarre delusions satisfies the symptom criteria for a diagnosis of schizophrenia (even in the absence of other psychotic symptoms), while precluding a diagnosis of delusional disorder.

In terms of scope, delusions vary from the circumscribed and monothematic to the widespread and polythematic (Langdon & Coltheart, 2000). A patient with “Capgras” delusion, for example, may believe that a loved one (usually a spouse or close relative) has been replaced by a physically identical impostor, yet remain quite lucid and grounded on other topics. Other individuals evince a more extensive loss of contact with reality. Nobel laureate John Nash, for example, believed not only that aliens were communicating with him, but also that he was the left foot of God and the Emperor of Antarctica (David, 1999).

2. Theoretical approaches: Motivational versus deficit

There have been many proposed theoretical explanations of delusions (for interesting reviews see Blaney, 1999; Garety & Freeman, 1999; Winters & Neale, 1983). Among the various models that have been put forward can be discerned two general classes of theoretical explanation, the *motivational* and the *deficit* (Bentall, Corcoran, Howard, Blackwood, & Kinderman, 2001; Blaney, 1999; Hingley, 1992; Venneri & Shanks, 2004; Winters & Neale, 1983). In brief, theories of the first type view delusions as serving a defensive, palliative function; as representing an attempt (however misguided) to relieve pain, tension and distress. Such theories regard delusions as providing a kind of psychological refuge or spiritual salve, and consider delusions explicable in terms of the emotional benefits they confer. This approach to theorizing about delusions has been prominently exemplified by the psychodynamic tradition with its concept of *defense*, and by the philosophical notion of *self-deception*. From a motivational perspective delusions constitute psychologically dexterous “sleights of mind” (McKay, Langdon, & Coltheart, 2005), deft mental manoeuvres executed for the maintenance of psychic integrity and the reduction of anxiety.

Motivational accounts of delusions can be generally distinguished, as a major explanatory class, from theories that involve the notion of *deficit* or *defect*. Such theories view delusions as the consequence of fundamental cognitive or perceptual abnormalities, ranging from wholesale failures in certain crucial elements of cognitive-perceptual machinery, to milder dysfunctions involving the distorted operation of particular processes. Delusions thus effectively constitute disorders of belief—disruptions or alterations in the normal functioning of belief mechanisms such that individuals come to hold erroneous beliefs with remarkable tenacity.

A deficit approach to theorizing about delusions would seem to be implicit in the field of *cognitive neuropsychiatry* (David & Halligan, 1996). Cognitive neuropsychiatry is a branch of cognitive neuropsychology, a discipline which investigates disordered cognition in order to learn more about normal cognition (Coltheart, 2002; Ellis & Young, 1988). Cognitive neuropsychiatry involves applying the logic of cognitive neuropsychology to psychiatric symptoms such as delusions and hallucinations (Ellis & Young, 1990; Langdon & Coltheart, 2000; Stone & Young, 1997). The aim of cognitive neuropsychiatry is thus to develop a model of the processes underlying the normal functioning of the belief formation system, and to explain delusions in terms of damage to processes implicated in this model of normal functioning.

Perhaps the best way to represent the distinction between the motivational and deficit approaches is to contrast a motivational account of a particular delusion with a deficit account of the same delusion. Let us take as our example the Frégoli delusion, first described in 1927 by Courbon and Fail (see Ellis, Whitley, & Luaute,

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