



Dream experience and a revisionist account of delusions of misidentification

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ABSTRACT

Standard accounts of delusion explain them as responses to experience. Cognitive models of feature binding in the face recognition systems explain how experiences of mismatch between feelings of “familiarity” and faces can arise. Similar mismatches arise in phenomena such as *déjà* and *jamais vu* in which places and scenes are mismatched to feelings of familiarity. These cognitive models also explain similarities between the phenomenology of these delusions and some dream states which involve mismatch between faces, feelings of familiarity and identities. Given these similarities it makes sense to retain that aspect of the standard account in the face of revisionist arguments that feature binding anomalies which lead to delusions of misidentification are not consciously experienced.

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1. Introduction

John Nash said of his delusions “Its kind of like a dream. In a dream it’s typical not to be rational”. His words express a common idea that dreams and delusions are both states characterised by absent or compromised “reality testing” (Bentall, 2004). This intuition is now supported by a research program which compares neural activity, cognitive processing and phenomenology in dreaming and waking (A. Hobson, 1999; J. Hobson, 1999).

This program depends on the fact that the basic cognitive *architecture* of the mind does not change during dreams. What changes in dreams are the patterns of neural activation, driven by brainstem neuroregulation, which drive processing in this cognitive architecture. Some cognitive processes, such as late states of perceptual processing, remain relatively intact in dreams, producing the characteristic stream of imagery. Others, such as logical argumentation, volitional control and planning are absent or reduced. Thus in REM dreams we experience cognitive fragments such as images and sensations juxtaposed incongruously in vignettes and scenarios rather than coherently organised in narratives or explanations (Braun et al., 1997, 1998; Desseilles et al., 2011; Domhoff, 2003; A. Hobson, 1999; J. Hobson, 1999; Manni, 2005; Roehrbach & Landis, 1995; Schwartz, 2002; Schwartz & Maquet, 2002; Solms, 2007).

This phenomenon reflects an important cognitive distinction between feature binding (the construction of a integrated perceptual or quasi-perceptual representation, Ashby et al., 1996) and contextual binding (the ability to organise such representations into a coherent metacognitive structure such as a narrative or theoretical explanation. See Revonsuo and Tarkko (2002)). Feature binding is a relatively modular process implemented in localised neural circuits whose processing properties are fairly rigid (Ashby et al., 1996; Coltheart, 1999; David, 1994). Context binding is a more flexible cognitive process dependent on the maintenance of coordinated activity in widely distributed circuitry. Consequently context binding is more vulnerable to disintegration when resources required to stabilise and synchronise the necessary distributed activation patterns are withdrawn (Cleeremans, 2003; Maia & Cleeremans, 2005).

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In this paper I apply some of these ideas to help explain the phenomenon of delusional misidentification. In these delusions people report seeing “doubles”, imposters, people in disguise, people changing appearance and identity (Breen et al., 2000; Ellis, 1991; Ellis, Luauté, et al., 1994; Ellis, Whitley, et al., 1994; Luauté & Bidault, 1994; Tzavaras et al., 1986). The phenomenology of these delusions can be explained in terms of abnormalities of feature binding, combined with abnormalities in context binding. The abnormality of feature binding produces a representation in which elements normally bound together such as face, name, autonomic response to a familiar person, and identity may dissociate (Noreika et al., 2010; Kahn & Hobson, 2003, 2005; Kahn et al., 2002). For example if a representation of a feature such as the face of a spouse is not bound to information which drives autonomic response to a familiar person the result will be an incongruity: the patient sees a person who appears familiar but the patient does not have a characteristic autonomic reaction to that person. This incongruity, produced by the failure to bind the familiar face to a signal initiating autonomic response, then leads to delusional context binding: “my wife has been replaced by an imposter”. The delusion binds the incongruous representation into a larger *explanatory* context which accounts for the fact that perception of the familiar person does not produce the “familiar” autonomic response (Ellis & de Pauw, 1994; Ellis & Lewis, 2001; Spier, 1992; Young et al., 1993).

2. The standard account

This way of putting things accords with an explanation of the Capgras delusion (of imposters or doubles) which has become standard within cognitive neuropsychiatry. This standard explanation decomposes the causal structure of delusion into two main components or “factors” which map onto the distinction between feature and context binding (Bayne & Pacherie, 2004; Breen et al., 2000; Coltheart, 2007; Davies et al., 2001; Langdon & Coltheart, 2000). The consensus is that the architecture of delusion formation includes cognitive processes which identify faces and higher order cognitive processes which *explain* abnormalities or incongruities produced by the face recognition system. The neural substrate of the former is the face recognition system, centred on the fusiform facial area. The neural substrate of higher order context binding involves prefrontal regions, especially, it is now hypothesised, right dorsolateral prefrontal cortex (A. Hobson, 1999; Coltheart, 2007; J. Hobson, 1999; Kahn et al., 2002; Wood & Grafman, 2003).

Given that dreams are characterised by activity in face recognition circuitry in the ventral visual processing stream, combined within reduced or absent activity in dorsolateral circuitry required for metacognitive context binding, some similarity in the phenomenology of both delusions of misidentification and dreams is not surprising. Equally, given that activity in the face recognition systems is evoked differently in delusions and dreams (by perceptual inputs in one case and subcortical afferents in the other) some differences are also to be expected. Similarly, dorsolateral inactivity in dreams has a different causal origin and cognitive signature to the hypoactivity or absence characteristic of delusions. So, while we might expect some similarities, we should not expect the consequent phenomenology to be identical.

Nonetheless examining similarities and differences between the two conditions might shed light on the nature of processes by which delusional beliefs arise as a consequence of activity in the face-recognition circuitry.

3. The revisionist account

In this paper I focus on a particular theory about the genesis of these delusions to which the phenomenology of dreaming is (I claim) especially relevant. That theory recently advanced by Coltheart and collaborators has two aspects. The first is that delusions are produced by processes of abductive inference: that is the production of an hypothesis which *if it were true* would explain a datum. In the case of delusions of misidentification the datum is the representation produced by unusual feature binding in the face recognition system (Coltheart et al., 2010).

This way of putting things is entirely compatible with the framework advocated above. The abductive inference is a form of contextual binding which explains anomalous feature binding in the face processing system.

My aim in this paper is not to dispute the conceptualisation of the second factor as an abductive inference. However there is another controversial aspect of the revisionist account which rejects one of the foundational ideas shared on all sides of the debate about the nature of delusion formation. That foundational idea is that these delusions are explanations of *experiences*. Coltheart et al. agree with the basic structure of the standard account but on their, revisionist, view the abnormality of face processing and the generation of the hypothesis which explains it occur *prior to conscious awareness*.

[U]nconscious processes of abductive inference are invoked to seek a hypothesis which, if true, would explain that abnormality, and a hypothesis is found which is judged satisfactory by these unconscious inferential processes. *After all of that unconscious processing has been completed, the hypothesis is accepted as a (delusional) belief, and enters consciousness.* (Coltheart et al., 2010, pp. 264–65) *My italics.*

Everything that preceded the occurrence of that belief and was responsible for the belief having come about; the stroke, the neuropsychological disconnection, the absence of an autonomic response when the wife is next seen, the invocation of a process of abductive inference to explain this and the successful generation of such an hypothesis. All of these processes are unconscious. (Coltheart et al., 2010, p. 264).

To recapitulate: on the standard account the first factor in delusion formation is anomalous feature binding in the face recognition system: for example a familiar face is not bound to activity in systems which initiate the autonomic response to familiar faces. This anomaly of feature binding produces an anomalous experience. Crudely put, something feels amiss

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