Delusions

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Abstract

A delusional belief is a belief adopted on the basis of insufficient evidence and held strongly in the face of much counterevidence. Some people with a delusional condition have a single delusional belief—this is *monothematic delusion* and much is now understood about what cause the various kinds of monothematic delusion. I describe six kinds of monothematic delusions. Other people with a delusional condition have many different delusional beliefs about a wide variety of topics—this is *polythematic delusion*, and what could be the causes of polythematic delusional conditions is still very poorly understood.

The *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision* (*DSM-IV-TR*), published by the American Psychiatric Association (APA, 2000), is a general handbook on psychopathological conditions that is widely used in psychiatry and clinical psychology. So a good place to begin a general discussion of delusional conditions might be with that Manual's definition of delusion, which is:

> "A false belief based on incorrect inference about external reality that is firmly sustained despite what almost everyone else believes and despite what constitutes incontrovertible and obvious proof or evidence to the contrary. The belief is not one ordinarily accepted by other members of the person's culture or subculture (e.g., it is not an article of religious faith)."

However, consideration of this definition soon reveals that it is of no use to scientists concerned with investigating delusional conditions, and of little use to clinicians concerned with the diagnosis and management of such conditions. Nevertheless, it does offer a good starting point for any discussion of delusion, because the various defects of this definition are instructive.

Consider, for example, the requirement that for any belief to be classified as delusional it must be false. Long ago Jaspers (1913, p. 106) used the example of delusional jealousy to show why this requirement is incoherent:

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"A delusion of jealousy, for instance, may be recognized by its typical characteristics without our need to know whether the person has genuine ground for his jealousy or not. The delusion does not cease to be a delusion although the spouse of the patient is in fact unfaithful."

The DSM-IV definition of delusion also requires that any belief that is ordinarily accepted by other member's of one's culture cannot be classified as delusional. But consider Koro, sometimes referred to as *penis panic*. Koro is a condition in which a person believes that his or her sexual organs (penis in men, vulva or nipples in women) are being retracted into the body, and that death will follow from full retraction. There are many reports from South-East Asia of this belief spreading rapidly throughout whole subcultures. For example, in just 10 days in 1967 in Singapore, 469 cases of this condition arose (Ngui, 1969); and in June to September 1982, there was an epidemic of this belief in northeastern India, in three districts of Assam and two nearby districts of Bengal, to the extent that "for a few weeks, the whole area was in the grip of a fear of the illness" (Sachdev, 1985, p. 434). It is hard to see what justification there could possibly be for asserting that Koro should not count as a delusional condition, yet that assertion follows from the DSM-IV definition.

In order for a person's belief about the infidelity of his or her spouse to be reasonably regarded as delusional, the truth of this belief is irrelevant: what's relevant is how good this person's reasons are for holding the belief. If the person cannot offer any good evidence to support a belief, and if in addition there is good evidence that this belief is false (even if in fact it happens be true), then that is the essence of what we mean by "delusional belief"; and these characteristics apply equally to an ungrounded belief in one's spouse's infidelity as to an ungrounded belief that one's sexual organs are shrinking into one's body. Similar criticisms can be raised about various other aspects of the DSM-IV definition (Davies, Coltheart, Langdon, & Breen, 2002).

A much more useful starting point for discussing forms of delusional belief is the distinction between polythematic and monothematic delusional conditions (Coltheart, 2013).

Some deluded people hold a wide variety of delusional beliefs about many unrelated topics. For example, the Nobel laureate John Nash, diagnosed with schizophrenia, believed that he was to become Emperor of Antarctica, that he was the left foot of God, and that his name was Johann von Nassau (Capps, 2004). Another well-known example of a case of a polythematic delusional condition is that of Daniel Schreber, a judge in the German Supreme Court in the nineteenth century; among the things he believed were that he was suffering from softening of the brain, that he had the plague, that divine forces were preparing him for a sexual union with God, and that this union would

Table 1
Some of the Forms of Monothematic Delusion

Type of monothematic delusion	An example of the content of the delusional belief
Capgras delusion	"That woman is not my wife, she is an impostor who looks like my wife" (the woman in question actually being the patient's wife)
Mirrored-self misidentification	"The person I see when I look in a mirror is not me, but some stranger who looks like me"
Cotard delusion	"I am dead"
Fregoli delusion	"People I know follow me around in the street, but I can't recognize who they are because they are in disguise"
Delusion of alien control	"Other people can cause my body top move without my willing such movement"
Somatoparaphrenia	"This arm (the patient's own arm) is not mine, it is my aunt's"

create a new race of humans who would restore the world to a lost state of blessedness (Bell, 2003).

These kinds of polythematic delusional cases strongly contrast with cases where the deluded person holds only a single delusional belief, or a small cluster of delusional beliefs centered on a single theme: that is, cases of monothematic delusion. Many different forms of monothematic delusion have been reported (for a review of these, see Coltheart, Langdon, & McKay, 2011). Some of these are summarized in Table 1.

In the clinic, polythematic delusion is seen much more often than monothematic delusion. Scientifically, though, monothematic delusion is far better understood than polythematic delusion. The most extensively studied monothematic delusion is Capgras delusion, named after Joseph Capgras, a French psychiatrist who first described the delusion in 1924. People with Capgras delusion believe that individuals emotionally close to them, such as family members, have been replaced by strangers who look very similar or identical to the replaced intimates. Capgras favored a psychodynamic, even Oedipal, explanation of this delusion, but seminal work by Ellis, Young, Quayle, and de Pauw (1997) clearly established that it is actually a neuropsychological condition.

Ellis and colleagues studied the responses of peoples' autonomic nervous systems (ANSs) to the sight of faces, testing both people with Capgras delusion and nondeluded psychiatric controls. The ANS is that part of the human nervous system on which arousal and emotion depend. It is known to react to the sight of faces, and reacts more strongly when the faces are familiar

ones. This is what Ellis and colleagues found in their nondeluded control patients. But in the patients with Capgras delusion, there was little response of the ANS to faces, and no greater response when the faces were familiar than when they were unfamiliar.

Now, when someone sees the face of a highly familiar person such as a spouse, they would expect a strong response of the ANS. How is a sufferer from Capgras delusion to understand the absence of a strong response when they see a spouse's face? There would not be a strong response if the person being viewed were a stranger rather than the spouse. That, Ellis and colleagues suggested, is where the idea that the spouse is actually a stranger comes from.

This seems a highly plausible account of the source of the delusional idea. But by itself it is not sufficient to explain the presence of the delusional belief, because this absence of strong autonomic response to familiar faces is also seen in people who have damage to ventromedial prefrontal cortex, the lower part of the center of the brain's frontal lobe area (Tranel, Damasio, & Damasio, 1995). Yet the patients with this form of brain damage are not delusional about the identities of their family members. Why are they not delusional when people with Capgras delusion are?

One solution to this puzzle is to propose that there is some additional cognitive impairment that people with Capgras delusion have but people with damage to ventromedial prefrontal cortex do not have. Without this second impairment, when the idea "This is not my wife" occurs, this idea does not become a belief because the evidence against this belief is so strong. If this is not your wife, how come she looks just like your wife? How come she knows details of your past life that no stranger would know? How come everyone—family, friends, and clinicians—is telling you that this woman really is your wife? Only if something has gone wrong with the patient's ability to evaluate potential beliefs with respect to evidence will the absence of autonomic response to the faces of family members turn into the delusion that these people are not family members, but strangers. It is possible (Coltheart, 2007, 2010) that this second impairment, this impairment of belief evaluation, is specifically related to damage to a particular small part of the right half of the brain, the right lateral prefrontal cortex. Neuropsychological assessments of people with Capgras delusion do generally show abnormalities of right frontal regions of the brain.

We are thus led to a two-factor theory of Capgras delusion. Factor 1 is the neuropsychological impairment that prompts the delusional idea. Factor 2 is a different neuropsychological impairment that prevents this idea from being rejected despite the strong evidence against it; instead, the idea is adopted as a belief—a delusional belief.

The Two-factor Theory of Monothematic Delusion

It has been proposed (Coltheart *et al*., 2011) that this kind of two-factor theory doesn't just offer an explanation for Capgras delusion: it offers explanations for all forms of monothematic delusion. The idea is that we study each monothematic delusion by first asking two questions:

(a) Where did the delusional idea come from—what prompted it?
(b) Why was this idea not rejected—why was it instead adopted as a belief?

Furthermore, if we want to show that two factors are always necessary if a monothematic delusion is to be present, we need to show, for each monothematic delusion, that patients have been reported where just the factor (a) for that delusion is present, and yet the patients are not delusional.

We have already seen how this two-factor theory works for the Capgras delusion. I will illustrate how the theory works more generally by applying it to the other five monothematic delusions described in Table 1.

Two patients with the mirrored-self misidentification delusion, TH and FE, were studied by Breen, Caine, Coltheart, Hendy, & Roberts (2000). Both were in the early stages of a dementing illness. Patient TH was found to have mirror agnosia—a loss of the ability to understand how mirrors work. For him, a mirror was the same thing as a window. Since any person you see through a window is in a different part of space than you, that person cannot be you. This, Breen *et al*. (2000) argued, was what prompted the idea in TH that the person he saw when he looked in the mirror was not himself, but a stranger. Patient FE did not have mirror agnosia, but he did have an impaired ability to perceive faces, which would have distorted the appearance of his face when he was looking into a mirror. So the face he saw in the mirror would not match his memory of what he looked like; and that may have been the source of the idea that the person he saw in mirrors was not himself but some stranger. Thus plausible—although different answers to question (a) can be given.

But people with mirror agnosia are not typically delusional about the identities of people they see in mirrors, and nor is this typically the case for people with impaired ability to perceive faces. So the mirror agnosia and the impaired face perception are not sufficient to explain the presence of the mirror delusion. However, both TH and FE were found to have neurological impairment of the right hemispheres of their brains, and it can be argued that this impairment compromised a cognitive system for belief evaluation (located in the right half of the brain), which prevented them from rejecting the delusional idea. It was not possible to localize their right-hemisphere brain impairments more precisely in these two patients, but in a new case of mirrored-self misidentification recently reported by Villarejo *et al*. (2011),

brain imaging revealed a lesion in the right dorsolateral prefrontal cortex, the specific brain region suspected to be associated with the cognitive process of belief evaluation. Hence, the two-factor theory of delusional belief offers a good account not just of Capgras delusion but also of the mirrored-self misidentification delusion.

Applications of this theory to explanations of the four other types of monothematic delusion listed in Table 1 are more speculative, but nevertheless eminently testable. It was suggested by Ramachandran and Blakeslee (1998) that what prompts the delusional idea in Cotard delusion (I am dead) is complete failure of the ANS to respond to any kind of stimuli, since when people are dead there is such complete failure. As Ramachandran and Blakeslee note, this theory is testable, as all one needs to do is to test whether in Cotard patients the ANS is completely unresponsive. This has not yet been investigated in any patents with Cotard delusion. But in any case, even if this is the source of the delusional idea in Cotard delusion, it cannot be the full explanation, because of the condition known as *pure autonomic failure*. People with this condition show no responses of the ANS to any kind of stimuli. But they do not show Cotard or any other kind of delusion. So there needs in addition to be the kind of belief evaluation impairment that, according to the two-factor theory, is present in all forms of monothematic delusion. Hence, this theory of the Cotard delusion predicts not only that patients with Cotard delusion will show complete failure of the ANS to respond to any kind of stimuli, but also that there should be damage to regions of the brain associated with the cognitive process of belief evaluation—presumably, damage to the right dorsolateral prefrontal cortex.

Ramachandran and Blakeslee (1998) have also proposed a speculative theory of Fregoli delusion. If the ANS, instead of being unresponsive to faces as in Capgras delusion, is over-responsive to faces, then even unfamiliar faces will feel familiar, promoting the idea that people who are actually strangers are in fact known to you—which is the Fregoli delusion. That theory is testable, by measuring how strongly the ANS responds to unfamiliar faces in people with Fregoli delusion: it should respond strongly. But even if this prediction were confirmed, the theory would not be sufficient to explain the delusion, because there are cases of people who experience the faces of strangers as highly familiar, yet who are not delusional (Vuilleumier, Mohr, Valenza, Wetzel, & Landis (2003). So once again we need to propose a second deficit, one of belief evaluation, in order to explain why a delusional idea turns into a delusional belief in some people; and, also once again, this directs our attention to assessing the integrity of right dorsolateral prefrontal cortex in people with Fregoli delusion, since it is damage here which might be associated with defective belief evaluation.

People with the delusion of alien control wrongly believe that actions they have voluntarily executed were not caused by them but by other people. How do I normally know, when I execute a voluntary action, that this action was caused by me and not by some other person? The accepted answer to this question is that when I form the intention to make a bodily movement, I create a representation of the sensory feedback that such a movement would produce, I execute the movement I intended, that generates some sensory feedback, and the received sensory feedback is compared to the predicted sensory feedback. If these match, that match is the evidence that it was I who caused the movement. If either type of feedback is for some reason not available, or if both are available but the comparison process is not carried out, the match will not be present, and the absence of this match is evidence that the movement was caused by someone else, not me (e.g., if someone picked up my arm and moved it, the match would be absent because I would not have created a representation of expected feedback). Frith (1992) proposed that in people with the alien control delusion there is impaired self-monitoring—that is, an abnormality in the system responsible for this matching process. That provides a plausible answer to our question (a) that is, it offers an explanation of what prompts the thought that someone else caused the movement of part of my body. But in the neurological disorder known as *haptic deafferentation* (Fourneret, Paillard, Lamarre, Cole, & Jeannerod, 2002), the patient cannot carry out this matching process because the disorder prevents any sensory feedback from limb movements. The patient can still execute limb movements; and, crucially, is not delusional about who caused these, even though the patient cannot determine that expected sensory feedback matches received sensory feedback. To arrive at the alien control belief here, some additional cognitive impairment would be needed—namely, a failure of accurate belief evaluation.

The sixth of our monothematic delusions, somatoparaphrenia, is typically seen only in people whose left limbs have been paralyzed because of the damage to those regions of the right hemisphere of the brain that controls movements of the left limbs. One might speculate that when a patient finds that he or she cannot move his or her left arm, the thought crosses his or her mind is that this might be because the left arm belongs to someone else. But most patients with this type of paralysis are not delusional about the ownership of their left arms, and if asked why they cannot move the arms will say that the arms are paralyzed because of the damage to the brain. What additional problem do somatoparaphrenic patients have that is responsible for the initial idea about limb ownership becoming a delusional belief? This might occur when the damage to the right half of the brain involves not just the motor-control regions of the right hemisphere, but also extends a little forward from these regions—to the right dorsolateral prefrontal cortex, which,

the idea is, needs to be intact if the cognitive process of belief evaluation is to be correctly executed.

In sum, then, bizarre though these monothematic delusional beliefs are, a great deal of progress has been made over the past 20 years in understanding what causes them to occur. In contrast, polythematic delusional conditions such as seen in the Nash and Schreber cases cited earlier remain mysterious—little insight has yet been achieved into what could possibly cause such conditions.

Much still remains to be done in the study of delusions, however. As noted above, the idea that Factor 1 in Cotard delusion is the loss of ANS sensitivity to and environmental stimuli, and that Factor 1 in Fregoli delusion is the occurrence of strong ANS responses to faces even when they are unfamiliar, are plausible but untested, so work needs to be done on ANS responding in these two types of monothematic delusional condition. The bold claim that in all types of monothematic delusion there is abnormality of right dorsolateral frontal cortex, and that this is because that region of the brain is involved in the cognitive process of belief evaluation in a variety of ways. It can be tested in delusional patients by structural or functional MRI focusing on this brain region; but this claim is also a claim about intact subjects, and so can be tested with such people. For example, with any tasks that require such subjects to evaluate beliefs, fMRI work should reveal activity in right dorsolateral frontal cortex, and disruption of the functioning of that region of the brain by transcranial magnetic stimulation (TMS) should have a specific harmful effect on the performance of such tasks.

A little work of this kind with intact subjects has recently been reported by McKay *et al*. (2013). They used a standard test for vestibular function, irrigating the left or right external ear canal with cold water. It is known that this activates regions of contralateral frontal lobe. The authors reasoned therefore that performance on tasks involving belief formation might be *improved* by left-ear (and not right-ear) irrigation. The task they used was one in which subjects estimated the likelihood that they will contract certain diseases in the future. People's belief evaluation systems function poorly here: they are unreasonably optimistic about the probability of avoiding disease. Right-ear stimulation did not reduce this unreasonable optimism, but as predicted left-ear stimulation did—that is, it made the subjects' belief evaluations more accurate. Hence, just as future work could employ TMS over right frontal lobe to impair belief evaluation in intact subjects, future work could employ left-ear irrigation to improve believe evaluation in intact subjects.

And of course, there is also much scope for future work using these two techniques in investigations of people with delusion: what effects will these techniques have on delusional beliefs? It is already clear that this is a very

promising avenue for future research, because left-ear irrigation has already been shown to transiently abolish two kinds of monothematic delusion. One of these is anosognosia for hemiplegia (the delusional belief in patients with a limb paralysis that they have no paralysis; see Cappa, Sterzi, Vallar, & Bisiach, 1987). The other is somatoparaphrenia (Bisiach, Rusconi, & Vallar, 1991).

All of the monothematic delusions discussed so far have been clearly neuropsychological in origin. But there are monothematic delusional conditions that are not, or at least not clearly, neuropsychological in origin that is, it is not clear that in such patients there has been brain damage. Hence, another particularly intriguing direction for future work is to consider whether the two-factor account of monothematic delusion might apply even in these non neuropsychological cases.

One example is alien abduction delusion: the belief that you have been abducted by beings from another planet and subsequently returned to the Earth. Coltheart *et al*. (2011, p. 11.21) offer a speculative two-factor explanation of this delusion, in which neither factor is a neuropsychological impairment.

Another example is provided by two conditions connected with body image.

Firstly, there is anorexia nervosa. It is often proposed that people with this condition overestimate how large their bodies are. Cornellison, Johns, and Tovee (2013) provide evidence that this is so; but show that it is also true of women with low body-mass index (BMI) but no anorexia nervosa. So overestimation of body-size might act as the first factor in a two-factor account of anorexia nervosa, but if that is so, then a second factor would have to be identified, which is present in people with low BMI who have anorexia nervosa and absent in people with low BMI who do not have this condition. This could be elucidated by detailed investigations of the actual beliefs about body size and body image held by these two groups of people.

Secondly there is body dysmorphic disorder, a delusional belief that some part of the affected person's body is defective or ugly when such is not at all the case. This may be the only delusion exhibited by such people. If in attempting to understand how this condition arises, one adopted the two-factor approach, one would seek answers to two questions: (i) what gave rise in the first place to the idea that some part of the patient's body is defective in some way? and (ii) as there is so much evidence against this idea(what one looks like in the mirror, what other people say) why was it accepted as a belief rather than being rejected?

It is clear how one might attempt to apply the two-factor theory to non neuropsychological monothematic delusional conditions such as these. Future work will tell us whether this can successfully be done.

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