Schizophrenia and Monothematic Delusions

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Numerous delusions have been studied which are highly specific and which can present in isolation in people whose beliefs are otherwise entirely unremarkable — "monothematic delusions" such as Capgras or Cotard delusions. We review such delusions and summarize our 2-factor theory of delusional belief which seeks to explain what causes these delusional beliefs to arise initially and what prevents them being rejected after they have arisen. Although these delusions can occur in the absence of other symptoms, they can also occur in the context of schizophrenia, when they are likely to be accompanied by other delusions and hallucinations. We propose that the 2-factor account of particular delusions like Capgras and Cotard still applies even when these delusions occur in the context of schizophrenia rather than occurring in isolation.

Key words: schizophrenia/delusion/right hemisphere/ cognitive behaviour therapy/belief evaluation

Introduction

A delusion is a belief that is poorly or not at all justified by evidence available to the believer and yet is tenaciously held by that person even when powerful evidence exists that the belief is false and when the believer's family, friends, and clinicians are providing constant affirmations that the belief ought to be abandoned because it is not true. A useful distinction can be drawn between "polythematic delusional systems" and "monothematic delusions." Someone exhibiting a polythematic delusional system exhibits a wide variety of delusional beliefs covering many different topics. Someone exhibiting a monothematic delusion possesses just a single delusional belief or at most a few such beliefs all related to a single theme.

Polythematic delusional systems are often noted in people diagnosed with schizophrenia. For eg, among the beliefs expressed by the mathematician John Nash when he was so diagnosed were that he would become Emperor of Antarctica, that he was the left foot of God on Earth, and that his name was really Johann von Nassau.[1] In contrast, in some people presenting with Capgras delusion (the belief that someone emotionally close, typically a spouse, has been replaced by a complete stranger who looks like this family member) where it is the only delusional belief apparent (as in, eg, case 1 of Frazer and Roberts[2] and in Bhatia[3], Rojo et al[4], and Burjorjee and Al-Adawi[5]), we have a monothematic delusion. Numerous different types of monothematic delusion have been documented in the literature: see table 1.

The cases of Capgras delusion just mentioned were not people with psychiatric diagnoses and, in particular, showed no evidence of schizophrenia or any other form of psychosis. But it is important to note that the extensive literature on Capgras delusion does include many cases in which the person with Capgras delusion had been diagnosed as suffering from schizophrenia. Hence, Capgras delusion can occur in the context of, but yet is not confined to, schizophrenia. Indeed, the review of cases of Capgras delusion by Edelstyn and Oyebode[7] documents cases of Capgras delusion in association with each of the following conditions: paranoid schizophrenia, schizoaffective disorder, affective disorder, Alzheimer's disease, Lewy body dementia, multi-infarct dementia, head trauma, epilepsy, cerebrovascular disease, pituitary tumor, multiple myeloma, multiple sclerosis, viral encephalitis, frontal lobe pathology, and AIDS.

This extreme etiological heterogeneity is a point to which we will return later, but first it is necessary to discuss the explanation of the Capgras delusion. This delusion was originally considered to have a psychiatric—specifically, psychodynamic—explanation. Early attempts at explaining Capgras delusion invoked the Oedipus and Electra complexes.[26] In more recent times, standard textbooks of psychiatry such as Enoch and Trethowan[27] attributed the delusion to inadequately repressed ambivalent feelings toward those whom one ought to love, such as close family members. But it is now clear that a neurobiological and a psychological approach are both necessary for explaining this delusion and that these approaches offer more promise than a psychodynamic approach. The key finding suggesting this was obtained by Ellis et al[28] Their work was based on the

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Table 1. Some Monothematic Delusions

Delusion	Example Content of False Belief	Representative Papers
Capgras delusion	"That's not my wife, it is an impostor who looks just like her"	Joseph et al[6]; Edelstyn and Oyebode[7]
Cotard delusion	"I am dead"	Young et al[8]; Young and Leafhead[9]
Fregoli delusion	"I am constantly being followed by people I know, but I can't recognize them because they are always in disguise"	De Pauw et al[10]; Mojtabaj[11]
Mirrored-self misidentification	"The person I see when I look in the mirror isn't me, it is some stranger who looks like me"	Spangenberg et al[12]; Breen et al[13]
Somatoparaphrenia	"This limb isn't mine, it is yours"	Bisiach et al[14]; Halligan et al[15]
Anosognosia for hemiplegia	"My left arm is not paralyzed"	Berti et al[16]; Davies et al[17]
Hysterical paralysis	"My left arm is paralyzed"	Marshall et al[18]; Vuilleumier et al[19]
Alien control	"Other people can control the movements of my body"	Frith and Done[20]; Stirling et al[21]
De Clerambault's delusion (erotomania)	"Person X is secretly in love with me" (Person X being some important or famous person who has never encouraged this idea)	Kennedy et al[22]; Calil and Terra[23]
Othello syndrome (pathological jealousy)	"My wife is having an affair"	Richardson et al[24]; Silva and Leong[25]

fact that when autonomic responses to faces are measured (via, for eg, skin conductance responses, SCRs), what is typically found is that appreciable SCRs are evoked by faces, even by unfamiliar faces, and the autonomic responses to familiar faces are considerably stronger. Ellis et al[28] confirmed this pattern of results in a control group of nondelusional psychiatric patients; but in 5 people with Capgras delusion (all of whom, as it happened, had received a diagnosis of schizophrenia), there was minimal autonomic responsivity to faces, even when these were familiar faces. Similar results in people with Capgras delusion have also been reported by Hirstein and Ramachandran[29] and by Brighetti et al[30] (neither of these was a case of schizophrenia). Note that face recognition itself is not seriously impaired in people with Capgras delusion: if it were, people with this delusion would not say "This woman looks exactly like my wife (but it is not her, it's some stranger)". The neuropsychological abnormality here is instead a disconnection between an intact face recognition system and an intact autonomic nervous system.

The inference seems irresistible that this disconnection is fundamental to the explanation of Capgras delusion. If in the course of people's lives they have learned that the faces of strangers generate weak or absent autonomic responses, and that such responses are strong when seeing the face of a loved one, what is a person to conclude when the face of someone who looks just like that person's spouse generates a weak or no autonomic response, rather than the expected strong response? Surely this must be the "source" of the belief that the person being seen is a stranger rather than the spouse?

But of course there is abundant evidence available to the patient that this belief is false. The spouse and other family members will be emphatic about that and indeed may even insist on tests of the belief, such as asking the spouse for details of spouse and partner's past life together, details which no stranger could possibly know about; the spouse will be able to provide these. And if the spouse has vanished, where to and why? Hence, even if the autonomic underreactivity initially prompted the thought that this is not the spouse but a stranger, this thought should rapidly be rejected rather than adopted as a belief because of the variety and strength of the evidence against such a belief. So theorists of the Capgras delusion are confronted with the following choice: either (a) the Capgras delusion is not in fact prompted by the failure of autonomic response to familiar faces, compelling as this inference may seem or (b) this failure of response is what "prompts" the impostor thought, but there is something additional at work, something which is responsible for the "failure to reject" the thought as a possible belief after it has come to mind. We will argue in favor of the second of these alternatives.

It is easy to show that a failure of autonomic responsivity to familiar faces is not sufficient to generate Capgras delusion because in people with damage to ventromedial frontal cortex, there is the same autonomic underreactivity to familiar faces, and yet there is no Capgras delusion; spouses are correctly identified as spouses.[31] If in people with damage to ventromedial frontal cortex and also in people with Capgras delusion there is autonomic underresponsivity to familiar faces, but if only the latter group are delusional, there must be some additional factor at work in just the second group which, when combined with the autonomic disconnection, is responsible for the occurrence of the delusion. But what could that second factor be?

To pursue this point, we need to consider a second kind of monothematic delusion—somatoparaphrenia. This is

the belief that some part of your body—say, your left arm—is not yours but belongs to some other person (often your neurological examiner, for example). People with sufficiently large right temporoparietal lesions will suffer paralysis of their left limbs. Some of these people will exhibit anosognosia for their hemiplegia, ie, will deny the paralysis of the left limbs, and some of these anosognosic people will attribute ownership of the paralyzed limbs to other people. The belief that your arm is someone else's arm is an example of a monothematic delusion.

Now, suppose we sought to construct a general theory of monothematic delusion that was not simply confined to the explanation of Capgras delusion but was meant to apply to the explanation of all kinds of monothematic delusion: to somatoparaphrenia, for example. Here we would need to identify something that has happened to a person with somatoparaphrenia which plausibly could have initially prompted the thought that the person's arm belongs to someone else (just as the autonomic disconnection could plausibly have prompted the impostor thought in Capgras delusion). There is an obvious candidate here: the left-sided paralysis. If your left arm actually were not yours, but someone else's, then obviously you would not be capable of moving that limb by an act of your own volition. Thus the belief in nonownership of the limb provides an explanation for the inability to move the limb.

But just as we have noted that autonomic underreactivity to familiar faces is not "sufficient" to cause Capgras delusion, so it is the case that paralysis of the left limbs is not sufficient to cause somatoparaphrenia. There are many people with left-sided paralysis caused by right temporoparietal damage who do not believe that their left limbs are not theirs and who indeed can offer the correct explanation of their paralysis—brain damage in the right hemisphere. What distinguishes left-hemiplegic people with somatoparaphrenia from these left-hemiplegic but nondeluded others?

Whatever this is, it is something to do with the right hemisphere. We can infer this because in cases of somatoparaphrenia the left hemisphere is typically intact. It is reasonable to conclude therefore that there is a region of the right hemisphere which, when damaged, prevents a hemiplegic person from rejecting the delusional belief about limb ownership that was originally prompted by the hemiplegia, despite the evidence inconsistent with this belief about limb ownership (for eg, people do not have 3 arms, but the belief that one's left arm belongs to one's neurological examiner requires acceptance of the belief that this person has 3 arms[32]). The function of this region of the right hemisphere is, therefore, belief evaluation. As we have argued above that what distinguishes patients with ventromedial frontal damage from patients with Capgras delusion is that the latter cannot correctly evaluate the belief about the spouse that

is prompted by the autonomic disconnection, it follows that people with Capgras delusion should exhibit right-hemisphere damage, and this has frequently been documented (see, eg, Edelstyn and Oyebode,[7] Bourget and Whitehurst,[33] Feinberg and Shapiro,[34] and Feinberg et al[35]).

Lesion and neuroimaging research on cases of monothematic delusion is not far enough advanced to allow anything really definite to be said about just which precise region of the right hemisphere is implicated here, but at least there is good evidence to suggest that this region is in the right frontal lobe. For eg, Papageorgiou et al[36] studied event-related potential (ERP) correlates of performance on a working memory task in people with delusions (a mixed group of 9 Capgras and/or Fregoli sufferers). What is especially interesting here is how these authors characterized the P300 ERP component they measured: it "is conceptualised as the physiological correlate of updating a cognitive hypothesis, or the working memory update of what is expected in the environment"[36(p366)]. Deciding whether or not to update a cognitive hypothesis is clearly a major component of belief evaluation. The only ERP electrode site at which the deluded patients differed from the nondeluded control group was a right frontal site, where P300 amplitude was significantly smaller in the deluded group. And Staff et al[37] compared deluded with nondeluded people with Alzheimer's disease. The 2 groups were matched on general severity of cognitive deterioration. single photon emission tomography brain imaging revealed a consistent pattern of hypoperfusion in right frontal (and limbic) brain regions in the deluded group compared with the nondeluded group.

We have thus arrived at a 2-factor theory of monothematic delusion, according to which for such delusions to occur there must be 2 abnormalities present. The first of these is what initially prompts the delusional belief and is responsible for the content of that delusion: this abnormality is different for each type of monothematic delusion. The second abnormality is what prevents the person from rejecting the belief in the light of the very strong evidence against it: this abnormality is hypothesized to be common to all varieties of monothematic delusion and speculated to arise as a consequence of damage to a belief evaluation system associated with right frontal cortex. (This 2-factor account of monothematic delusion has been applied not only to Capgras delusion and somatoparaphrenia but also to a number of other monothematic delusions, including Cotard delusion, Fregoli delusion, mirrored-self misidentification, and the delusion of alien control: for review, see Coltheart[38].)

Now we can return to the question of the association between Capgras delusion and schizophrenia. Some people with schizophrenia exhibit this delusion, but this has nothing directly to do with schizophrenia itself because

not only are there people with schizophrenia who do not exhibit Capgras delusion but also there are people with Capgras delusion who do not exhibit schizophrenia. As mentioned earlier, Capgras delusion is etiologically highly diverse: we listed 15 different neuropsychiatric or neuropathological conditions (of which 3 were psychiatric, 1 being paranoid schizophrenia) in which cases of Capgras delusion have been reported. We argue however that although Capgras delusion is etiologically heterogeneous, it is cognitively homogeneous (it is always due to the combination of the same 2 cognitive impairments, reduced affective responsivity to familiar faces plus impaired belief evaluation) and neuropsychologically homogeneous (it is always due to the combination of the same 2 neuropsychological impairments, disconnection of the face recognition system of the brain from the autonomic nervous system plus damage to a specific region of right frontal lobe). It is when the brain abnormalities of people with schizophrenia affect these 2 regions that Capgras delusion and schizophrenia will co-occur.

Although Capgras delusion and schizophrenia doubly dissociate—each can occur without the other—they do seem to be statistically associated, at least as far as the paranoid subtype of schizophrenia is concerned. Paranoid schizophrenia is characterized by the presence of prominent delusions (or auditory hallucinations) in the absence of general cognitive disorganization. Delusions are typically persecutory or grandiose, although other themes may occur. Edelstyn and Oyebode[7] in their Table 1 list 21 cases of Capgras or other misidentification delusions in people with paranoid schizophrenia. It has been claimed that right hemisphere damage is characteristic of schizophrenia; perhaps the defective evaluation of beliefs, which we have suggested occurs as a consequence of damage to a particular region of the right frontal lobe, is necessary for the occurrence even of the persecutory and grandiose delusions that are common in paranoid schizophrenia. One would need here to offer a plausible account of what might have prompted a paranoid or grandiose thought in the first place, of course, if one wanted to extend the 2-factor account of Capgras delusion and somatoparaphrenia (and the other delusions mentioned above) to delusions with paranoid and grandiose content. But this idea has at least the attractive feature that it offers an account of the association of Capgras delusion with paranoid schizophrenia: the same neuropsychological impairment of belief evaluation is required for both, and in cases of patients with persecutory or grandiose delusions where the neuropathology also has affected the pathways from face recognition to the autonomic nervous system, Capgras delusion will also be present.

Of course, this account demands that we go beyond the general finding of right hemisphere abnormalities in the brains of people with schizophrenia; it requires that people with schizophrenia who are delusional can be demonstrated to have right frontal damage. We are not aware of any investigations of right frontal lobe functioning in schizophrenia, which have specifically compared delusional vs nondelusional patients; but studies comparing schizophrenia patients with healthy controls have yielded abundant evidence for specific right frontal deficits. Structural magnetic resonance imaging (MRI) work has indicated right but not left frontal hypergyria[39,40] and white matter deficits in the right but not left frontal lobe[41] associated with schizophrenia. When schizophrenia patients are performing the Wisconsin Card Sorting Task, a test of frontal lobe functioning, functional MRI reveals reduced right frontal but not left frontal activation in the patients compared with normal controls.[42] Regional cerebral blood flow measurements have detected abnormal metabolism in right but not left frontal lobe in schizophrenia patients.[43,44] magnetic resonance spectroscopy has revealed correlations between concentrations of phosophodiesters and phosphocreatine in right frontal lobe and the hostility-suspicion and anxiety-depression subscale scores of the Brief Psychiatric Rating Scales (BPRS), but not the other 3 BPRS scales,[45] with no BPRS correlations for left frontal lobe concentrations. ERP work has shown a specific reduced amplitude of late-latency gamma-band responses over right but not left frontal scalp regions in schizophrenia.[46]

It would be of value to know whether these selective right frontal abnormalities are specifically associated with the presence of delusions. Single case studies are of value here; for eg, it is hard not to be impressed by reports such as that by Silva et al.[47] The patient they describe had a diagnosis of paranoid schizophrenia and presented with the delusion that he was Jesus Christ. The report of CT scanning identified just 1 region of abnormality in this patient's brain: a small region of hypodensity in the right frontal lobe.

Treatment for Delusions

The 2-factor theory of delusional belief has considerable plausibility, but it is certainly not without its problems; these problems and how they might be solved are discussed by Coltheart,[38] and one is especially relevant here. There are many reports of monothematic delusion in which waxing and waning of the delusion is noted. For eg, there are cases of Capgras delusion in which the affected person complains about his wife being an impostor on some occasions while on other occasions accepts her as genuinely his wife (Coltheart[38] describes 1 example of this). How could this be explained if Capgras delusion requires the presence of a pair of neuropsychological deficits which themselves must, presumably, be permanently present?

The speculative solution offered by Coltheart[38] runs as follows. It is extremely common in cases of monothematic

delusion for family members of the deluded person to be distressed about the delusion and to try to get rid of it by constantly challenging it. Suppose that the deluded person's defective belief evaluation system is just that: defective rather than destroyed. The evidence of the senses (the failure of autonomic response to the spouse's face in Capgras delusion; the paralysis in somatoparaphrenia) supports the delusional belief to a constant degree; the evidence against the belief mounts up over time as more and more of it is provided by the family. Eventually, even a defective belief evaluation procedure might accept that the weight of evidence is against the belief. So the belief is rejected, ie, the delusion is abandoned. When this happens, the family has no further need to continue to provide evidence against the delusional belief, but the senses continue to provide evidence in favor of it, and so eventually it returns. Might this be the mechanism operating in those cases where a delusional belief comes and goes?

There is semianecdotal evidence consistent with this account. There are 2 ways families of deluded people go about trying to dispose of the delusion. One is direct challenge: pointing out to the deluded person the absurdity of the belief. The psychological threat this engenders may be the reason for the high incidence of violent behavior by Capgras sufferers, which sometimes stretches to murder, typically of the putative impostor (for review, see, eg, Bourget and Whitehurst[33] and Förstl et al[48]). The other way is indirect persuasion by persistent tactful offering of evidence that is inconsistent with the delusional belief. One example was given above: suggesting to the deluded person that he interrogate the person he believes to be impersonating his spouse about his and the spouse's past life together. Another example is described by N. Breen (personal communication, 2000). This involved a man who believed not that his wife was a stranger but that she was a person he had known from his past life, a former business partner; this delusional belief arose after a serious head injury (for details of this case see Breen et al[49]). A number of efforts were made by Breen (a clinical neuropsychologist) to gently persuade this person to test out the possibility that the woman he was with was in fact actually his wife. It was pointed out to him, for eg, that the woman was wearing a wedding ring exactly like the one he had described as having been bought by him for his wife. When he offered the interpretation that what must have happened was that the former business partner had bought another of these rings from the same shop, he was shown the initials engraved inside the ring—which were the initials of his wife, not his former business partner—and when asked explain how this could have come about, he could not offer an explanation. Within a week of this kind of benign probing, he had abandoned the delusion and accepted that the woman in question was in fact his wife.

This kind of procedure is of course exactly what is prescribed in cognitive behavior therapy: "cognitive-behavioral therapy for delusions involv[es] engagement, the building of trust, discussing a range of explanations for the delusional beliefs, and reality testing (eliciting examination of evidence, logical inquiry, and reasoning)".[50] We therefore consider that our 2-deficit account of monothematic delusion, in particular the idea of how a defective but not destroyed belief evaluation system might be able to respond appropriately if provided with sufficiently strong evidence, offers a theoretical basis for the use of cognitive-behavioral therapy for the treatment of delusional beliefs, including when these are seen in the context of psychosis.

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