Monothematic Delusions: Towards a Two-Factor Account

Davies, Martin, 1950-
Coltheart, Max.
Langdon, Robyn.
Breen, Nora.

Philosophy, Psychiatry, & Psychology, Volume 8, Number 2/3, June/September 2001, pp. 133-158 (Article)

Published by The Johns Hopkins University Press
DOI: 10.1353/ppp.2001.0007

Monothematic Delusions: Towards a Two-Factor Account

Martin Davies, Max Coltheart, Robyn Langdon, and Nora Breen

Abstract: We provide a battery of examples of delusions against which theoretical accounts can be tested. Then we identify neuropsychological anomalies that could produce the unusual experiences that may lead, in turn, to the delusions in our battery. However, we argue against Maher's view that delusions are false beliefs that arise as normal responses to anomalous experiences. We propose, instead, that a second factor is required to account for the transition from unusual experience to delusional belief. The second factor in the etiology of delusions can be described superficially as a loss of the ability to reject a candidate for belief on the grounds of its implausibility and its inconsistency with everything else that the patient knows, but we point out some problems that confront any attempt to say more about the nature of this second factor.

Keywords: belief, experience, cognitive neuropsychiatry, misidentification delusions, schizophrenia, affective nonresponse, attributional biases, mirrored self

Introduction: Definition and Examples

Amongst several entries for the word "delusion," the *Oxford English Dictionary* (1989) lists this:

3.a. Anything that deceives the mind with a false impression; a deception; a fixed false opinion or belief with regard to objective things, *esp*. as a form of mental derangement.

There is more than one idea here, and the definition offered by the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*) seems to be based on something similar to the second part of the *OED* entry:

Delusion: 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 (American Psychiatric Association 1994, 765).

There is much in these putative definitions that is intuitively helpful for the psychological study of delusions, but equally, they raise many questions. The *DSM* definition prompts us to ask at least the following:

- Is a delusion always a belief? Might some delusions have some but not all of the characteristic properties of a belief? For example, Currie (2000) suggests that some delusions may actually be imaginings that the delusional subject misidentifies as beliefs.
- Does the belief have to be false? It may indeed be part of the ordinary meaning of the term that a delusion must be false. But if a true belief is sustained in just the same way as a delusion is sustained, despite its implausibility and in the face of all the available evidence, then it seems that, for the purposes of psychological theory, it should be grouped together with delusions.

The definition of delusions

The clinical literature suggests that conventional definitions of delusions have weaknesses. For instance, *DSM-IV* defines delusions as "false" beliefs. However, many authors have challenged the falsity criterion.

We have drafted two definitions, one using the person's subculture as the reference point for deciding on the presence of a delusion (Definition One) and the other attempting to use aspects of the form and content of the belief to make this decision (Definition Two).

Please write your comments about each of these definitions in the spaces provided and, after this, draft the definition you use in clinical practice.

Definition One: A delusion is a belief not in keeping with the beliefs held by persons of the same cultural and socioeconomic background. The evidence supporting it is frequently inadequate or bizarre. The belief is resistant to contradictory argument or evidence that would be accepted by a person of equivalent cultural and socioeconomic background. Irrespective of the plausibility or bizarreness of the delusion, it is held with a strong sense of conviction at least some of the time.

Definition Two: A delusion is a problematic belief that is highly personalised or idiosyncratic. Though the content of the belief may have some understandable link to the person's psychological background, it is out of keeping with the holder's knowledge or experience. Usually the evidence offered in support of the belief is inadequate, overly personal, or bizarre. The belief has the quality of being self-evidential and unfalsifiable (irrefutable) either because of the nature of the belief or because of the refusal of the holder of the belief to admit anything that would falsify the belief. Irrespective of the plausibility or the bizarreness of the belief, it is held with a strong sense of conviction and reality most of the time.

Figure 1: The PORS questionnaire on the definition of delusions

- Does the belief have to be based on inference? A subject might arrive at a delusional belief simply by taking a misleading perceptual experience as veridical. Forming a belief in this way does not obviously involve a step of inference.
- Does the belief have to be about external reality? Some delusional beliefs concern the subject's own body or even the subject's own thoughts.
- Does the belief have to be different from what almost everyone else believes? If a bizarrely implausible belief is formed and sustained in ways that are characteristic of delusions, then it seems that, for the purposes of psychological theory, it should be grouped together with delusions even if many other subjects believe the same thing.

There is research aimed at improving on the *DSM* definition. In Figure 1, we display part of a questionnaire that is being used by Stanley Catts and a research team at the University of New South Wales to elicit responses to two draft definitions of delusions.[1] However, it is a highly non-trivial task to provide a definition that meets the needs of both psychological theory and clinical practice. Here, we shall not attempt to refine the definition of delusion. We shall simply assume that delusions are beliefs and proceed to some examples.

Ancient Examples: Two cases of Misidentification

Ancient literature provides many stories of misidentification, including the horrific case of Agave who is tricked by the god, Dionysus, into murdering her own son, Pentheus, under the impression that she is killing a lion (see Way 1988: *Bacchae*, lines 1277–78, 1281–83).

Cadmus: "Whose head—*whose?*—art thou bearing in thine arms?"
Agave: "A lion's—so said they which hunted it . . . ."
Cadmus: "Gaze, gaze on it, and be thou certified . . . . Seems it to thee now like a lion's head?"
Agave: "No!—Wretched!—Wretched!—Pentheus' head I hold!"

Rather similarly, Hercules is duped by the demon, Lyssa, into killing his own sons, thinking that they are the sons of Eurystheus (see Way 1988: *The Madness of Hercules*).

In both of these examples, the misidentification arises from misperception; the protagonist is deceived by the gods. This constitutes a delusion according to another of the *OED* entries: "The fact or condition of being cheated and led to

believe what is false." It is not so clear, however, that these are misidentification *delusions* in the modern sense of the term, for a false belief that arises from misleading experience is not necessarily a delusion. Perhaps our intuitive judgments on these two cases are conditioned by the fact that there is nothing available to Agave or to Hercules that "constitutes incontrovertible and obvious proof or evidence to the contrary." In fact, Hercules's wife and child do try to persuade him that they are his wife and children, but he seems oblivious. To this extent, his case seems closer to a delusional belief.

Varieties of Delusion: Monothematic or Polythematic; Circumscribed or Elaborated

A *monothematic* delusion is simply one that is specific to a particular topic. As Stone and Young (1997) note, monothematic delusions that result from brain injury are often also *circumscribed* or unelaborated. Monothematic and circumscribed delusions can be contrasted with the *polythematic* and *elaborated* delusions or delusional systems that are characteristic of some schizophrenic patients. The following case description refers to a patient who was interviewed by Robyn Langdon. The patient had polythematic and elaborated delusions, some of which were interwoven and some of which stood apart. The delusional themes included somatic concerns, paranoia, ideas of reference, grandiosity, and loss of boundary experiences.

A Thirty-One-Year-Old Woman with Chronic Schizophrenia. The patient had been ill for seven years. At the time of the interview, she reported olfactory and somatic hallucinations but no auditory or visual hallucinations. She noticed an occasional unexplained powdery smell about her body—something like the smell of baby powder—and was distressed by the experience of physical blows raining down on her head on a daily basis. Marked paranoia was present; she avoided all contact with her family, believing that they would harm her if they knew of her location. She also avoided public places, being generally distrustful of other people. She believed that people followed her with their eyes and gossiped about her whenever she went out in public. The only places that she was prepared to frequent on a relatively regular basis were the gym (at times when other people were unlikely to be present) and a local church that she had started attending. She had a history of somatic concerns. At the time of the interview, she was slightly concerned that her genitals had been changing in shape and size but was not particularly distressed, because the changes had appeared to stop with everything going back to normal since she had resumed going to church. She was also keeping an eye on one of her teeth; there was something special about this tooth. At the time of the interview, she did not quite know what this something special was but was sure that if she kept watching the tooth, its importance would become apparent; it was going to be a special sign for her. She also described a special freckle on her body; this was a freckle that God had made especially for her to mark her as his chosen. She believed that she had a special relationship with God; God had stepped in to save her from her enemies, and this was because she had a mission. That was why she had returned to the church: to find out about her mission for God. Finally, the patient reported thought broadcast delusions; she believed that her thoughts seeped out of her head like signals that could be understood by other people. This meant that she had to be very careful about what she thought; she should only think good thoughts.

Just as polythematicity and elaboration cooccur in the delusional systems of some schizophrenic patients, so also monothematicity and circumscription tend to go together, but it is worth noting that monothematicity and circumscription are, strictly speaking, independent of each other. In particular, a neurological patient may elaborate a monothematic delusion to some extent. Furthermore, although psychiatric patients often have polythematic and elaborated delusional systems, they sometimes have delusions that are monothematic and at most only somewhat elaborated. Here are two more descriptions of patients who were interviewed by Robyn Langdon. The first case is an example of an individual with a more or less circumscribed monothematic delusion:

A Twenty-One-Year-Old Man with Psychotic Symptoms. The patient had started presenting with psychotic symptoms two years earlier during his second year at university. At the time of the interview, he was receiving, and responding well to, medication. His only concern was the occasional recurring thought that he was physically unwell. In the past these concerns had developed into a fervently held hypochondrial delusion that he was diseased and would die from this disease; this despite doctor after doctor failing to find anything at all wrong with his body. Now, he said, he was trying to stop these thoughts from getting a firm hold by using reality testing. No other symptoms were present.

The second case is an example of an individual with a monothematic but somewhat elaborated delusional system:

A Thirty-Three-Year-Old Man with Chronic Schizophrenia. The patient had been ill for fourteen years. At the time of the interview, he was preoccupied and distressed by the firm belief that he had no internal organs. Although his doctors had told him that this was a physiological impossibility, and despite some acknowledgement on the part of the patient that he could not quite understand how such a thing was possible, the patient said that he could not rid himself of the belief. The patient also expressed the belief that spirit doctors had come to his room one night to perform a magical operation in order to remove his internal organs. This happened, he believed, because he was being punished by God for some evil or sin that he had committed, although he was uncertain about the nature of that sin. The most distressing aspect of the delusion for this patient was the pervasive worry that, when he died, he would be rejected from heaven because he was no longer a proper human being.

Modern Examples: Eight Monothematic Delusions

We now present a battery of eight monothematic delusions against which a theoretical account of delusions might be tested.

(1) The first is the Capgras delusion: "One of my closest relatives has been replaced by an impostor." Although it is often remarked that the Capgras delusion usually concerns someone close to the patient, Ellis and de Pauw (1994, 320) note, "Clearly the [Capgras delusion] does involve the belief that some individuals have been replaced by doubles or robots. But these are not necessarily people in close relationship with the patient. Often neighbours, nursing staff, doctors, and other patients are the objects of what some have termed hypoidentification." This is one of several considerations that tell against a psychodynamic account of the delusion. One patient investigated by Young et al. (1993) said, "There's been someone like my son's double which isn't my son . . . . I can tell my son because my son is different . . . but you have to be quick to notice it." The first major report of the Capgras delusion was in 1923, and the delusion is not especially uncommon. There are hundreds of published cases (Capgras and Reboul-Lachaux 1923; Berson 1983; Signer 1987; Förstl, et al. 1991).

(2) The second delusion in our battery is the Cotard delusion: "I am dead" (Cotard, 1882; Berrios and Luque 1995). It is worth noting that, in fact, only one of Cotard's original eight pure cases involved the delusional belief that the patient herself was dead; more common were self-deprecatory delusions and negation of self (see Young and Leafhead [1996] for discussion). Young et al. (1992) describe the case of a patient, WI, who, following a head injury, became convinced that he had died. WI's delusion was elaborated to the extent that, based on the heat in South Africa where he was traveling, he also believed that he had been taken to hell.

(3) The third delusion is the Frégoli delusion: "I am being followed around by people who are known to me but who are unrecognizable because they are in disguise" (Courbon and Fail 1927; de Pauw et al. 1987; Ellis et al. 1994).

(4) The fourth is mirrored-self misidentification: "The person I see in the mirror is not really me" (Breen et al. 2000b).

(5) The fifth delusion in our battery is reduplicative paramnesia (Breen et al. 2000b). A patient, DB, who had suffered a right parietal stroke, affirmed that her husband had died and was cremated four years earlier (which was true) and that her husband was a patient on the ward in the same hospital that she was in (which was not true). DB seemed to have two separate, and inconsistent, records or dossiers for her husband.

(6) The sixth delusion is one that is sometimes found in patients with unilateral neglect. Unilateral ne-

Table 1. Eight monothematic delusions

Capgras delusion
Cotard delusion
Frégoli delusion
Mirrored-self misidentification
Reduplicative paramnesia
Unilateral neglect
Alien control
Thought insertion

glect occurs in people who have had major damage to one hemisphere of the brain (almost invariably the right hemisphere). Since the right hemisphere controls the left half of the body, these patients normally have paralyzed left arms and left legs. They show a marked deviation of eyes, head, and trunk away from the left side of space as if they are captivated by the right side of their world. They are liable to collide with objects on their left side, they leave the food on the left half of their plates, they do not dress or clean their left limbs, and they do not respond to conversation if the speaker is to their left. Some patients with unilateral neglect actually deny ownership of their left arm or left hand, even when it is placed so that they have no difficulty attending to it. In one striking case reported by Edoardo Bisiach, the patient (PR) insisted that his left arm was the examiner's and accepted the consequence that the examiner had three arms (Bisiach 1988, 469. For further discussion of PR, see Bisiach and Geminiani [1991, 34]. For further examples of denial of ownership of a contralesional limb, see Critchley [1953]. For reviews of unilateral neglect, see Bisiach and Berti [1995] and Vallar [1998]). Bisiach and Geminiani (1991, 32–33) also provide a vivid description of a patient, LA-O, who denied ownership of her left hand:

> On request, she admitted without hesitation that her left shoulder was part of her body and *inferentially* came to the same conclusion as regards her left arm and elbow, given, as she remarked, the evident continuity of those members. She was elusive about the forearm but insisted on denying ownership of the left hand, even when it had been passively placed on the right side of her trunk. She could not explain why her rings happened to be worn on the fingers of the alien hand.

The final two delusions in our battery are characteristic of schizophrenia (Frith 1992):

(7) The seventh is the delusion of alien control: "Someone else is able to control my actions. I am a puppet and someone else is pulling the strings."
(8) The eighth is the delusion of thought insertion: "Someone else's thoughts are being inserted into my mind."

Since we are offering a battery of monothematic delusions, we should note that "loss of boundary" delusions, including alien control and thought insertion, often co-occur with other delusions in schizophrenia. However, they can also be found in isolation. In this paper, we are mainly concerned with monothematic delusions that result from brain injury, but we hope that our account can be extended to cover delusions, particularly monothematic delusions, arising in psychiatric patients.

We end this introductory section by noting the incidence rate of delusional beliefs. Approximately one person per hundred will be diagnosed as having schizophrenia. Since delusions are first-rank markers of schizophrenia, virtually all these patients will have delusional beliefs. Other psychiatric patients who experience delusions might be diagnosed as having schizoaffective disorder, affective disorders with psychotic episodes, or schizophreniform disorder. The incidence of delusions as a result of brain injury is lower than the incidence of schizophrenia, but overall, we can take it that, at a minimum, between one and two people per hundred will have delusional beliefs at some time in their lives.

1. Delusions and Experience

Brendan Maher (1974, 1988, 1992, 1999) famously defends the view that delusions are false beliefs that arise as normal responses to unusual experiences. (Maher has been primarily concerned with the role of anomalous experiences in the etiology of delusions in schizophrenia.) A recent article provides an outline of his account (Maher 1999, 550–51):

- Delusional beliefs, like normal beliefs, arise from an attempt to explain experience.
- The processes by which deluded persons reason from experience to belief are not significantly different from the processes by which non-deluded persons do.
- Defective reasoning about actual personal normal experience is not the primary contributor to the formation of delusional beliefs.
- The origins of anomalous experience may lie in a broad band of neuropsychological anomalies.

1.1 Maher's Account of Primary Schizophrenic Delusions

Maher suggests that the delusions that Jaspers (1963) called *primary*, as well as those that he called *secondary*, can be brought within the scope of this general account. (See Maher [1999, 552]: "I hope to show that Jaspers's distinction between primary and secondary delusions can be satisfactorily encompassed with the single model already described.") Jaspers's account of primary delusions is problematic in a number of respects (see Eilan 2000), but the basic idea is that a primary delusional belief does not arise from earlier psychological events in a way that is intelligible from the subject's point of view; it is psychologically irreducible and incomprehensible. A secondary delusion, in contrast, emerges in a comprehensible way from other psychological events or, as Jaspers puts it, "out of a given personality and situation." He includes among secondary delusions, or "delusion-like ideas," "the transient deceptions due to false perception, etc." (Jaspers 1963, 107) Consequently, it would be natural to classify as secondary a delusion that arises from an attempt to explain an unusual perceptual experience.

Given this contrast between primary and secondary delusions, it is initially surprising that Maher should seek to apply his account to primary delusions, but Jaspers also speaks of a "general delusional atmosphere" (Jaspers 1963, 98) and says:

> Delusions proper [primary delusions] are the vague crystallizations of blurred delusional experience and diffuse, perplexing self-references which cannot be sufficiently understood in terms of the personality or the situation; they are more the symptoms of a disease process (Jaspers 1963, 107).

In line with these and other remarks, Maher proposes that the subject of a primary schizophrenic delusion experiences *feelings of significance* that are endogenously generated but may become attached to aspects of the subject's mental life, such as thoughts and images, and to perceived features of the external environment.

A vivid description of this kind of experience is provided by a schizophrenic patient, Norma MacDonald:

> I became interested in a wide assortment of people, events, places, and ideas which normally would make no impression on me . . . .I . . . felt that there was some overwhelming significance in all this, produced either by God or Satan, and I felt that I was duty-bound to ponder on each of these new interests, and the more I pondered the worse it became. The walk of a stranger on the street could be a "sign" to me which I must interpret (MacDonald 1960, 218).

Roderick Anscombe, who has compiled a rich collection of patients' descriptions of schizophrenic experience, comments: "The significance is spurious, but the patients feel impelled to react by making sense of it in some way" (Anscombe 1987, 249).

On Maher's account, a subject may offer a delusional explanation for a generalized and persistent feeling of significance (Maher 1999, 560): "[I]f no specific concrete change can be found, and the feeling of significance persists, everything must have changed in some fundamental way." He continues, "If everything in the environment is significant, the significance is likely to be widespread and profound. Themes of world dissolution, that people have all become robots, or are the 'walking dead,' that the individual has himself died and is looking at the world from beyond life, are some of the common possibilities." Alternatively, it may be that when a subject experiences these feelings of significance, an apparently significant train of thought and its associated imagery come to provide content for a delusional belief. Thus Anscombe, who suggests that feelings of significance are the result of a disorder of attention, says that "certain of the person's own thoughts may be imbued with a significance that is out of proportion to their real importance, simply because they happen to cap-

ture the attentional focus" (Anscombe 1987, 252). In either case, the delusional belief then gives meaning to the sense of significance that attaches to perceived features of the environment.

Maher illustrates how this account of the etiology of primary delusions works by considering the classic example of "a patient who looked at the marble tables in a café and suddenly became convinced that the end of the world was coming" (Maher 1999, 559; the example is from Arthur 1964, 106). For this patient, the experience of seeing the marble tables was full of significance, shot through with meaning about the ending of the world, but, Maher suggests, there was nothing anomalous about the visual experience itself; there was, for example, no abnormality in visual information processing. Thus it is not that the visual experience had some unusual property that figured as a causal factor in the generation of the delusion. Rather, the delusion arose in some way from feelings of significance. Then, as a causal result of the presence of the delusional belief with its specific content, a particular significance came to be attached to the perceptual experience of seeing the marble tables. John Campbell (this issue) describes this case as involving a "top-down loading of the perception by the delusional content . . . ."

If we focus on the visual perceptual experience of the marble tables, then the fact that the significance attached to that experience was causally posterior to the delusional belief makes it initially surprising that Maher should include this kind of example within the scope of his account. However, what Maher stresses in this case is not the causal role of the visual *perceptual experience* but rather the causal role of the *feelings of heightened significance* that coincided with viewing the marble tables:

> The delusional meaning comes as "given" by the combination of experience [of feelings of significance] and other coincidental elements of consciousness, but it is the experience [of feelings of significance] that creates the combination; it is not the delusional explanation that creates the experience (Maher 1999, 561).

In general, the anomalous experience that figures in the etiology of primary schizophrenic delusions, on Maher's account, is not a perceptual experience; it is a compelling, generalized, and persistent feeling of significance.

Lesser feelings of significance—experienced as the sense that something has changed, that someone is familiar, that some event was surprising, or that something wonderful is about to happen—occur in everyday life (Maher 1999, 552-58). A vague sense that something has changed, for example, normally prompts a search for a specific difference, and it is a common enough occurrence that such a search may end in a quite false view about what it is that has changed. When the feeling of significance is so much more compelling, generalized, and persistent, is endogenously generated, and co-occurs coincidentally with various thoughts, images, and perceptual experiences, bizarrely false beliefs may arise and may then condition other aspects of the subject's experience.

This account of primary delusions does not, of course, rule out the possibility that in other cases, feelings of significance, of difference, or of familiarity might attach directly to particular perceptions of objects in the environment. The resulting experiences could then figure in the etiology of delusions that would be classified as secondary. The specific contents of those delusional beliefs would be intelligible in the light of the content and nature of the antecedent experiences.

Maher's account of primary schizophrenic delusions is also entirely compatible with the idea that delusions may arise in an intelligible way from unusual experiences that result from brain injury. Indeed, he says

> [D]elusional interpretations of circumscribed anomalies of experience arising from psychopathology are not confined to schizophrenia . . . .[T]he model of delusion formation . . . posits that the basic origin lies in the anomalous experience, regardless of how that anomaly arose (Maher 1999, 566).

1.2 Anomalous Experiences in the Etiology of Monothematic Delusions

For the purposes of the argument in this paper, we agree with Maher that unusual experiences are one factor in the etiology of monothematic delusions and particularly of delusions that result from brain injury. Our main claim is that

there must be a second factor as well. The argument for this claim comes in Section 2 (below). In the remainder of this section, we focus on the point of agreement. Is it possible to identify experiences that could figure in a psychologically intelligible way in the etiology of the eight delusions in our battery?[2]

The Capgras Delusion

Hadyn Ellis, Andy Young, and colleagues propose that the Capgras delusion ("One of my closest relatives has been replaced by an impostor") arises from a deficit in face processing that is a kind of mirror image of prosopagnosia (see, for example, Ellis and Young 1990). The face recognition system is intact, but there is a loss of affective responses to familiar faces.[3] This is a neuropsychological explanation of the delusion, not a psychiatric explanation. It has to do with the way our bodies respond emotionally to familiar faces compared with the faces of strangers. According to Ellis and Young, the Capgras patient has an experience of seeing a face that looks just like a close relative (usually the spouse), but without the affective response that would normally be an integral part of that experience. This hypothesis about the patient's experience is supported by the finding that Capgras patients have a reduced galvanic skin response to faces and in particular do not respond more to familiar faces than to unfamiliar faces (Ellis et al. 1997; Hirstein and Ramachandran 1997). Brain damage has disrupted the connections in the patient's brain between the face recognition system and the autonomic nervous system. The delusion can then be explained in terms of the patient's trying to make sense of this peculiar experience:

> When patients find themselves in such a conflict (that is, receiving some information which indicates that the face in front of them belongs to X, but not receiving confirmation of this), they may adopt some sort of rationalizing strategy in which the individual before them is deemed to be an impostor, a dummy, a robot, or whatever extant technology may suggest (Ellis and Young 1990, 244).

This is not to say that the patient must be aware that there is a reduced affective response.[4] The deficit in unconscious affective processing might just have the result that the patient has a general feeling that "something is different" or that "something is different, and the difference is related to this person in front of me." The delusional belief would then arise as a specific hypothesis about what has changed. (It might even be that the deficit in unconscious affective processing has the result that it seems to the patient that "something is different, and the difference is that this person in front of me is not my spouse;" see section 4.2 below.)

The Cotard Delusion

In the case of the Capgras delusion, it is plausible that when the patient sees a familiar face, there is a reduced affective response, and as a result, the patient has an unusual experience. We can now ask whether experiences caused by loss of affective responsiveness can help explain other delusions, such as the Cotard delusion ("I am dead"). One suggestion made by Young et al. (1992) is that the Cotard patient has the same kind of experience as the Capgras patient but explains the experience in a different way (see section 3.1 below). Another possibility (which may be more plausible) is that the neuropsychological anomaly in the Cotard patient is a more general flattening of affective responses to stimuli. This might result in a loss of strong emotional experiences and a feeling of emptiness (Ramachandran and Blakeslee 1998; Gerrans 1999, 2000; Young 2000). Alternatively, the patient might have a general feeling that "everything is different."

The Capgras delusion and the Cotard delusion may arise from brain injury, but they also occur in patients suffering from psychosis. The patient who in the early stages of psychotic breakdown described his experience in the following way, seems to be heading in the direction of a nihilistic delusion like the Cotard delusion (McGhie and Chapman 1961, 109):

> "Things just happen to me now and I have no control over them . . . .At times I can't even control what I want to think about. I am starting to feel pretty numb about everything because I am becoming an object and objects don't have feelings."

Anscombe comments on this passage:

> The thoughts and actions that he performs do not have *his* stamp upon them, because in some basic, experiential sense, it does [not] feel as if *he* has initiated them . . . . It becomes harder for the patient to be an agent on his own behalf . . . .Objects don't have feelings; only people who connect with themselves do (Anscombe 1987, 254–55).

The Frégoli Delusion

If experiences caused by flattened affective responses can lead to delusions, might some other delusions be explained in terms of experiences that are caused by anomalously *heightened* affective responsiveness? Following a suggestion by Ramachandran and Blakeslee (1998), we invite you to suppose that you were to suffer a form of brain damage with the result that you had strong affective responses to most faces, not just the faces of people that you know. What experience would that give rise to? It might seem to you that most of the people that you see are people who you know (even people who are really complete strangers). If this were indeed the nature of your experience, you might naturally wonder why, if these are people that you know, you cannot recognize them. A possible explanation might be that, although these are people that you know, they are *in disguise*. The suggestion is, then, that experiences caused by anomalously heightened affective responses to faces might lead to the Frégoli delusion ("I am being followed around by people who are known to me but who are unrecognizable because they are in disguise"). (In fact, Ramachandran and Blakeslee [1998, 171] suggest that a heightened affective responsiveness to faces might cause a subject to have repeated experiences of seeing the same familiar face.)

Reduplicative Paramnesia

Nora Breen et al. (2000b) suggest that a similar explanation in terms of heightened affective responses might be offered for DB's reduplicative paramnesia. Recall that DB affirmed that her husband had died and was cremated four years earlier and that her husband was a patient on the ward in the same hospital that she was in.

It would not be quite right simply to take over the idea of a heightened affective response even to unfamiliar faces and to propose that it seemed to DB that some stranger on the ward was familiar to her, with the result that the stranger was misidentified as her husband, for DB did not identify anyone who was actually present as her husband. Instead, she said that her husband was on the ward in the hospital and she mentioned, for example, that he was talking to other people about having a stroke. What Breen et al. (2000b) suggest is that DB had a heightened sense of personal significance attached to certain remembered events. DB remembered an occasion when a patient on the ward had some visitors; perhaps she also remembered that the patient talked to his visitors about having a stroke. She then explained the sense of personal significance attached to this memory by the delusional hypothesis that the patient was her husband.[5]

Alien Control

In the case of the delusion of alien control ("Someone else is able to control my actions"), Christopher Frith and colleagues propose that the delusion arises when internal monitoring of self-initiated action is lost (Frith 1987, 1992; Frith and Done 1989; Mlakar et al. 1994). (For an authoritative review of the role of efference copies in the internal monitoring of actions, see Jeannerod 1997, chap. 6.) When visual and kinesthetic feedback of movement is intact, but the sense of self-initiation is lost, the resultant experience is of being a puppet with someone else pulling the strings. (For vivid descriptions of the alien control experiences of schizophrenic patients, see Spence et al. 1997.) Evidence for the hypothesis that the alien control experience is the result of a failure of monitoring is provided by the finding that patients with alien control experiences have difficulty correcting errors in their performance on a video game when they must monitor their movements in the absence of visual feedback (Frith and Done 1989).

Thought Insertion

Frith (1992) extends the idea of a failure to monitor the initiation of action to include the failure to monitor the initiation of thought or inner speech. Just as failure to monitor the initia-

tion of action leads to experiences of alien control, so also (Frith 1992, 81): "If we found ourselves thinking without any awareness of the sense of effort that reflects central monitoring, we might well experience these thoughts as alien and, thus, being inserted."

Frith also appeals to a failure to recognize that inner speech is self-initiated in his account of auditory hallucinations (Frith 1992, 73), but it may be that to give an account of both auditory hallucinations and thought insertion, we need to distinguish two components in our conception of first-personal ownership of thoughts. My thoughts are mine in the sense that they occur in my mind, in "inner space" rather than in extrapersonal space; they are also mine in the sense that I initiate or generate them. The experience of thought insertion results from a failure to recognize self-initiation, but the inserted thought is still experienced as occurring in inner space.[6] The experience of auditory hallucinations seems to be different from the experience of thought insertion (even when the inserted thoughts are verbalized). It may be suggested that auditory hallucinations result from a combination of deficits: a failure to monitor the initiation of inner speech and a loss of the sense of subjective privacy that is normally associated with hearing one's thoughts in "the mind's ear." (For a detailed account of auditory hallucinations, see David 1994.) If the sense of subjective privacy were to be lost while recognition of self-initiation remained intact, then a patient might have an experience that could figure in the etiology of the delusion of thought broadcasting (for further discussion, see Langdon and Coltheart 2000).

Denial of Ownership of a Limb in Unilateral Neglect

We asked whether it is possible to identify experiences that could lead to the eight delusions in our battery. So far we have suggested that six of the eight delusions might have a basis in experiences resulting from anomalous flattening or heightening of affective responses or from failures of monitoring. In the case of the seventh delusion in the battery—the denial of the ownership of a limb in patients with unilateral neglect—it is plausible that the delusion is to be explained, at least in part, in terms of the unusual experience that results from paralysis and the loss of kinesthetic and proprioceptive feedback from the arm. Bisiach and Geminiani (1991, 20) speak of "the feeling of alienness of the limbs contralateral to the brain lesion."

This leaves us with just the eighth of our delusions to consider. Does mirrored-self misidentification arise from an unusual experience of oneself as seen in a mirror?

Mirrored-Self Misidentification

It is important here to note that different types of brain disruption and, correspondingly, different types of cognitive deficit might give rise to unusual experiences of mirrored-self. One of the patients described by Breen et al. (2000b), FE, had a disorder of face processing and, for example, performed poorly on a face-matching task. In his case it is plausible that the visual appearance of his own face seen in a mirror was unusual to the extent that the way that the person in the mirror looked now was different from the way he remembered himself looking in earlier years. In contrast to the case of FE, another patient with a delusion of mirrored-self misidentification, TH, showed no significant face-processing disorder.[7]

However, when TH was asked to take in his hand various objects that were held up behind his shoulder, and so visible only in a mirror, he repeatedly attempted to reach into the mirror, scratching on its surface, or attempted to reach behind it. What TH had lost, it seems, was the ability to interact fluently with mirrors, even though he had not lost his encyclopedic knowledge about mirrors, since he was able to provide appropriate answers to such questions as, "What do people use mirrors for?"

At the beginning of this subsection, we said that we agreed with Maher that unusual experiences are one factor in the etiology of monothematic delusions. In what way, then, did TH have an abnormal visual perceptual experience of mirrored space or of himself as seen in a mirror? Based on his reaching into the mirror when asked to take reflected objects in his hand, it is certain-

Table 2. Experiences that could lead to delusions

Capgras delusion	Unusual experience of faces or a sense that "something is different" as a result of flattened affective responses
Cotard delusion	Loss of strong emotional experiences and a feeling of emptiness or a sense that "everything is different" as a result of global affective flattening
Frégoli delusion	Unusual experience of people as a result of heightened affective responses
Reduplicative paramnesia	Unusual experience as a result of heightened affective responses or a heightened sense of personal significance attached to remembered events
Alien control, thought insertion	Loss of experience of self-initiation of action or thought
Unilateral neglect	Loss of kinesthetic and proprioceptive experience of the arm and a feeling of the arm as being alien
Mirrored-self misidentification	Unusual experience of one's own face seen in the mirror or experience of reflected objects as if they were on the other side of the glass with loss of the ability to interact fluently with mirrors

ly natural to say that he saw those objects as being in a space behind the glass, but there is more than one account that might be given concerning the nature of our normal visual perception of mirrored space, and so it is not immediately clear whether TH's visual experience is abnormal.

A normal subject who is given the information that a sheet of glass is a mirror can make use of a complex set of visuo-motor transformations and reach fluently into the space in front of the glass to grasp reflected objects. One way of describing this situation is to say that a normal subject's visual perception of mirrored space *changes* once she learns that a sheet of glass is a mirror. Before the change, she sees objects as being in a space behind the glass, as if the glass were a window; after the change, she sees reflected objects is being in the space in front of the glass, and this is where she reaches to grasp those objects. On this way of describing the situation, a normal subject who knows that a sheet of glass is a mirror has a visual perceptual experience that is quite different from TH's experience when he looks in a mirror. A normal subject sees reflected objects as being in the space in front of the glass, but TH still sees reflected objects as being in a space behind the glass, as if the glass were a window. If this is the correct way to describe the situation, then TH's visual perception of mirrored space is certainly different from that of a normal subject.

In contrast, the situation might be described in a rather different way. It might be said that a normal subject's *visual perception* of mirrored space is the same both before and after she learns that the sheet of glass is a mirror. Once she learns that it is a mirror, it is her *dispositions to reach and grasp* that change as she draws on a set of transformations that recode spatial information derived from the visual perception into mirrored-space coordinates. On this second way of describing the situation, what normal subjects have and TH lacked is not a particular kind of visual perception of mirrored space but just the reaching and grasping abilities that are grounded in the accessibility of those visuo-motor transformations.

Table 3. Neuropsychological anomalies that could lead to delusions

Capgras delusion, Cotard delusion, Frégoli delusion, reduplicative paramnesia	Reduced or abnormal affective processing
Alien control, thought insertion	Failure of internal monitoring
Unilateral neglect	Loss of kinesthetic and proprioceptive feedback
Mirrored-self misidentification	Disorder of face processing or inaccessibility of visuo-motor transformations for mirrored space

However, we suggest that this second description is, as yet, incomplete. Before a normal subject realizes that a sheet of glass is a mirror, she may see her mirrored-self as someone just like her (but left-right reversed) in a space behind the glass, but once she learns that it is a mirror, she sees her reflected self as herself. It seems quite plausible that this difference at the level of conscious awareness is a result of the accessibility of the visuo-motor transformations, even if the subject is not actually required to do any reaching and grasping. Since the visuo-motor transformations were inaccessible for TH, it seems plausible that TH saw his reflected self as someone just like him, but not as himself. This difference from normal visual perception of mirrored-self would be a result of TH's neuropsychological anomaly.

In this subsection of our paper, we have identified experiences that could lead to the eight delusions in our battery (Table 2) and have also indicated the kinds of neuropsychological anomalies that could produce those experiences (Table 3). We are agreeing, then, with Maher when he says (Maher 1999, 551): "The origins of anomalous experience may lie in a broad band of neuropsychological anomalies." However, according to Maher, the delusion itself can be seen as a normal response to the unusual experience that results from a neuropsychological anomaly. This we wish to question. On our proposal, a second factor is required to account for the transition from the experience to a delusional belief. We propose that an unusual experience is perhaps necessary, but it is not sufficient. We now turn to the argument for that claim.

2. The Argument for a Second Factor

Suppose it is agreed that Table 2 offers a plausible account of the kind of experience that is associated with each of the eight monothematic delusions in our battery. On Maher's view, simply suffering from any one of these experiences would be sufficient to produce a delusion, because a delusion is the normal response to such unusual experiences. It follows that anyone who has suffered neuropsychological damage that reduces the affective response to faces should exhibit the Capgras delusion; anyone with a right hemisphere lesion that paralyzes the left limbs and leaves the subject with a sense that the limbs are alien should deny ownership of the limbs; anyone with a loss of the ability to interact fluently with mirrors should exhibit mirrored-self misidentification, and so on. However, these predictions from Maher's theory are clearly falsified by examples from the neuropsychological literature.

Reduced Affective Response to Faces without the Capgras Delusion

Tranel et al. (1995) describe a number of patients with damage to regions of both frontal lobes of the brain. Like the Capgras patients of Ellis et al. (1997), these patients did not show the typical affective response to familiar faces; they did not discriminate autonomically between familiar and unfamiliar faces. However, these patients, who we assume have the same kind of experience of faces as Capgras patients, were not delusional. (It is possible to question the assump-

tion that the patients studied by Tranel et al. [1995] have the same kind of anomalous experience as Capgras patients, since they had bilateral ventromedial frontal lesions, whereas at least some Capgras patients have different lesion sites. For further discussion, see Langdon and Coltheart [2000, 189–90].)

Global Affective Flattening without the Cotard Delusion

People with severe depression experience global affective flattening, as do those schizophrenic patients who exhibit the symptom known as flattening of affect. In both cases, the delusion that one is dead can be absent.

Many patients who are classified as suffering from the Cotard delusion do not explicitly claim to be dead but have other self-nihilistic delusions. We assume that these versions of the Cotard delusion are also associated with unusual experiences that result from affective flattening. However, it is possible to have self-nihilistic experiences without becoming delusional. Galen Strawson describes the following case:

> A friend who recently experienced depersonalisation found that the thought "I don't exist" kept occurring to him. It seemed to him that this exactly expressed his experience of himself, although he . . . knew, of course, that there had to be a locus of consciousness where the thought "I don't exist" occurred (Strawson 1997, 418).

Loss of Experience of Self-Initiation of Action without the Delusion of Alien Control

Patients with the alien control delusion say that an alien is controlling their actions, but patients with depersonalization disorder may say (among other things) that it is *as if* an alien were controlling their actions (Davison and Neale 1998). According to the *DSM*, an individual suffering from depersonalization disorder "may feel like an automaton" and may have "a sensation of lacking control of [his or her] actions." However, "The individual with Depersonalization Disorder maintains intact reality testing (e.g., awareness that it is only a feeling and that he or she is not really an automaton)" (American Psychiatric Association 1994, 500–02). Therefore, the alien control *experience* is not sufficient for the alien control *delusion*.

Loss of Kinesthetic and Proprioceptive Experience of a Limb without Denial of Ownership

The occurrence in some unilateral neglect patients of the delusional belief involving denial of ownership of a limb is not well understood, but there are many patients who exhibit the pattern that is crucial for the argument of this section. These are patients in whom damage to the sensory and motor areas of the right hemisphere has caused paralysis and loss of kinesthetic and proprioceptive feedback from their left limbs, who (presumably) share the unusual experience of the delusional patients and who neglect their left limbs, but who are not deluded concerning the ownership of those limbs. Bisiach and Geminiani (1991, 20) attribute "the feeling of alienness of the limbs contralateral to the brain lesion" to a range of patients not all of whom deny ownership of a contralesional limb.

Failure to Recognize One's Face in the Mirror because of a Disorder of Face-Processing without Mirrored-Self Misidentification

We stressed that different kinds of cognitive deficit could play a role in mirrored-self misidentification. In the case of patient FE, it is plausible that he has a disorder of face-processing and that the visual experience of his own face as seen in a mirror is anomalous (see Breen et al. 2000b, 87, 101–02). Patients with severe prosopagnosia are unable to recognize familiar faces and even unable to recognize themselves in a mirror, but the prosopagnosic patient does not have the delusional belief that the person in the mirror is not him.

Loss of Ability to Interact Fluently with Mirrors without Mirrored-Self Misidentification

We suggested that in some cases of mirrored-self misidentification, one factor may be a loss of the ability to interact fluently with mirrors (see Breen et al. 2000b, 91–92, 101). Binkofski et al.

(1999) have investigated thirteen patients who lack this ability to varying degrees. Patients classified as *mirror agnosic* reach towards the "virtual object" in the mirror and are not capable of changing their behavior; TH would belong in this category. Patients classified as *mirror ataxic* do learn to guide their arms towards the real object but not in a fluent way; they continue to make many errors. For the purposes of our argument, the crucial question is whether these patients suffer from mirrored-self misidentification. This question is not answered in the Binkofski et al. (1999) report, but Binkofski has informed us (pers. comm.): "As far as our parietal patients are concerned, none of them had difficulties in mirror self-recognition."

Now Maher (1999, 550) maintains, "The processes by which deluded persons reason from experience to belief are not significantly different from the processes by which non-deluded persons do." However, he also allows (Maher 1999, 566), "Many normal people have anomalous experiences but do not develop delusions. From this it is argued that something additional is necessary." This is roughly how we have argued, although we have not restricted ourselves to the anomalous experiences of normal subjects.[8] What is Maher's own response to examples of anomalous experience without delusion?

> One response to this is that the kinds of anomalous experience that deluded patients have appeared to be much more intense and prolonged than those that occur in the population in general. Their intensity and duration is determined by the prolonged duration or frequent repetition of the pathological state creating the experience (Maher 1999, 566).

This response can be adapted to our examples of non-delusional patients who have unusual experiences as a result of neuropsychological anomalies, but in our view, the response thus adapted is not really adequate.

It is certainly not generally true that delusions arise only after a prolonged period of anomalous experience. A delusion may be present very shortly after the onset of "the pathological state creating the experience." Thus consider again patient LA-O, described by Bisiach and Geminiani (1991, 32–33):

> Shortly before admission [to hospital] she had suddenly developed left hemiplegia [i.e., paralysis of the left side of the body] without loss of consciousness. Alert and cooperative, she claimed . . . that the left hand did not belong to her but had been forgotten in the ambulance by another patient . . . .Two days later the symptoms had partially faded . . . .She volunteered the information that only on that very morning she had begun to recognize her left hand as her own while being perfectly reminiscent of her past denial.

In this example, and surely in others, the response adapted from Maher will have to place weight on the notion of the intensity rather than the duration of the experience. However, although the duration of an experience can, in principle, be readily measured, it is not so clear how the intensity of an experience is to be quantified. (For this purpose, measurement of the intensity of an anomalous experience must not, of course, appeal to whether the subject is delusional.)

In any case, an experiment by Cahill et al. (1996) casts doubt on any proposal that the intensity of an experience is the crucial factor in explaining why some subjects who have that kind of experience become delusional while others do not. Subjects in this experiment heard their own voices through headphones, but the experience was anomalous because the pitch of the voice was distorted to varying degrees. A pilot study showed that normal volunteers were able correctly to attribute the heard voice to themselves, despite the pitch distortion. However, schizophrenic patients often attributed the heard voice to another agent, with one subject going so far as to say, "I think it's an evil spirit speaking when I speak" (Cahill et al. 1996, 209). The frequency of attributing the heard voice to another agent was correlated with the current severity of the patients' delusions and also with the degree of pitch distortion. Thus, delusional patients tended to make a false attribution of the heard voice to another agent, while normal subjects presented with the same distorted stimuli had no difficulty in correctly attributing the heard voice to themselves. Cahill and her colleagues note that these results do not support Maher's proposal that "erroneous beliefs reflected the operation of normal reasoning processes in the context of the experience of abnormal perceptions" (Cahill et al. 1996, 209). Instead,

We conclude that the "hallucination-like" reports elicited by our paradigm [the attribution of the heard voice to another agent] resulted from an interaction between an unusual perceptual experience (distorted auditory feedback) and an abnormal mechanism for belief formation present in deluded patients (Cahill et al. 1996, 201).

It is not easy to resolve all the issues here, but we know of no reason to suppose that in all the examples described above, the absence of a delusional belief can be explained in terms of the lower intensity or shorter duration of the unusual experience by comparison with delusional patients. Consequently we conclude that, while an unusual experience produced by a neuropsychological anomaly such as a deficit in perceptual or affective processing may be one factor in the etiology of a delusion, there must also be at least one other factor present if a delusion is to occur.

Our proposal is that for a monothematic delusion to occur, two factors must be present. One factor is a neuropsychological anomaly with some manifestation in the experience of the subject, but this is not a sufficient condition for the occurrence of delusions. The nature of the first factor varies from delusion to delusion and from patient to patient, and as Tables 2 and 3 indicate, there are different kinds of neuropsychological anomalies and different kinds of unusual experiences associated with different kinds of delusion. In contrast, on the boldest version of our proposal, the nature of the second factor is the same for all deluded patients. It is the second factor that explains the difference between a mirrored-self misidentification patient such as TH and a patient with mirror agnosia or between a patient with the alien control delusion and a patient with alien control experiences as part of depersonalization disorder.

The argument for a second factor is, of course, strengthened if we can find other examples of non-delusional subjects who have neuropsychological anomalies and consequent unusual experiences. Indeed, the ideal situation would be to have non-delusional cases corresponding to each of the eight delusions in our battery. There is more work to be done here. We do not know of any such non-delusional cases that correspond to the Frégoli delusion, to reduplicative paramnesia, or to thought insertion, but in our view, based on the examples of non-delusional patients that we have mentioned in this section, the argument for the two-factor proposal is already strong.

3. Biases in the Formation of Beliefs

In the last section, we argued that cases of a neuropsychological anomaly and consequent unusual experience without delusional belief show that Maher's account of delusions is not adequate—a second factor is required. The problem for Maher's claim that delusional beliefs are normal responses to unusual experiences can also be seen if we consider *two possible routes from experience to belief*. A subject might arrive at a belief by constructing and adopting an explanation of the occurrence of an experience, just as a scientist explains the occurrence of empirical data, or a subject might arrive at a belief by simply taking perception to be veridical.[9] Consider now the two possible routes from *unusual* experience to *delusional* belief, routes that Maher must regard as "normal."

Suppose, first, that delusional patients are aware that there is something unusual or anomalous about their experience and that they construct an explanation for the occurrence of this unusual feature. This seems to be the route that Maher has in mind when he says:

[T]he explanations (i.e., the delusions) of the patient are derived by cognitive activity that is essentially indistinguishable from that employed by non-patients, by scientists, and by people generally . . . .[A] delusion is a hypothesis designed to explain unusual perceptual phenomena (Maher 1974, 103).

The suggestion that delusions arise from the normal construction and adoption of an explanation for an unusual feature of experience faces the problem that delusional patients construct explanations that are not plausible and adopt them even when better explanations are available. This is a striking departure from the more normal thinking of non-delusional subjects who have similar unusual experiences. Consider, for example, the following two possible dialogues between a clinician and a right-hemisphere-damaged patient:

C: "Why can't you move this arm?"
P: "Because it isn't my arm; it's yours."

versus

C: "Why can't you move this arm?"
P: "Because I had a stroke in my right hemisphere; would you like to see my CT scan?"

(Anscombe [1987, 250] makes the same point about Maher's account as it applies to schizophrenic delusions: "[S]chizophrenic patients come up with explanations of their perceptual aberrations, particularly heightened significance, that are not sane. The sane explanation is that they have schizophrenia, or something like it.")

Suppose, alternatively, that a delusional belief arises because a patient takes an unusual perception to be veridical. If delusional patients simply believe what they perceive, implicitly assuming that perception is veridical, then they do what we all normally do as we use perception to find out about the world. Nevertheless, if the suggestion is that the route from unusual experience to delusional belief goes via the unreflective acceptance of veridicality, then it faces a problem. For delusional patients seem to proceed from experience to belief even when there are overwhelming reasons not to trust experience. This is once again in contrast with the more normal thinking of non-delusional subjects who have unusual experiences as a result of brain injury.

In our view, these problems reinforce the doubt about Maher's account. We agree with Young when he says:

> We think that the Capgras delusion represents just one among a number of ways in which people might try to explain similar anomalous perceptual experiences to themselves, and that to properly understand this delusion we need to understand not just the perceptual anomaly but also the factors which create and sustain the relatively bizarre impostor explanation (Young 2000, 63; see also Young 1998, 40–41; 1999, 572).

Young mentions attributional biases and reasoning biases, but recent research suggests that we should consider data-gathering biases instead of reasoning biases. For example, Garety and Freeman (1999, 131) say: "People with delusions do not, it seems, have a probabilistic reasoning bias, . . . but have a data-gathering bias." In our view, neither attributional biases nor data-gathering biases can play the role of the second factor in the etiology of monothematic delusions. In the remainder of this section, we consider them in turn.

3.1 Attributional Biases

Consider again the first possible route from experience to belief, i.e., delusional patients construct an explanation for the occurrence of an unusual experience. This account faces the problem that delusional patients construct explanations that are not plausible and adopt them even when better explanations are available. *Why* do delusional patients prefer the less good explanation?

Richard Bentall and others propose that persecutory delusions result from biases in the kinds of explanations that subjects give of their own behavior and the behavior of other people (Bentall 1994, 1995; Kinderman 1994; Bentall and Kinderman 1998). In particular, patients with persecutory delusions tend to blame other people when something goes wrong. The appeal to differences in attributional style may go some way towards explaining the difference between delusional and non-delusional subjects who have the same kind of unusual experience. It also opens the possibility of explaining different delusions as differently biased attempts to explain the same unusual feature of experience.

Thus, for example, concerning the Capgras and Cotard delusions, Young (2000, 65) suggests: "Although these delusions are phenomenally distinct, they may represent the patients' attempts to make sense of fundamentally similar experiences" (see also Young et al. 1992). The basic idea here is that, starting from the same unusual feature of experience, the Capgras delusion arises from an externalizing attributional style that is associated with feelings of persecution while the Cotard delusion arises from an internalizing attributional style that is associated with depression.

This is a fascinating, and in many ways plausible, suggestion that gathers support from a case in which a single subject experienced the Cotard and Capgras delusions in sequence (Wright et al. 1993). However, the account of the Capgras and

Table 4. Summary of the attributional-bias account

Capgras delusion = loss of affective responsiveness + a personal bias to externalize blame
Cotard delusion = loss of affective responsiveness + a personal bias to internalize blame

Cotard delusions that appeals to differences in attributional style cannot be fully general since there are cases of *concurrent* Capgras and Cotard delusions (Joseph 1986; Wolfe and McKenzie 1994; Butler 2000).

We accept that attributional biases may, in some cases, explain why a particular delusional hypothesis is prioritized as a *candidate* explanation of an unusual experience, but the appeal to attributional biases does not adequately address the argument for a second factor in the etiology of delusions, because it does not explain why the hypothesis is actually *adopted* and *maintained* as a belief.

3.2 Data-Gathering Biases

Consider now the second possible route from experience to belief, i.e., delusional patients simply believe what they perceive. This account faces the problem that delusional patients seem to proceed from experience to belief even when there are overwhelming reasons not to trust experience. *Why* do delusional patients trust experience when there are good reasons not to do so?

Philippa Garety and others propose that one factor in the etiology of delusions is a tendency to jump to conclusions based on insufficient evidence (Huq et al. 1988; Garety et al. 1991; Garety and Hemsley 1994; Garety and Freeman 1999; see also Dudley et al. 1997a,b). Results in probabilistic reasoning experiments with delusional patients do not reveal a total inability to reason probabilistically, but subjects do show a bias towards early acceptance of hypotheses. Does this bias in data gathering offer an explanation of how delusions arise based on unusual experiences and why they are maintained?

We accept that a tendency to jump to conclusions may help to explain why the delusional hypothesis is initially adopted as a belief, but it does not explain why the delusional belief is *maintained* so tenaciously. Indeed, in Garety's experiments, delusional subjects jump to conclusions more rapidly than normal subjects but also tend to abandon existing hypotheses based on very little evidence. The appeal to a data-gathering bias, like the appeal to attributional biases, falls short as a response to the argument for a second factor in the etiology of delusions.

4. The Second Factor

The strength of the two-factor account of monothematic delusions is the evidence that a first factor (often a neuropsychological anomaly in perceptual or affective processing) is demonstrably present in many patients with delusions, yet is not itself sufficient to cause the delusion. The weakness of the account as it currently stands is the inadequate characterization of the nature of the second factor. The second factor might be described as a loss of the ability to reject a candidate for belief on the grounds of its implausibility and its inconsistency with everything else that the patient knows. However, attempts to say in more detail what this loss of ability amounts to face many problems. In this final section, we describe some of these.

4.1 Two Problems: Monothematicity and Appreciation of Implausibility

In this paper we are concerned with monothematic delusions,[10] but the very fact of *monothematicity* already presents a challenge. The patients that we have described typically have just one delusional belief or a small set of related delusional beliefs. Outside the specific domain of the delusion, their belief systems are not abnormal. If these patients suffer from some kind of deficit or abnormality in their belief evaluation system, what is it that protects them from adopting bizarrely false beliefs in other domains?

At least some delusional patients show considerable *appreciation of the implausibility* of their delusional beliefs. This is evident in the following extract from an interview with a man who thought that his house and family had been replaced by duplicates (Alexander et al. 1979, 335):

E: Isn't that [two families] unusual?
S: It was unbelievable!
E: How do you account for it?
S: I don't know. I try to understand it myself, and it was virtually impossible.
E: What if I told you I don't believe it?
S: That's perfectly understandable. In fact, when I tell the story, I feel that I'm concocting a story . . . . It's not quite right. Something is wrong.
E: If someone told you the story, what would you think?
S: I would find it extremely hard to believe. I should be defending myself.

Delusional patients with this kind of appreciation of the implausibility of their delusional belief are not surprised that the people around them refuse to accept the belief as true. (Young [1998, 37] says, "Capgras delusion patients can be otherwise rational and lucid, able to appreciate that they are making an extraordinary claim. If you ask 'what would you think if I told you my wife had been replaced by an impostor?', you will often get answers to the effect that it would be unbelievable, absurd, an indication that you had gone mad.") If these patients suffer from a loss of the ability to evaluate beliefs for plausibility and consistency with other things that they know, how are they able to judge that other people will find their belief implausible?

We can see how these two problems arise for putative accounts of the second factor in the etiology of delusions if we briefly consider the idea that delusional patients are unable to make appropriate and effective use of stored knowledge. There is evidence, for example, that schizophrenic patients may be prone to "over-inclusive" thinking, classifying an airplane in the category *bird* because it has wings and flies (Chen et al. 1994). A categorization problem of this kind is consistent with the idea that schizophrenic patients ignore stored information about "exceptions." In line with that idea, recent research by Sellen et al. (2000), using a conditional inference task, suggests that high-schizotypy normal subjects make less use of information about "exceptions" than low schizotypy subjects.[11] It might be proposed, then, that monothematic delusions are the result of failure to use information in encyclopedic or "semantic" memory to assess the plausibility of a candidate for belief. Any such proposal faces both the *monothematicity problem* and the *appreciation of implausibility problem*. If delusional patients cannot use information in semantic memory to reject the delusional belief, then why do they not end up with a host of other bizarrely implausible beliefs? If delusional patients cannot use information in semantic memory to assess the plausibility of their delusional belief, then how do they appreciate so clearly that other people will indeed find it implausible?

4.2 The Two Routes from Experience to Delusional Belief

In Section 3, we distinguished two possible routes from unusual experience to delusional belief. On the first route, delusional patients are aware that there is something unusual or anomalous about their experience, and they construct an explanation for the occurrence of this unusual feature. On the second route, delusional patients simply believe what they perceive; they implicitly assume that perception is veridical. Consider now how the Capgras delusion might arise by one or other of these routes.

Suppose first that the patient has a visual perception of someone who looks just like a close relative and that this is accompanied by awareness that there is a reduced affective response or by a sense that "it doesn't feel like her," or just by a general feeling that "something is different." Suppose that the patient sets out to construct an explanation for the occurrence of this unusual feature of experience. This might lead to the consideration, and ultimately the adoption, of the putatively explanatory hypothesis that the person who looks like the close relative is not really a close relative but an impostor. Intuitively, the patient goes wrong in adopting, and all the more so in maintaining, an explanatory hypothesis that should be rejected.

Suppose, on the other hand, that the patient's unusual experience represents the situation as follows: "This is someone who looks just like my close relative *but is not really her/him*." If the delusional hypothesis is already part of the *representational content* of the patient's perception,

then the route to a delusional belief involves nothing more than accepting the perception as veridical.[12] In this case, it seems that the patient goes wrong in not making a critical assessment of, and particularly in not rejecting, the veridicality of this unusual perceptual experience.

The remainder of this section is organized around the distinction between the route to belief that involves the construction, adoption, and maintenance of an explanatory hypothesis and the route to belief that simply involves taking a perception as veridical. We consider in turn possible accounts of the second factor that assume either the first or the second route.

4.3 Delusional Beliefs and Explanatory Hypotheses

Suppose, once again, that a Capgras patient has a visual perception of someone who looks just like a close relative and that this is accompanied by awareness that there is a reduced affective response, or by a sense that "it doesn't feel like her," or just by a general feeling that "something is different." We need to account for the construction, adoption, and maintenance of the explanatory hypothesis that the person who looks like the close relative is not really a close relative but an impostor.

Even if we leave aside the question why it occurs to the patient to construct this bizarre hypothesis (perhaps the idea of an attributional bias can help here), we need some cognitive account of why the hypothesis is adopted and maintained, rather than being rejected on the grounds of its implausibility and its inconsistency with everything else that the patient knows. In particular, we need to answer three questions: First, why can the delusional hypothesis not be refuted by using first-person experience? Second, why can it not be refuted by using stored encyclopedic knowledge? Third, why can it not be refuted by relying on the testimony of other people?

In answer to the first question, it may be helpful to recall, from Catts's draft Definition 2 (Figure 1), the suggestion that a delusional belief "has the quality of being self-evidential and unfalsifiable (irrefutable) either because of the nature of the belief or because of the refusal of the holder of the belief to admit anything that would falsify the belief." There are two alternative proposals here. One is that the belief is unfalsifiable because of its content; the other is that the subject elevates the belief to such a status that nothing is allowed to count against it. The second of these is quite close to what we are trying to explain, namely, the adoption and maintenance of an implausible hypothesis, but the first proposal might contribute to an explanation if we interpret it as saying that a delusional belief has a content that makes it especially difficult to refute by using first-person experience. Indeed, this does seem somewhat plausible in the case of delusions of misidentification such as the Capgras delusion. The delusional hypothesis cannot be readily refuted by evidence concerning, for example, the observable characteristics of the supposed impostor or the way that he or she answers questions.

Even if it is correct that the content of a delusional hypothesis makes it difficult to refute by using first-person experience, we still need to answer the second and third questions. We have already seen (Section 4.1) that an answer to the second question is liable to be problematic if it appeals to a general problem in the use of stored knowledge. However, we might speculate that there is a more specific problem that arises because of the particular contents of delusions. For example, it might be that using stored information to refute the delusional hypothesis would require the information to be available in explicit or declarative form, while the information that would be needed is usually stored in an implicit or procedural form. As for an answer to the third question, we might speculate that in delusional patients, reliance on the testimony of others is impaired either by a theory of mind deficit (see Langdon et al. [2002] for a review of theory of mind deficits in patients with schizophrenia) or else by a social psychological problem of shrinking the "in group" of people whose opinions can be trusted.[13]

These answers to the three questions add up to a complex, underspecified, and highly speculative story about the etiology of the Capgras delusion or any other delusional belief. However,

it might be thought that the answers to the first and second questions do at least offer some resources for addressing the monothematicity problem. The reason is that there might be only a rather narrow class of hypotheses meeting the two conditions of being "self-evidential" or "unfalsifiable" in the required sense, while also, for example, being immune to refutation by stored information unless the information is converted from a procedural to a declarative format. We suggest, though, that an account based on these kinds of answers to the three questions about why the delusional hypothesis is not rejected still faces a version of the monothematicity problem. An account of this kind seems to yield the problematic prediction that a delusional subject will differ from normal subjects in being liable to adopt and maintain false beliefs based on misleading perceptual experiences presented by certain visual illusions. Let us explain what we see as the problem here.

Suppose that a normal subject knows that her twin sons, Bill and Ben, are the same height and now sees them through the viewing window of an Ames room in which Bill is standing in the tall corner to the left while Ben is standing in the short corner to the right (see Gregory 1970, 26–28). From the viewing position, Bill looks shorter than Ben, but we take it that, even without entering the Ames room to measure her sons, the normal subject will reject the hypothesis that now, in the Ames room, Ben is taller than Bill. She will prefer the hypothesis that there is something strange—as yet, she knows not what—about the way things look in this room.

Now consider a delusional patient who also knows Bill and Ben well and sees them in the Ames room. Bill, on the left, looks shorter than Ben, on the right. Suppose that this patient constructs the hypothesis that people and things change their height when they go into this room: Those who go to the left of the room get shorter; those who go to the right of the room become taller. It is not a straightforward matter for the patient to refute this hypothesis by using first-person experience, whether he remains at the viewing position or enters the room himself to measure Bill and Ben. For according to the hypothesis, both he and his measuring rod become shorter as he moves to the left to measure Bill and then grow taller as he moves to the right to measure Ben.

The normal subject makes use of the stored knowledge that height does not change with mere change of position, but it is fairly plausible that this is just the kind of knowledge about the physical world that might be stored in procedural form. The delusional patient may not be able to make use of this stored knowledge to refute his hypothesis. Given our answer to the third question about why it is difficult for him to refute the delusional hypothesis, he may not be able to make use of the testimony of other people either. In short, we have the prediction that the delusional patient who sees Bill and Ben in the Ames room is liable to adopt and maintain a second bizarrely false hypothesis.

It is true that we do not have any data to report about the ways in which delusional patients respond to visual illusions. It might conceivably turn out that even patients whose delusions are monothematic would indeed arrive at a second, and unrelated, bizarrely false belief if they were placed in the situation that we have described. Nevertheless, the prediction is one that we would prefer to avoid. If we assume the first route from experience to belief and try to give an account of why the delusional hypothesis is not rejected, then we face the monothematicity problem in the form of an unwanted prediction.

We have taken some time to explain how the monothematicity problem arises from our speculative answers to three questions about why a delusional explanatory hypothesis might be difficult to refute. However, we do not need to take very much time over the problem about appreciation of implausibility. We are supposing that, in virtue of its content, a delusional hypothesis is difficult to refute by using first-person experience or by relying on stored encyclopedic knowledge, and that a delusional patient's reliance on the testimony of others is also impaired. Against the background of these assumptions, it surely is puzzling that patients should have such a keen appreciation of the implausibility of their delusional beliefs.

4.4 Delusional Beliefs and the Assumption that Perception is Veridical

The prospects for a solution to the appreciation of implausibility problem seem to be better if we assume the second route from experience to belief. It is part of the representational content of a Capgras patient's visual perception that "This is someone who looks just like my close relative but is not really her/him," and the patient simply accepts this perceptual experience as veridical.

Normal subjects usually assume perception to be veridical; they believe what they perceive. We might describe this transition from experience to belief as a *pre-potent doxastic response*. However, normal subjects are also able to suspend their unreflective acceptance of veridicality and make a more detached and critical assessment of the credentials of their perceptual experiences. We might conceptualize what happens in the Capgras patient or other delusional patients as *failure to inhibit a pre-potent doxastic response*. (The suggestion here is not that delusional patients have a general inability to inhibit pre-potent responses.) If this were the nature of the second factor in the etiology of delusions, then hypotheses that were generated from the patient's own perceptual experience would be resistant to being critically assessed and recognized as implausible, but hypotheses generated by someone else would be assessed in the normal way. Thus, the pre-potent doxastic response proposal seems to promise help with the appreciation of implausibility problem. (The pre-potent response proposal also seems to allow for the circumscribed nature of many monothematic delusions. A delusional patient who is unable to reject a perceptually generated hypothesis might be able to offer more or less normal assessments of the plausibility of its inferential consequences. Quite consistently with the idea that a delusional patient is unable to inhibit the pre-potent response of believing what he perceives, a delusional patient might still refrain from believing an implausible consequence of a delusional belief.)

However, the pre-potent doxastic response proposal still faces the monothematicity problem in the form of an unwanted prediction about the ways in which delusional patients will respond to visual illusions. A delusional patient who fails to inhibit the pre-potent doxastic response of taking perception to be veridical is liable to adopt and maintain other false beliefs as a result of having misleading perceptual experiences. For example, if such a patient sees the twins Bill and Ben in the Ames room, then he will accept that things are as they appear to be and so will believe that Bill is shorter than Ben. Even if he knows Bill and Ben well and knows that they are the same height, this stored knowledge will not undermine the perceptually generated belief. According to the pre-potent doxastic response proposal, if a delusional patient first sees Bill and Ben outside the Ames room, then he will believe that they are the same height, whether or not he already knows them. If he later sees them inside the Ames room, then he will believe that *now* Bill is shorter than Ben, even while admitting—presumably—that a little earlier they were the same height.

Once again we do not have any data to report. It is conceivable that patients with monothematic and circumscribed delusions resulting from brain injury would form additional bizarrely false beliefs if presented with visual illusions. Nevertheless, we would prefer an account of the second factor that avoided this prediction.

Starting from the two routes from experience to belief, we have considered two possible accounts of the second factor in the etiology of monothematic delusions. Both accounts face problems. In particular, both face versions of the monothematicity problem in the form of unwanted predictions. However, regarding the two accounts, problematic though they are, we prefer the pre-potent doxastic response proposal. It is less complex and seems to be better able to allow for delusional patients' appreciation of the implausibility of their delusional beliefs.[14]

Conclusion

In this paper, we have taken some steps towards a two-factor account of monothematic delusions, while also acknowledging the problems that such an account faces.

Starting from Maher's view that delusions are false beliefs that arise as normal responses to

unusual experiences, we identified experiences that result from neuropsychological anomalies and could lead to the eight delusions in our battery (Section 1). We then provided examples of unusual experiences in non-delusional subjects and argued that a second factor is required to account for the transition from unusual experience to delusional belief (Section 2).

We considered two possible routes from experience to belief. One route involves the construction, adoption, and maintenance of an explanatory hypothesis; the other route is simply a matter of taking perception to be veridical. We then briefly considered the role that might be played by attributional biases and data-gathering biases and argued that appeals to these biases do not provide an adequate account of the adoption and maintenance of delusional beliefs (Section 3).

The second factor in the etiology of delusions can be described superficially as the loss of the ability to reject a candidate for belief on the grounds of its implausibility and its inconsistency with everything else that the patient knows. However, in the final section we pointed out two problems that confront any attempt to say more about the nature of this second factor: the monothematicity problem and the appreciation of implausibility problem. At present, we do not know how those problems are to be solved, but our provisional view is that we do better to assume the second route from unusual experience to delusional belief and then to conceptualize the second factor as a failure to inhibit a pre-potent doxastic response.

It is not wholly surprising that there is a real difficulty here. What we are trying to do is to describe an abnormality in the cognitive systems that are responsible for the evaluation and fixation of beliefs, but we do not yet have an adequate theory of the normal operation of those systems.[15]

Acknowledgments

We are grateful to Adam Andrews, Tony Atkinson, Tim Bayne, Daniel Friedrich, Philip Gerrans, Christoph Hoerl, and Tony Stone for comments and conversations.

Notes

1. Thanks to Dr. Stanley V. Catts and the PORS (Psychosis Observational Rating Scale) Research Team at UNSW for permission to use this material. The results of their questionnaire study will be published in due course.

2. Maher speaks of "anomalous experiences," but it is not really clear that, when an experience figures in the etiology of a delusion, the experience itself is anomalous. It may be better to speak, more neutrally, of aspects of the conscious mental life of the subject that arise from neuropsychological anomalies.

3. See Ellis and Young (1990), Ellis and de Pauw (1994), Ellis et al. (1997), and Young (1998, 1999, 2000). Prosopagnosia is the breakdown of overt or conscious recognition of familiar faces. Some prosopagnosic patients continue to show a heightened affective response (as measured by skin conductance) to familiar faces: see Bauer (1984) and Tranel and Damasio (1985). Ellis, Young, and colleagues appeal to Bauer's (1984) dual-route model of face recognition. Breen et al. (2000a) criticize Bauer's model and propose an alternative model of face recognition. For further discussion, see Ellis and Lewis (2001), Breen et al. (2001), and Lewis and Ellis (2001).

4. In fact, the idea that the patient would be aware that there is a reduced affective response does not sit very happily with the proposal that the Capgras delusion is a mirror image of prosopagnosia. There is no reason to suppose that prosopagnosic patients are aware that they have affective responses to familiar faces that they can no longer recognize as being familiar.

5. See Breen et al. (2000b, 93) for DB's delusional belief and her apparent recollection that her husband "was talking to some of the men that he had a stroke and he could feel it coming on." See Breen et al. (2000b, 102) for the suggestion that "DB's paramnesic delusion involved the false attribution of a sense of personal familiarity to a recent remembered experience" and page 103 for a discussion of the relevance of heightened affective responses to delusions of misidentification in general.

6. Hoerl (this issue) begins from a description of obsessional thoughts offered by Frankfurt (1976, 240–41): "It is tempting . . . to suggest that they are not thoughts that *we think* at all, but rather thoughts that we *find* occurring within us." Hoerl then motivates an account of thought insertion by considering some differences in decision making between obsessional patients and schizophrenic patients. See also Campbell (1999).

There remains a question why, when there is a failure to recognize self-initiation, a thought is experienced as having been initiated *by someone else* rather than merely as having arisen unbidden. One possibility here is to appeal to an externalizing attribution bias

(Bentall [1994] and see below, section 3.1). It may be, however, that there is some more purely experiential difference between thought insertion and ordinary unbidden thoughts.

7. Breen et al. (2000b) report that FE's responses to photographs of familiar and unfamiliar faces and to unfamiliar objects indicate "a heightened sense of familiarity" (87) and that "Similarly to FE, but to a much lesser extent, TH did make false positive identifications of strangers' faces as familiar" (91). They connect these points with their discussion of the role of heightened affective responsiveness in the etiology of delusions (103).

8. Anscombe argues in the same way concerning schizophrenic delusions by appealing to the similarities between the phenomenology of schizophrenic patients who are delusional and of people who take LSD but are not delusional (Anscombe 1987, 250): "This indicates that heightened significance alone is not sufficient to induce delusion, and that we require more extreme pathology to justify the transition from illusion to delusion."

9. It might be said that taking an experience as veridical is just a matter of adopting a particular explanation of that experience, namely, that things really are as they appear to be. In response, we would make two points. First, what is described as taking an experience as veridical need not involve treating the experience itself as something that needs to be explained. Second, constructing an explanation of an experience may go far beyond bare acceptance that things are as they appear to be. The distinction between the two routes seems to be clear enough for our purposes.

10. We started out thinking that this would be a simplifying assumption, but we were wrong.

11. Subjects first viewed a conditional statement, such as "If Claire turned on the air conditioner, then she felt cool." This was followed by an additional fact, such as the antecedent of the conditional, "Claire turned on the air conditioner" and a conclusion, such as "Claire felt cool." Subjects were asked to rate how sure they were that the conclusion could be drawn. Low schizotypy subjects were more influenced than high schizotypy subjects by the existence of "disabling conditions," such as the air conditioner being broken, the possibility of which would be available in encyclopedic ("semantic") memory. High schizotypy subjects thus showed a more "logical" pattern of performance.

12. The representational content of a perceptual experience is the way that the perception, considered as a conscious folk psychological state, represents the world as being. If it is part of the representational content of a perceptual experience that so-and-so is the case, then a subject who took the perception as veridical would, without needing to make use of collateral information, judge that so-and-so is the case. The representational content of visual perception is not just a matter of colored patches; it can involve kinds of material objects. Given that I know what a computer is and what a computer looks like, it can be part of the representational content of my visual experience that there is *a computer* on the desk in front of me. Nor is the representational content of perception just a matter of kinds of objects; it can involve known individuals. It might be part of the representational content of my visual experience that *my favorite cufflinks* are on the dressing table, or that *that man from the art gallery* is now in this wine bar, or that *Andy Young* has just stepped up to the lectern. If this is right, then it does not seem that the notion of representational content has built into it any impediment to the idea of a perception with the content: "This is someone who looks just like my close relative but is not really her/him."

It remains a difficult question how a perceptual experience with that content would be generated (see Campbell, this issue). Information-processing models of face recognition are often relatively silent about the relationship between the model and the phenomenology of recognizing a familiar face. However, it does not seem to be ruled out that conscious recognition of a familiar face is underpinned by a sufficiently high level of activation of a person identity node and that this level of activation may be reduced by an impairment or disconnection involving the affective response system. See Breen et al. (2000a, 2001), Ellis and Lewis (2001), and Lewis and Ellis (2001).

13. Here we are indebted to Alex Haslam.

14. However, these advantages for the pre-potent response proposal are purchased at the price of specific assumptions about the representational content of the delusional patient's perceptual experience, and these assumptions might not be tenable for all the delusions in our battery. See again note 12.

15. See Fodor (2000). For the same reason, we are not yet able to pursue the normal double dissociation methodology of cognitive neuropsychology. Our argument depends on cases in which there is an unusual experience, but the second factor is absent. However, we do not know what to expect from subjects who suffer from the abnormality in belief evaluation but whose experience is normal.

REFERENCES

Alexander, M. P., D. T. Stuss, and D. F. Benson. 1979. Capgras syndrome: A reduplicative phenomenon. *Neurology* 29:334–39.

American Psychiatric Association 1994. *Diagnostic and statistical manual of mental disorders*, Fourth Edition (*DSM-IV*). Washington, DC: American Psychiatric Association.

Anscombe, R. 1987. The disorder of consciousness in schizophrenia. *Schizophrenia Bulletin* 13:241–60.

Arthur, A. Z. 1964. Theories and explanations of delusions: A review. *American Journal of Psychiatry* 121:105–15.

Bauer, R. 1984. Autonomic recognition of names and faces: A neuropsychological application of the Guilty Knowledge Test. *Neuropsychologia* 22:457–69.

Bentall, R. P. 1994. Cognitive biases and abnormal beliefs: Towards a model of persecutory delusions. In *The neuropsychology of schizophrenia,* eds. A. S. David and J. C. Cutting, 337–60. Hove, East Sussex: Psychology Press.

———. 1995. Brains, biases, deficits and disorders. *British Journal of Psychiatry* 167:153–55.

Bentall, R. P., and P. Kinderman. 1998. Psychological processes and delusional beliefs: Implications for the treatment of paranoid states. In *Outcome and innovation in the psychological treatment of schizophrenia*, eds. T. Wykes, N. Tarrier, and S. Lewis, 119–44. Chichester: John Wiley and Sons.

Berrios, G. E., and R. Luque. 1995. Cotard's syndrome: Analysis of 100 cases. *Acta Psychiatrica Scandinavica* 91:185–88.

Berson, R. J. 1983. Capgras syndrome. *American Journal of Psychiatry* 140:8.

Binkofski, F., G. Buccino., C. Dohle et al. 1999. Mirror agnosia and mirror ataxia constitute different parietal lobe disorders. *Annals of Neurology* 46:51–61.

Bisiach, E. 1988. Language without thought. In *Thought without language*, ed. L. Weiskrantz, 464–84. Oxford: Oxford University Press.

Bisiach, E., and A. Berti. 1995. Consciousness in dyschiria. In *The cognitive neurosciences*, ed. M. S. Gazzaniga, 1331–40. Cambridge, MA: MIT Press.

Bisiach, E., and G. Geminiani. 1991. Anosognosia related to hemiplegia and hemianopia. In *Awareness of deficit after brain injury: Clinical and theoretical issues*, eds. G. P. Prigatano and D. L. Schacter, 17–39. Oxford: Oxford University Press.

Breen, N., D. Caine, and M. Coltheart. 2000a. Models of face recognition and delusional misidentification: A critical review. *Cognitive Neuropsychology* 17:55–71.

Breen, N., D. Caine, M. Coltheart et al. 2000b. Towards an understanding of delusions of misidentification: Four case studies. In *Pathologies of belief*, eds. M. Coltheart and M. Davies, 75–110. Oxford: Blackwell Publishers.

Breen, N., M. Coltheart, and D. Caine. 2001. A two-way window on face recognition. *Trends in Cognitive Sciences* 5:234–35.

Butler, P. V. 2000. Diurnal variation in Cotard's syndrome (copresent with Capgras delusion) following traumatic brain injury. *Australian and New Zealand Journal of Psychiatry* 34:684–87.

Cahill, C., D. Silbersweig, and C. Frith. 1996. Psychotic experiences induced in deluded patients using distorted auditory feedback. *Cognitive Neuropsychiatry* 1:201–11.

Campbell, J. 1999. Schizophrenia, the space of reasons, and thinking as a motor process. *The Monist* 82:609–25.

Capgras, J., and J. Reboul-Lachaux. 1923. L'illusion des 'sosies' dans un délire systématisé chronique. *Bulletin de la Société Clinique de Médicine Mentale* 11:6–16.

Chen, E. Y. H., A. J. Wilkins, and P. J. McKenna. 1994. Semantic memory is both impaired and anomalous in schizophrenia. *Psychological Medicine* 24:193–202.

Cotard, J. 1882. Du délire des négations. *Archives de Neurologie* 4:152–70, 282–95.

Courbon, P., and G. Fail. 1927. Syndrome d''illusion de Frégoli' et schizophrenie. *Bulletin de la Société Clinique de Médecine Mentale* 20:121–25.

Critchley, M. 1953. *The parietal lobes*. London: Hafner Press.

Currie, G. 2000. Imagination, delusion and hallucinations. In *Pathologies of belief*, eds. M. Coltheart and M. Davies, 167–82. Oxford: Blackwell Publishers.

David, A. S. 1994. The neuropsychological origin of auditory hallucinations. In *The neuropsychology of schizophrenia*, eds. A. S. David and J. C. Cutting, 269–313. Hove, East Sussex: Psychology Press.

Davison, G. C., and J. M. Neale. 1998. *Abnormal psychology*, Seventh Edition. New York: Wiley.

de Pauw, K. W., T. K. Szulecka, and T. L. Poltock. 1987. Frégoli syndrome after cerebral infarction. *Journal of Nervous and Mental Diseases* 175:433–38.

Dudley, R. E. J., C. H. John, A. W. Young et al. 1997a. Normal and abnormal reasoning in people with delusions. *British Journal of Clinical Psychology* 36:243–58.

———. 1997b. The effect of self-referent material on the reasoning of people with delusions. *British Journal of Clinical Psychology* 36:575–84.

Eilan, N. 2000. On understanding schizophrenia. In *Exploring the self: Philosophical and psychopathological perspectives on self-experience*, ed. D. Zahavi, 97–113. Amsterdam: John Benjamins.

Ellis, H. D., and K. W. de Pauw. 1994. The cognitive neuropsychiatric origins of the Capgras delusion. In *The neuropsychology of schizophrenia*, eds. A. S. David and J. C. Cutting, 317–35. Hove, East Sussex: Psychology Press.

Ellis, H. D., and M. B. Lewis. 2001. Capgras delusion: A window on face recognition. *Trends in Cognitive Sciences* 5:149–56.

Ellis, H. D., and A. W. Young. 1990. Accounting for delusional misidentifications. *British Journal of Psychiatry* 157:239–48.

Ellis, H. D., J. Whitley, and J. P. Luauté. 1994. Delusional misidentification: The three original papers on the Capgras, Frégoli and intermetamorphosis delusions. *History of Psychiatry* 5:117–46.

Ellis, H. D., A. W. Young, A. H. Quayle et al. 1997. Reduced autonomic responses to faces in Capgras delusion. *Proceedings of the Royal Society: Biological Sciences* B264:1085–92.

Fodor, J. 2000. *The Mind Doesn't Work That Way*. Cambridge, MA: MIT Press.

Förstl, H., O. P. Almeida, A. M. Owen et al. 1991. Psychiatric, neurological and medical aspects of misidentification syndromes: A review of 260 cases. *Psychological Medicine* 21:905–10.

Frankfurt, H. 1976. Identification and externality. In *The identities of persons*, ed. A. O. Rorty, 239–51. Berkeley, CA: University of California Press.

Frith, C. D. 1987. The positive and negative symptoms of schizophrenia reflect impairments in the perception and initiation of action. *Psychological Medicine* 17:631–48.

———. 1992. *The Cognitive neuropsychology of schizophrenia*. Hove, East Sussex: Lawrence Erlbaum Associates.

Frith, C. D., and D. J. Done. 1989. Experiences of alien control in schizophrenia reflect a disorder in the central monitoring of action. *Psychological Medicine* 19:359–63.

Garety, P. A., and D. Freeman. 1999. Cognitive approaches to delusions: A critical review of theories and evidence. *British Journal of Clinical Psychology* 38:113–54.

Garety, P. A., and D. R. Hemsley. 1994. *Delusions: Investigations into the psychology of delusional reasoning*. Oxford University Press. (Reprinted, Hove, East Sussex: Psychology Press, 1997.)

Garety, P. A., D. R. Hemsley, and S. Wessely. 1991. Reasoning in deluded schizophrenic and paranoid patients: Biases in performance on a probabilistic inference task. *Journal of Nervous and Mental Disease* 179:194–201.

Gerrans, P. 1999. Delusional misidentification as subpersonal disintegration. *The Monist* 82:590–608.

———. 2000. Refining the explanation of Cotard's delusion. In *Pathologies of belief*, eds. M. Coltheart and M. Davies, 111–22. Oxford: Blackwell Publishers.

Gregory, R. L. 1970. *The intelligent eye*. London: Weidenfeld and Nicolson.

Hirstein, W., and V. S. Ramachandran. 1997. Capgras syndrome: A novel probe for understanding the neural representation of the identity and familiarity of persons. *Proceedings of the Royal Society: Biological Sciences* B264:437–44.

Huq, S. F., P. A. Garety, and D. R. Hemsley. 1988. Probabilistic judgments in deluded and non-deluded subjects. *Quarterly Journal of Experimental Psychology* 40A:801–12.

Jaspers, K. 1963. *General psychopathology*, trans. J. Hoenig and M. W. Hamilton. Manchester: Manchester University Press.

Jeannerod, M. 1997. *The cognitive neuroscience of action*. Oxford: Blackwell Publishers.

Joseph, A. B. 1986. Cotard's syndrome in a patient with coexistent Capgras' syndrome, syndrome of subjective doubles, and palinopsia. *Journal of Clinical Psychiatry* 47:605–606.

Kinderman, P. 1994. Attentional bias, persecutory delusions and the self concept. *British Journal of Medical Psychology* 67:53–66.

Langdon, R., and M. Coltheart. 2000. The cognitive neuropsychology of delusions. In *Pathologies of belief*, eds. M. Coltheart and M. Davies. Oxford: Blackwell Publishers, 183–216.

Langdon, R., M. Davies, and M. Coltheart. 2002. Understanding minds and understanding communicated meanings in schizophrenia. *Mind and Language* 17:68–104.

Lewis, M. B., and H. D. Ellis. 2001. A two-way window on face recognition: Reply to Breen et al. *Trends in Cognitive Sciences* 5:235.

MacDonald, N. 1960. The other side: Living with schizophrenia. *Canadian Medical Association Journal* 82:218–21.

Maher, B. A. 1974. Delusional thinking and perceptual disorder. *Journal of Individual Psychology* 30:98–113.

———. 1988. Anomalous experience and delusional thinking: The logic of explanations. In *Delusional beliefs*, eds. T. F. Oltmanns and B. A. Maher, 15–33. Chichester: John Wiley and Sons.

———. 1992. Delusions: Contemporary etiological hypotheses. *Psychiatric Annals* 22:260–68.

———. 1999. Anomalous experience in everyday life: Its significance for psychopathology. *The Monist* 82:547–70.

McGhie, A., and J. Chapman. 1961. Disorders of attention and perception in early schizophrenia. *British Journal of Medical Psychology* 34:103–16.

Mlakar, J., J. Jensterle, and C. D. Frith. 1994. Central monitoring deficiency and schizophrenic symptoms. *Psychological Medicine* 24:557–64.

Ramachandran, V. S., and S. Blakeslee. 1998. *Phantoms in the brain: Human nature and the architecture of the mind*. London: Fourth Estate.

Sellen, J., M. Oaksford, and N. Gray. 2000. Delusional beliefs: The role of reasoning and over-inclusive thinking. Poster, British Psychological Society Annual Conference.

Signer, S. F. 1987. Capgras syndrome: The delusion of substitution. *Journal of Clinical Psychiatry* 48:147–50.

Simpson, J. A., and E. S. C. Weiner (eds.) 1989. *Oxford English Dictionary*, 2d ed. Oxford: Clarendon Press.

Spence, S. A., D. J. Brooks, S. R. Hirsch, P. F. Liddle, J. Meehan, and P. M. Grasby. 1997. A PET study of voluntary movement in schizophrenic patients experiencing passivity phenomena (delusions of alien control). *Brain* 120:1997–2011.

Stone, T., and A. W. Young. 1997. Delusions and brain injury: The philosophy and psychology of belief. *Mind and Language* 12:327–64.

Strawson, G. 1997. 'The self'. *Journal of Consciousness Studies* 4:405–28.

Tranel, D., and A. R. Damasio. 1985. Knowledge without awareness: An autonomic index of facial recognition by prosopagnosics. *Science* 228:1453–54.

Tranel, D., H. Damasio, and A. R. Damasio. 1995. Double dissociation between overt and covert recognition. *Journal of Cognitive Neuroscience* 7:425–32.

Vallar, G. 1998. Spatial hemineglect in humans. Trends in Cognitive Sciences 2:87–97.

Way, A. S. (trans.) 1988. Euripides, Vol. 3 (Loeb Classical Library). Cambridge, MA: Harvard University Press.

Wolfe, G., and K. McKenzie. 1994. Capgras, Frégoli and Cotard's syndromes and Koro in folie a deux. British Journal of Psychiatry 165:842.

Wright, S., A. W. Young, and D. J. Hellawell. 1993. Sequential Cotard and Capgras delusions. British Journal of Clinical Psychology 32:345–49.

Young, A. W. 1998. Face and mind. Oxford: Oxford University Press.

———. 1999. Delusions. The Monist 82:571–89.

———. 2000. Wondrous strange: The neuropsychology of abnormal beliefs. In Pathologies of belief, eds. M. Coltheart and M. Davies, 47–73. Oxford: Blackwell Publishers.

Young, A. W., and K. M. Leafhead. 1996. Betwixt life and death: Case studies of the Cotard delusion. In Method in madness: Case studies in cognitive neuropsychiatry, eds. P. W. Halligan and J. C. Marshall, 147–71. Hove, East Sussex: Psychology Press.

Young, A. W., I. Reid, S. Wright et al. 1993. Face processing impairments and the Capgras delusion. British Journal of Psychiatry 162:695–98.

Young, A. W., I. H. Robertson, D. J. Hellawell et al. 1992. Cotard delusion after brain injury. Psychological Medicine 22:799–804.