Delusional Belief

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ABSTRACT

Delusional beliefs are seen in association with a number of neuropathological conditions, including schizophrenia, dementia and traumatic brain injury, A key distinction exists between *polythematic delusion* (here the patient exhibits delusional beliefs about a variety of topics that are unrelated to each other) and *monothematic delusion* (here the patient exhibits just a single delusional belief, or else a small set of delusional beliefs that are all related to a single theme). A great deal of recent research has focused on identifying and investigating various different forms of monothematic delusion. We discuss a general theoretical approach to the understanding of monothematic delusions – a two-factor approach according to which understanding the nature and genesis of any kind of monothematic delusion involves seeking answers to two questions. The first question is: what is it that brought the delusional idea to mind in the first place? The second question is: why is this idea accepted as true and adopted as a belief when the belief is typically bizarre and when so much evidence against its truth is available to the patient? We discuss in detail six different kinds of monothematic delusion, showing for each of them how neuropsychological considerations allow a first factor (responsible for the content of the belief) and a second factor (responsible for the failure to reject the belief) to be plausibly identified. Five difficulties confronting this two-factor account of monothematic delusion are then identified, and attempts are made to address each one.

INTRODUCTION

Delusions are the archetypal signs of madness (Jaspers, 1963) and the core feature of "psychosis". "Psychosis" translates from the Greek into "illness of the soul or mind" and is used today to refer to mental illness characterised by "losing touch with reality". Many people with psychosis profess bizarre and unsubstantiated beliefs about reality, beliefs not shared by other people within their community – that is, delusional beliefs. Other people with psychosis report aberrant sensory experiences that carry the same sense of reality as genuine percepts but lack any corresponding external reality –that is, hallucinations, which often co-occur with delusions. Some people with psychosis use language idiosyncratically and have difficulty in communicating effectively with other people, in social settings.

Such psychotic symptoms have been documented since the 1700's. Today these symptoms are key diagnostic criteria for major psychiatric illnesses like schizophrenia, although they also occur in many organic medical conditions like dementia. The distinctions between delusions and hallucinations (and disturbances in communicative ability) within psychotic phenomenology are relatively straightforward to understand. Delusions are commonly understood as "false beliefs", while hallucinations are commonly understood as "false percepts"; the latter need not involve any mistaken belief (e.g., many patients with Lewy body dementia will correctly attribute their vivid visual hallucinations to a disease of the brain). The distinctions between delusions and other "reality distortion" symptoms are less clear-cut. "Anosognosia", for example, refers to an unawareness, or a denial, of genuinely present injury and/or illness, and is conventionally considered as distinct from delusion; and yet anosognosia might be conceived of as the delusional belief that one is not suffering from any illness/injury, or, at least, that one is not really as ill as the doctors say (see, e.g., Davies, Aimola Davies & Coltheart, 2005). "Confabulation" refers traditionally to a falsification of memory arising unintentionally in the context of organic memory impairment (see, e.g., Berlyne, 1972; Berrios, 1998). Like anosognosia, confabulation is conventionally considered as distinct from delusion; and yet confabulations might be conceived of as delusional beliefs about one's past (or present, or future).

We begin by discussing the received clinical wisdom concerning the definition of delusion, according to the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision* (*DSM-IV-TR*: APA, 2000) and various objections to this definition. We then review the relative prevalence of delusions and the various conditions in which they occur, along with common delusional themes, before discussing key phenomenological characteristics. We conclude by considering how the genesis and nature of delusional beliefs might be explained within a cognitive-neuropsychiatric framework.

THE CLINICAL BACKGROUND: DELUSIONS ACCORDING TO THE DSM-IV-TR

There are two well established and widely used systems for the classification of mental disorders: Chapter V of the *International Statistical Classification of Diseases and Related Health Problems, Tenth Revision (ICD-10)*, published by the World Health Organization (WHO; 1992), and the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision* (*DSM-IV-TR*), published by the American Psychiatric Association (APA; 2000). The *DSM-IV* is, internationally, the more widely used system for research purposes (the *ICD-10* is more widely used for clinical diagnosis and training; see Mezzich, 2002), and provides the most often quoted definition of delusion. So we will consider how delusions are treated by the *DSM-IV-TR*. We will keep these remarks relatively brief, however, as *DSM-V* is currently under development.

Delusions are mentioned in various places in the *DSM-IV-TR*, but they feature most prominently in the chapter on *Schizophrenia and Other Psychotic Disorders* (pp. 297-343). As befits the manual's function and its categorical medical-model approach, delusions are typically considered for the purposes of differential diagnosis. Delusion is one of the set of characteristic symptoms used in assigning a diagnosis of schizophrenia. Other characteristic symptoms in this set include hallucinations, disorganised speech or disorganised behaviour (which, along with delusions, are referred to as "positive" symptoms as they are considered abnormal by virtue of the presence of something), and apathy and anhedonia, an absence of experiences of joy or pleasure (these are referred to as "negative" symptoms as they are considered abnormal by virtue of the absence of

something). Other diagnostic categories which also feature delusions include:

- Schizophreniform Disorder (diagnosed if the criteria for schizophrenia are present but have not been observed for the requisite six months);

- Schizoaffective Disorder (diagnosed if mood symptoms also occur, and are sometimes present when symptoms of schizophrenia are currently absent);

- Delusional Disorder (diagnosed if delusions are the sole major symptom shown by the patient);

- Brief Psychotic Disorder (diagnosed if symptoms resolve within one month);

- Psychotic Disorder Due to a General Medical Condition;

- Substance-Induced Psychotic Disorder, and

- some cases of Mood Disorders.

The *DSM-IV-TR* distinguishes bizarre delusions (such as the belief that a stranger has removed and replaced one's internal organs without leaving scars) from mundane delusions (such as an unfounded belief in one's spouse's unfaithfulness). This is a distinction of diagnostic import, since just the presence of bizarre delusions permits the diagnosis of schizophrenia (even if no other symptoms are present), whilst precluding a diagnosis of Delusional Disorder. Thus the diagnosis of Delusional Disorder requires that the presenting delusions be mundane.

Delusions also feature in the *Mood Disorders* (pp. 345-428) section of the *DSM-IV-TR*, where their presence or absence is taken into account in specifying a patient's current clinical status. For example, the "Severe With Psychotic Features" specifier denotes the presence of either delusions or hallucinations during a current Major Depressive, Manic or Mixed Episode. Most commonly, delusional content is consistent with the relevant mood (which can be depressed or manic), in which case the clinician specifies "Mood-Congruent Psychotic Features". Examples include delusions of guilt or poverty in the

context of depressive mood, or delusions of grandiosity in the context of manic mood.

The *DSM-IV-TR* provides this often-quoted definition of "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. The belief is not one ordinarily accepted by other members of the person's culture or subculture (e.g., it is not an article of religious faith). When a false belief involves a value judgment, it is regarded as a delusion only when the judgment is so extreme as to defy credibility. Delusional conviction occurs on a continuum and can sometimes be inferred from an individual's behavior. It is often difficult to distinguish between a delusion and an overvalued idea (in which case the individual has an unreasonable belief or idea but does not hold it as firmly as is the case with a delusion). **(**American Psychiatric Association, 2000, p. 821)

The *DSM-IV-TR* definition attempts to capture the nature of delusion according to a set of necessary and sufficient criteria, an approach that is the accepted wisdom in psychiatry and clinical psychology, but that is far from acceptable with regard to its theoretical adequacy (see, e.g. David, 1999; Oltmanns, 1988; Smith, 1968), so it is worth reviewing objections to this definition.

One contentious issue for the *DSM-IV-TR* definition of delusion concerns what it says about there being incontrovertible evidence against the delusional belief . The problem here is that some delusions, such as the conviction that one's poetry displays extraordinary artistic merit (Fulford, 1994), are irrefutable in principle because they lie in the realm of subjective evaluation and arguably lack truth values (Blaney, 2009). The *DSM-IV-TR* definition seems to acknowledge this insofar as it stipulates that value judgments must "defy credibility" to be considered delusional, but the fact that the phrase "false belief" is still used here indicates that there is at least some confusion concerning the distinction between abnormal beliefs and abnormal value judgments. This putative distinction touches on the thorny issue of distinguishing between delusions and over-valued ideas. An over-valued idea is an isolated sustained belief which is not as inflexible or as idiosyncratic as a delusion - it is more like a passionate religious or fanatical political conviction - and which typically does not prevent the individual from remaining functional (Veale, 2002). Over-valued ideas feature in

anorexia nervosa or bulimia, body dysmorphic disorder, gender dysphoria and body integrity identity disorder, where the patients worry, with an obsessive passion, that their body or some feature of their body is inadequate or deformed, that they have been born the wrong gender, or that one of their limbs is not a 'real' part of them and that they would be better off if it were removed.

Secondly (and perhaps surprisingly), one of the most contentious aspects of the *DSM-IV-TR* definition is the proviso that delusions be false (Peters, 2001; Spitzer, 1990). For one thing, incontrovertible proof against delusions is often not available, as we have just noted: many delusional claims may be unfalsifiable, in practice if not in principle. Consider a patient with a delusion of "control" or "loss of boundary" who claims that an invisible agent is controlling her every move – what evidence could count against this belief? A second issue is that certain non-bizarre delusions might turn out to be "accidentally" true, so to speak – delusional claims of marital infidelity, for example (Coltheart, 2009; Jaspers, 1963; Spitzer, 1990). As Jaspers (1963, p. 106) wrote: "A delusion of jealousy, for instance, may be recognized by its typical characteristics without our needing to know whether the person has genuine ground for his jealousy or not. The delusion does not cease to be a delusion although the spouse of the patient is in fact unfaithful - sometimes only as a result of the delusion." These points suggest that the relevant consideration is not whether a claim is true or false, but whether the claim shows the characteristic signs of a delusion, one of which is the absence of sufficient justificatory evidence (Spitzer, 1990; cf. Leeser & O'Donohue, 1999).

A third contentious issue concerns the seemingly fundamental stipulation that delusions are beliefs. Although we adopt this "doxastic[1] conception" of delusions (our article is, after all, entitled "Delusional belief"), a number of authors have criticized it (see Bayne & Pacherie, 2005, for a review). We'll discuss two of these criticisms.

The first criticism of the doxastic conception of delusion involves the extent to which deluded individuals act on their delusions. Of course, in order to be identified at all, a delusion must manifest itself in behaviour of some sort, even if only in a verbal

[1] "Doxastic" means "pertaining to belief"

declaration. And there are certainly cases where delusions are more vigorously acted upon – sometimes with tragic consequences (see de Pauw & Szulecka, 1988; Silva, Ferrari, Leong and Penny, 1998). Nevertheless, it is true that some deluded individuals fail to take the actions that one might expect them to take if they believed their delusions (Sass, 1994). For example, those who claim to be Jesus or Napoleon are rarely seen attempting to communicate with their disciples or troops (Young, 2000) and those who express the belief that their spouse has been replaced by a stranger or impostor (Capgras delusion, discussed later) do not try to establish the whereabouts of the missing spouse. Delusional beliefs are thus often "encapsulated". Such failures to act would represent difficulties for the doxastic conception of delusions if one insisted that an apparent belief does not count as a belief unless it is acted upon. But that criterion for what counts as a belief does not have to be accepted; and in any case even if it were, there are various reasons why a deluded patient might not act on the basis of the delusional belief. Firstly, action is not caused by cognitive states alone, but by cognitive states in conjunction with motivational states – and deluded patients have disrupted affective and emotional states. Secondly, patients might realise that acting on their beliefs might result in hospitalization. Thus there may be strong countervailing reasons not to act on a delusional belief. A more extensive discussion of the issue of acting on the basis of delusional belief may be found in Bayne & Pacherie (2005).

A second criticism of the doxastic conception is that some delusions are pragmatically self-defeating (Bayne & Pacherie, 2005; Bermúdez, 2001). Indeed, the assertion of some patients with Cotard delusion that they themselves do not exist seems to violate one of the most famous propositions in the history of philosophy (the Cartesian *cogito*; see Young & Leafhead, 1996; McKay & Cipolotti, 2007). In response to objections like these, some authors have developed alternatives to the standard doxastic conception of delusions. For example, the "metacognitive" account of Currie and colleagues (Currie, 2000; Currie & Jureidini, 2001) conceives of delusions as imaginings that are misidentified as beliefs. On this account, the patient who claims to be Napoleon does not erroneously believe that he is Napoleon: he merely imagines that he is Napoleon, and erroneously identifies this imagining as a belief.

A fourth contentious issue for the *DSM-IV-TR* definition of delusion concerns its stipulation that delusions be "based on incorrect inference about external reality", Davies and Coltheart (2000) note that certain delusions might be arrived at simply by taking an illusory experience as veridical. In such cases, the delusional belief that is adopted (e.g. "an external power is controlling my actions") would have the same representational content as an anomalous experience (i.e., an experience of an external power controlling my actions). The delusion would not therefore be constructed as an *explanation* of the anomalous experience, but would be a straightforward consequence of simply *endorsing* that experience (see Bayne & Pacherie, 2004a, 2004b; Pacherie, 2009). This route from experience to delusional belief would not involve an inferential step, although unconscious inference-making might be involved in the generation of the experiential content.

Assuming an inference *is* involved, however, what kind of inference must it be? The *DSM-IV-TR* specifies that the inference is both "incorrect" and "about external reality". The latter stipulation is easy to dispute, as some delusions clearly concern *internal* reality – not just internal in a bodily sense, such as the delusion that there is a nuclear reactor inside one's body (David, 1990) or that one is pregnant with an impostor foetus (Silva, Leong, Longhitano, & Botello, 1991) - but internal in a psychological sense as well (e.g., delusions of thought insertion). The stipulation that the inference is incorrect, however, is debatable. Brendan Maher (e.g., Maher, 1974) has consistently argued that delusions arise when normal inferential reasoning is employed to explain intense and unusual phenomenological experiences. Others, however (e.g., Huq, Garety & Hemsley, 1988; Garety, Hemsley & Wessely, 1991; Garety & Freeman, 1999), have explored the possibility that the inferential reasoning of deluded individuals departs systematically from that of healthy individuals.

A fifth such issue is the requirement that delusions must be "firmly sustained". Peters (2001) has noted that many delusions are not firmly sustained, nor are they necessarily impervious to contrary evidence. The conviction of many delusional patients may wax

and wane, vacillating between avowal and disbelief (Sharp, Fear, Williams, Healy, Lowe et al., 1996; David, 1999; Coltheart, 2007).

A sixth and final contentious issue is the fact that the *DSM-IV-TR* defines delusion in such a way as to exclude any belief held by a sufficiently large number of people. This has been contested by Davies, Coltheart, Langdon and Breen (2002), who argue that any bizarrely implausible belief formed and maintained in ways characteristic of unambiguous delusions should, for theoretical purposes, be classified as a delusion. Nevertheless, the clause about religious faith can be defended as reasonable (at least insofar as it pertains to conventional religious belief) on both pragmatic grounds (to avoid pathologising most of the world's people) and on scientific grounds (the production of supernatural beliefs is arguably a proper biological function of naturally selected belief mechanisms; see Johnson & Bering, 2006).

Prevalence of delusion

As noted in the *DSM-IV-TR*, delusions can occur in both the "functional" and the "organic" psychoses, although it is generally agreed today that this etiological distinction between functional and organic psychoses is now largely of historical interest only. Many of the historically termed "functional psychoses", which used to be conceived of as occurring in the absence of known organic brain damage - schizophrenia, for example - are now believed to have a strong biological basis, though the specific nature of this biological basis is still debated. Schizophrenia is commonly conceptualised today as a neurodevelopmental disorder, with onset of characteristic symptoms typically delayed until a crucial time of brain development in adolescence, though it is also considered that genetic vulnerability and early perinatal insults play important causal roles (see, e.g., Fatemi & Folsom, 2009; Jarskog, Miyamoto & Lieberman, 2007).

Since the presence of delusion is a diagnostic criterion for the historically termed functional psychoses, the lifetime and current prevalence of delusions and the incidence

of new cases of delusions in these conditions are necessarily high. Prevalence and incidence rates of delusion are also high, however, in many of the historically termed organic psychoses; several reviews (see, e.g., Cummings, 1985; Fricchione, Carbone & Bennett, 1995; Nasrallah, 1992) have documented that delusions are common in the dementias (present in the order of 15-56% of those with Alzheimer's Disease, and in 27-60% of those with multi-infarct dementia), in temporal lobe epilepsy (estimated presence 7-23%), in Huntington's disease (estimated presence 20-83%) and in Parkinson's disease. A more recent review (Aarsland, Ballard, Larsen & McKeith, 2001) has suggested that 29-54% of people with Parkinson's disease and dementia experience delusions, with the prevalence rate lower, at 7-14%, in Parkinson's disease without dementia.

Delusions are less frequent, although still clinically significant, in traumatic brain injury (TBI). TBI is conservatively estimated to increase the risk of developing delusions two to three times (at the very least) relative to the risk in the general population (see, e.g., Arciniegas, Harris & Brousseau, 2003, David & Prince, 2005, for reviews). While the increased prevalence of delusions suggests that TBI is causally implicated in the onset of delusions, there is typically a gap of months to years between the occurrence of the TBI and the onset of the symptoms, which casts doubt on the role of TBI in the genesis of delusions – e.g., it is not uncommon for clinicians to take the view that the experience of TBI is simply another stressor for an individual with a pre-injury vulnerability to develop a fundamentally functional psychosis. In support of this latter view is the finding that post-TBI psychosis is difficult to distinguish from the phenomenology of schizophrenia (Sachdev, Smith & Cathcart, 2001). However, this latter finding is not surprising, since postmortem and structural imaging findings suggest decreased cortical volume and abnormal neuronal structure in the frontal and temporo-limbic regions (hippocampus, amygdala, basal ganglia, thalamus: see, e.g., Pantelis et al., 2003) in schizophrenia, and since Fujii and Ahmed (2002) found that most TBI patients who develop delusions show magnetic resonance imaging (MRI) and/or electroencephalographic (EEG) abnormalities in fronto-temporal areas.

While delusions are common to many conditions, delusional themes vary across these

conditions, and are often in accord with the particular diagnostic condition (e.g. grandiose delusions in the context of manic mood in Bipolar Disorder) and/or the site of brain damage, when this can be established.

Delusional themes

Persecutory and referential delusions

In psychiatric illnesses, persecution and self-referentiality are the most common delusional themes. Persecutory delusions involve beliefs that other people are intending threat or harm to oneself (Freeman and Garety, 2000), whereas delusions of reference involve beliefs that unrelated or commonplace phenomena in the world (events, objects or other people) refer directly to oneself and carry a special personal significance (Startup & Startup, 2005). Delusions of persecution and reference commonly co-occur (often along with hallucinations). Sartorius et al. (1986) examined symptoms in 1379 people with the signs of schizophrenia who were making a first-in-lifetime contact with a clinical service. These data were collected for the World Health Organisation (WHO) Study across ten countries. Sartorius and colleagues found persecutory and referential delusions to be the most common, experienced by about 50% of the sample. Persecutory delusions are likewise common in other psychiatric conditions; e.g., it is estimated that 48% of people with depressive psychoses experience persecutory delusions (Frangos, Athanassenas, Tsitourides, Psilolignos & Katsanou, 1983), while the prevalence of persecutory delusions in bipolar patients in a manic episode is estimated at a significant 28% (Goodwin & Jamison, 1990). The International Pilot Study of schizophrenia (World Health Organization, 1973) reported that 67% of people with schizophrenia experience delusions of reference. Jorgensen (1986) reported an even higher rate, at 75%, in people with bipolar psychosis and co-occurring paranoid symptoms. Perhaps not surprisingly, referential delusions are common in body dysmorphic disorder (BDD), along with somatic delusions. Phillips, McElroy, Keck, Pope and Hudson (1993) reported that two thirds of BDD patients experience referential thinking, half of whom will be delusional about their referential ideas.

Referential delusions vary with regard to their specific content; some patients believe

that others are communicating with them via subtle and oblique means, such as hints, innuendos or gestures (referential delusions of communication), while others believe that they are being observed, monitored or followed by others, who might be gossiping and spreading rumours about them (referential delusions of observation). Referential delusions might thus be secondary to other delusions with themes of persecution, guilt or grandiosity (or hallucinations) or they might arise independently of other symptoms when the delusional person becomes suddenly and unrealistically convinced (via, e.g., a subjective sensation of aberrant, heightened significance: Kapur, 2003) that a particular event refers to them with a special significance (Wing, Cooper & Sartorius, 1974).

Persecutory and referential delusions are also common in medical conditions with a known organic basis. Among delusional Alzheimer's patients, the prevalence of persecutory delusions is estimated at 18.5-79%, with delusions of reference estimated in 14.9-54%. These delusions often incorporate themes of stealing, estimated in 34.5-76% of delusional Alzheimer's patients (see, e.g., Rao & Lyketsos, 1998, and Bassiony & Lyketsos, 2003, for reviews). Post-TBI psychosis is another organic condition in which persecutory and referential themes are common, although mixed combinations of different delusional themes and hallucinations, as is often seen in psychosis associated with epilepsy, can also occur. Fujii and Ahmed (2002) reported that 35% of the delusions in their patients who had post-TBI delusions were persecutory, in contrast to 15% that were grandiose and/or somatic. Sachdev et al. (2001) similarly reported a high prevalence of 56% for persecutory delusions in post-TBI psychosis, with referential delusions the second most common, present in 22%. Persecutory and/or referential delusions are also reported in epilepsy (Trimble, 1992) and are common in Substance-Induced Psychotic Disorders, being particularly associated with chronic use of cannabis and methamphetamine (see Corlett, Frith & Fletcher, 2009, for general discussion of the specific relationships between drugs and delusions).

Control, passivity or loss of boundary delusions

Whereas persecutory and/or referential delusions are commonly seen in both "functional" and "organic" psychoses, "loss of boundary" delusions, including

"control" delusions, are considered to be more specifically characteristic of schizophrenia. Kurt Schneider (1959) sought to make the diagnosis of schizophrenia more reliable by identifying "first-rank" symptoms (FRS's) which were less likely to occur in other psychoses. These FRS's include loss of boundary delusions, along with voices heard conversing or commenting on one's behavior. Patients with loss of boundary delusions might believe that thoughts are being inserted into their minds (thought insertion) or withdrawn from their minds (thought withdrawal), that their thoughts are being broadcast so as to be accessible to others (thought broadcasting), that other people can read their minds (delusions of mind-reading), or that their actions and/or thoughts are in some manner under the influence of an external force (delusions of alien control).

Ihara et al. (2009) recently examined the prevalence of FRS's in a first-episode psychoses sample of 426. They found FRS's to be more common in schizophrenia (*DSM-IV*: 55%; *ICD-10*: 51%) than affective psychoses (*DSM-IV*: 31%; *ICD-10*: 29%). Peralta and Cuesta (1999a) similarly reported FRS's to be almost entirely absent in Delusional Disorder, with experiences of thought broadcast, thought insertion, and control more prevalent in DSM-III-R schizophrenia spectrum conditions (i.e., Schizophrenia, Schizophreniform disorder, and Schizoaffective disorder) than in Mood Disorder with Psychosis or Brief Reactive Psychosis (e.g. thought broadcasting occurred in 40.5-54.5% of the patients with schizophrenia spectrum conditions, 20.5% of the Mood Disorder patients and 16% of those with Brief Reactive Psychosis). Similar comparisons apply with regard to the organic psychoses; e.g., Sachdev et al. (2001) reported that 22% of their patients with post-TBI psychosis experienced control delusions as compared to 56% who experienced persecutory delusions. Peralta and Cuesta (1999a) caution, however, that the diagnostic criteria for schizophrenia-related conditions often emphasis FRS's; hence their apparent diagnostic discriminatory value may be a tautology.

Factor-analytic studies of delusional themes

The findings of factor-analytic studies generally confirm marked heterogeneity with regard to delusional themes, including passivity/control themes, even within the

schizophrenia spectrum conditions. Factor-analytic studies which aim to identify relatively independent, co-occurring groups of symptoms commonly use the Scales for the Assessment of Positive and Negative Symptoms of schizophrenia (SAPS and SANS: Andreasen, 1983, 1984). The SAPS incorporates items for rating delusions with themes of persecution, reference, jealousy, guilt or sin, grandiosity, control, thought insertion, thought broadcasting, mind-reading and thought withdrawal, as well as religious/spiritual delusions and somatic delusions. Delusions of jealousy and guilt/sin are the least common, present in fewer than 10% of psychotic patients, and are usually excluded from factor-analytic studies, while grandiose and/or religious delusions are quite common, with Wing et al. (1974) reporting a prevalence rate of 30% in schizophrenia. Of interest with regard to those who conceive of similarities between temporolimbic epilepsy and schizophrenia (see, e.g., Saver & Rabin, 1997, for discussion), grandiose and/or religious delusions are also common in delusional people with epilepsy, particularly temporolimbic epilepsy; in such cases, ecstatic seizures, which purportedly stem from a temporolimbic source, and which involve experiences of unity, harmony and/or divinity, are likely to be implicated.

With regard to the relevant findings from factor-analytic studies, initial interest focused on the global SAPS/SANS ratings (for, e.g., delusions, hallucinations, and bizarre behaviour). More recent studies have, however, focused, instead, on the individual SAPS/SANS items (see Peralta & Cuesta, 1999b, for discussion of the importance of analysing symptoms at the level of individual items). Studies of the latter type have typically revealed a far more complex structure than the three-factor structure (of reality distortion, disorganization and negative symptoms) that is typically reported when global SAPS/SANS ratings have been examined. For example, Peralta and Cuesta (1999b) reported 12 principal components, with loss of boundary delusions being distinguishable from: (a) delusions with grandiose, spiritual or guilt themes; (b) persecutory or referential delusions; and (c) somatic delusions, which co-occurred with somatic and olfactory hallucinations. Similar components were reported by Minas, Stuart, Klimidis, Jackson, Singh and Copolov (1992) and Stuart, Malone, Currie, Klimidis and Minas (1995), although some caution is warranted with regard to

interpreting results of this type since the specific factors that are identified will inevitably be influenced by the specific items that are included in the analysis.

Heterogeneity of delusional themes

Studies of the factor-analytic type highlight the need for theoretical accounts of delusion which can explain why some patients develop, say, persecutory delusions, while other patients develop, say, somatic delusions. With regard to the thematic distinctions that have been drawn with regard to the historically termed "organic" psychoses, Cummings (1985) approached the question of heterogeneity of delusional content by proposing four general types of organic delusions: 1) simple persecutory delusions, 2) complex persecutory delusions, 3) grandiose delusions, and 4) delusions associated with specific neurological defects. Simple persecutory delusions were conceived of as being relatively simple mistaken beliefs, which are often transient, such as delusions concerning theft of one's belongings or spouse infidelity. Complex delusions were conceived to be more inflexible and persistent. Delusions associated with specific neurological conditions included conditions like reduplicative paramnesia and Anton's syndrome. Reduplicative paramnesia is a condition in which patients believe that they are in one place which is a duplicate of some other place in another geographical location; this condition is typically associated with right hemisphere dysfunction. Patients with Anton's syndrome suffer from cortical blindness and yet deny that they are blind.

But matters are more complicated than the Cummings scheme might suggest, because even within the category of "delusions associated with specific neurological deficits", there is extreme thematic heterogeneity. We have already mentioned two very different forms of delusion that belong in this category (reduplicative paramnesia and Anton's syndrome) but there are many others. For example, there are delusions of misidentification (the brain-damaged patient has a delusional belief about the identities of specific people), delusions about the ownership of body parts (the brain-damaged patient believes that one of his limbs is the limb of someone else), and delusions about the brain damage itself (the brain-damaged patient denies having suffered such damage

and denies the presence of symptoms which are in fact present and are due to the brain damage: this is anosognosia).

Even within the single category of misidentification delusion, there is thematic heterogeneity. Young (2000) discusses several examples:

• A woman who suffered a right temporo-parietal infarct developed Frégoli delusion, which is the delusional belief that one is being followed by people whom one knows but who are disguising their appearance – in this case, the patient believed that she was being followed by a cousin in disguise (de Pauw, Szulecka & Poltock, 1987);

• A man who suffered a right hemisphere stroke developed a misidentification delusion about a postgraduate student who came to test him, whom he wrongly believed to be his daughter (Young, Flude & Ellis, 1991); and

• A man who had sustained right temporal and bilateral frontal brain damage believed that his house and family had been duplicated and existed in two different places (Alexander, Stuss & Benson, 1979).

• In Capgras delusion (typically associated more with right than left hemisphere brain damage), the patient believes that some highly familiar person such as a spouse has been replaced by a stranger or impostor who looks just like the spouse (Edelstyn & Oyebode, 1999; Bourget & Whitehurst, 2004; Feinberg & Shapiro, 1989).

Monothematic versus polythematic delusions

In the four types of delusion associated with brain damage that have just been described, the delusions are *monothematic*: the patient exhibits just a single delusional belief, or else a small set of delusional beliefs that are all related to a single theme. This is also common in DSM-IV Delusional Disorder, although here the monothematic delusion is by definition mundane rather than bizarre. But in other cases of delusion, the delusions are polythematic: the patient exhibits delusional beliefs about a variety of topics that are unrelated to each other; this is particularly associated with DSM-IV Schizophrenia. For example, there is the celebrated case of the German Supreme Court

judge Daniel Schreber: in addition to a varied array of hypochondriacal symptoms (Schreber was convinced that he had the Plague and that his brain was softening), he believed that divine forces were preparing him for a sexual union with God, a union that would engender a new race of humans who would restore the world to a lost state of blessedness (Bell, 2003). Another celebrated example was the Nobel laureate John Nash, also diagnosed with schizophrenia: amongst the beliefs he expressed were that he would become Emperor of Antarctica, that he was the left foot of God on Earth, and that his name was really Johann von Nassau (Capps, 2004)

Some key signs of delusions

Despite the difficulties in defining delusion, and the complications with regard to the different psychiatric and medical conditions in which delusions can occur, as well as the variations in associated phenomenology, most clinicians will recognise a delusion when they see one, even though mistakes sometimes occur. Bell, Halligan and Ellis (2003) describe the case of Martha Mitchell, the wife of the American attorney general, whose entirely appropriate concerns about corruption in Nixon's White House were initially dismissed as delusional. The three inter-related key signs – properties of the belief - that clinicians will typically use to help diagnose the presence or otherwise of a delusion are: (1) impossibility or falsity; (2) incorrigibility; and (3) unwarranted subjective certainty (see Spitzer, 1990, 1992, for discussion).

The notion of falsity is problematic, as discussed earlier; but the idea of impossibility (or implausibility) need not be, although the precise meaning of this delusional impossibility remains disputed. The *DSM-IV-TR* reflects the attempt to shift from falsity to impossibility when it describes delusions as "erroneous beliefs that usually involve a misinterpretation of perceptions or experiences" (p. 299). The influence of Schneider (1959) is at work here. Schneider conceived of "delusional perception" as one of the first-rank signs of schizophrenia; in delusional perception, a normal everyday perceptual experience is said to become imbued with a special significance and meaning for no apparent logical reason. To the observer, there is something contextually incomprehensible in the manner in which the delusional content arises from the focus of

perception and the general context. Rather than focus on delusional perception, the nature of which is disputed (see, e.g., Spitzer, 1990), we will try to discuss these three signs in relation to the nature of the phenomenology, as experienced by the patient, and as it presents to the clinician.

Impossibility or implausibility is most obvious when the delusional content is blatantly fantastic. The belief that one is dead despite being able to walk and talk (Cotard delusion), or the belief that one has no internal organs (a somatic delusion), or the belief that one can see despite the presence of bilateral damage to the occipital lobes which causes a form of cortical blindness (Anton's syndrome), are impossible in this sense. More mundane delusions do not present with the same sort of sheer implausibility - delusions like the persecutory delusion that one's neighbours are plotting against one (such things can and do happen), or that one's partner is having an affair (delusional jealousy). In these latter cases, the impossibility is indicated by the contextual implausibility of the belief; the belief does not appear to derive meaningfully from the context, and the patient lacks evidence for the belief, or lacks the kind of evidence that would warrant the subjective conviction with which the belief is espoused. When probed as to why the patient believes as she does, she might be unable to provide any evidence to justify her belief, or, if "evidence" is produced, it is not compelling, and often not even relevant to the belief in question. One of us, R.L., once observed a persecutory patient who was asked about the last time that she knew her neighbour was intending to harm her. The patient replied by describing the irritating way in which the neighbour had intentionally jingled her keys in a provocative manner when walking in front of the patient.

Incorrigibility refers to the fixity of the delusion and its intransigent resistance to rational counter-argument and counter-evidence, and is likewise linked with unwarranted subjective conviction.

Unwarranted subjective conviction refers to the experience of the delusional patient. The delusional patient espouses her belief with a sense of absolute knowing, as if the delusional content could not be other, and so is beyond any requirements for objective

justification. The delusional belief appears to be experienced as self-evident, without the need for any justification of its truth, and the patient seems unable to even entertain the possibility that the belief might be incorrect (see, e.g., Spitzer, 1990, for discussion).

The key signs of incorrigibility and unwarranted subjective certainty, as described above, seem at odds with dimensional approaches, which allow, e.g., that the level of delusional conviction can vary, and that delusional fixity can give way to delusional confusion and uncertainty (see, e.g., Garety & Hemsley, 1994, for discussion). However, these approaches need not be incompatible if, for example, the dimensions of fixity and conviction refer to the waxing and waning of the delusional state, the core nature of which is characterised by impossibility (sheer or contextual), incorrigibility and unwarranted subjective conviction.

Heterogeneity and the cognitive-neuropsychiatric approach to understanding delusional belief

We have discussed the extreme heterogeneity of the various psychiatric syndromes in which delusions figure. There is etiological heterogeneity (delusions as seen in many different psychiatric and neuropathological conditions) and there is doxastic heterogeneity (many different kinds of content are seen in delusional beliefs, and a distinction between the monothematic category of delusions and the polythematic category exists). Because of this extreme heterogeneity, it is not appropriate to treat any of these syndromes as objects of scientific enquiry if one wants, as we do, to develop cognitive-level explanations of specific psychiatric symptoms. For example, if there is no psychiatric symptom or set of symptoms which every person with schizophrenia exhibits, what would it mean to ask "What is the impairment of cognition that causes the psychiatric symptoms of schizophrenia to arise?" This is why in the discipline of cognitive neuropsychiatry the unit of investigation is the symptom, not the syndrome. Cognitive neuropsychiatrists aim to account for specific psychiatric symptoms in terms of what remains intact and what has been disrupted in some mental information processing system that is relevant to the particular symptom in question. One might be trying to discover, at the level of cognition, what causes hallucinations, for example, or

what causes delusions. Even here heterogeneity exists and this has to be recognized: there are different kinds of hallucination (auditory, visual, olfactory, somatic) and as we have already discussed there are many different kinds of delusion. So in scientific studies of delusions, each different kind of delusion requires separate investigation. Does it follow from this that there can be no general theory of the cause of delusional belief, but only separate theories for the separate delusions? We now turn to a consideration of this question (our answer to the question will be: Yes and No).

A TWO-FACTOR THEORY OF MONOTHEMATIC DELUSION

Earlier in this chapter, we pointed out the need for theoretical accounts of delusion that can explain why some patients develop, say, persecutory delusions while other patients develop, say, somatic delusions. Put this way, the scientific task seems quite formidable. But in fact it is even more formidable than this point suggests, since the issue is not just the understanding of why a patient with a monothematic delusion has one particular specific delusional belief rather than another: there's the larger problem of understanding why some patients develop polythematic delusions – sprawling delusional systems that incorporate many and unrelated delusional beliefs – whilst others develop monothematic delusions, where there is often just one single belief (or set of beliefs about just one single theme) held by the patient that is a delusional belief, and the patient's cognition seems otherwise perfectly normal.

Rather than trying to start with the larger problem of understanding why some patients develop polythematic delusions whilst others develop monothematic delusions, we might try to make some initial progress by focusing on just one of these two broad categories of delusion; and that is the step that has been taken by many scientists working on delusional belief. Over the past twenty or so years, many have chosen just to investigate monothematic delusions. Even here, there seems to be a formidable scientific challenge; what possible explanation could there be for why so many different delusional themes can be seen in a set of patients with monothematic delusions? Why does patient A believe she is dead, whilst patient B believes his left arm is actually his

niece's arm, and patients C and D believe their spouses have been replaced by strangers or impostors? What determines the content of these monothematic delusions?

Challenging though these questions appear, a great deal of progress has been made over the past twenty years in obtaining answers to them. Seminal work by Ellis and Young (1990) and by Frith (1992), which led to the foundation of a new discipline now known as cognitive neuropsychiatry (Ellis, 1998; Halligan and David, 2001), offered clear promise for understanding in cognitive terms the nature and genesis of monothematic delusions. The Capgras delusion has been particularly important here.

Capgras delusion and the two-factor theory of delusional belief

Ellis, Young, Quayle and de Pauw (1997) investigated Capgras delusion from a cognitive-neuropsychiatric perspective, by studying autonomic responses to familiar (famous) and unfamiliar faces in sufferers from this delusion. It is known that autonomic responses to faces, as measured by changes in the skin conductance response (SCR), are larger when faces are familiar than when they are not, and Ellis and colleagues replicated this result with healthy control subjects and also with psychiatric control subjects, all of whom were delusional but whose delusions did not concern mistaken identities. But in a group of five patients with Capgras delusion, this effect of familiarity on autonomic responding was absent; SCRs to faces were no larger to familiar (famous) faces than to unfamiliar faces. Hirstein and Ramachandran (1997) obtained the same result; and Brighetti, Bonifacci, Borlimi and Ottaviani (2007) confirmed that this lack of an effect of face familiarity on autonomic responding to faces in Capgras delusion is observed when the familiar faces are not famous faces but the faces of the deluded person's family members, i.e. the faces of the very people about whom the patient is delusional.

Ellis et al. (1997) reported that their Capgras patients could overtly recognize photographs of every one of the famous faces used in their experiment. So the absence of SCRs to familiar faces is not due to an impairment of the face recognition system

itself. Nor is it due to an impairment of the autonomic nervous system itself, since these patients did not show any generalized autonomic hyporeactivity. Hence there must have been in these five patients a *disconnection* between the face recognition system (itself intact) and the autonomic nervous system (itself also intact).

When a patient with Capgras delusion sees his wife, he will still be able to know that the woman he is looking at looks just like his wife, because of the intactness of his face recognition system. He will therefore be confronted with a paradoxical situation: this woman certainly looks exactly like his wife, but if she were his wife, seeing her should have evoked an autonomic response. Yet no such response occurred. Why not?

The move from the absence of an autonomic response on seeing the wife's face to the thought "This woman is not my wife" involves an inferential step; but the type of inference involved is not deductive inference (establishing what conclusion logically follows from some set of premises) or inductive inference (inferring a general principle from a collection of instances). Instead, it is *abductive inference,* otherwise know as "inference to the best explanation": "Motivated by the observation of a surprising fact or an anomaly that disappoints an expectation, abductive reasoning is a strategy of solving problems and discovering relevant premisses. It is 'inference to the best explanation'. Abductive reasoning has the logical form of an inverse modus ponens and is 'reasoning backwards' from consequent to antecedent." (Wirth, 1998, p. 1).

The abductive inference to proposition P from datum D is a legitimate one if and only if it is true that, if P were the case, one would expect D to arise, As the logician C.S Peirce, who was responsible for the development of the concept of abductive inference, wrote (Peirce 1903/1997, p. 245): "It must be remembered that abduction, although it is very little hampered by logical rules, nevertheless is logical inference, asserting its conclusion only problematically or conjecturally, it is true, but nevertheless having a perfectly definite logical form. Long before I first classed abduction as an inference it was recognized by logicians that the operation of adopting an explanatory hypothesis -- which is just what abduction is -- was subject to certain conditions. Namely, the

hypothesis cannot be admitted, even as a hypothesis, unless it be supposed that it would account for the facts or some of them. The form of inference, therefore, is this:

The surprising fact, D, is observed;

But if P were true, D would be a matter of course,

Hence, there is reason to suspect that P is true[2]."

It is critical to note that this does not require that the proposition P itself be reasonable. It can be the case that the truth of P makes the fact D something to be expected (" a matter of course" in Peirce's terms) even if the proposition P is bizarre or impossible. In the Capgras example we are considering, the datum D (a "surprising fact" in Peirce's terms) consists of a lack of autonomic response when encountering a woman, and the proposition P is the proposition "This woman is not my wife"; and it is indeed the case that if P were true one would expect the datum D. Consequently, the abductive inference in the Capgras case is a legitimate one, even though the proposition it yields is bizarre.

We perhaps should say a little more about exactly what we mean by "legitimate" in relation to an abductive inference. If someone generates a proposition P by abductive inference from some datum D, it might be that D indeed would be expected if P were true (in which case the abductive inference process has functioned correctly). But it might also be the case that the truth of P gives no plausible grounds for expecting D (in which case the abductive inference process has malfunctioned). We define the term "legitimate abductive inference" as referring to the former of these two cases. Any abductive inference is legitimate when and only when there are reasons why the truth of the abductively-inferred proposition P should lead us to expect the occurrence of the datum D from which P was abductively inferred.

[2] For the sake of clarity we have here replaced Peirce's symbols for the datum and the hypothesis with ours.

As we have said, the plausibility of P is irrelevant here. The job of abductive inference is to generate propositions that, if true, would lead one justifiably to expect the occurrence of the data from which these propositions were inferred; the propositions are not required to have the additional property of truth, or even plausibility. The propositions yielded by abductive inference are not beliefs, but hypotheses or candidates for belief. For any such proposition to be adopted as a belief, it must be submitted to, and survive, a belief-evaluation process, and it is here that plausibility has a critical role. We, like Coltheart, Menzies and Sutton (2010), are attracted to the idea that this belief-evaluation process operates according to Bayesian principles (see also Corlett, Frith and Fletcher, 2009).

The idea that delusional beliefs are normal attempts to explain abnormal perceptual or affective phenomena goes back to William James: ''The delusions of the insane are apt to affect certain typical forms, very difficult to explain. But in many cases they are certainly theories which the patients invent to account for their bodily sensations'' (James, 1890/1950, chap. XIX), and more recently has been proposed by Brendan Maher: ''A delusion is a hypothesis designed to explain unusual perceptual phenomena'' (Maher, 1974, p. 103). Maher was originally concerned with delusions of reference, but his idea can be explored in relation to monothematic delusions: as we have seen, there is an unusual phenomenon related to the sight of a spouse's (or other familiar person's) face in Capgras delusion (the unexpected absence of autonomic response), and the abductive inference "That's not my spouse" provides an explanation of this unusual phenomenon.

So this seems a promising explanation for the Capgras delusion. However, it turns out to be insufficient. If this were the complete explanation of the delusion, then anyone who suffered any form of brain damage that prevented familiar faces from evoking responses of the autonomic nervous system would suffer from Capgras delusion; and that is not the case. Patients with damage to ventromedial regions of frontal cortex are like Capgras patients in that they too show no greater autonomic responding to familiar than unfamiliar faces (Tranel, Tranel & Damasio, 1995); but these patients are not delusional. One might therefore conclude that the autonomic disconnection

demonstrable in people with Capgras delusion is just coincidental i.e. irrelevant to the explanation of the delusion. But this seems too drastic a step to take because the content of the delusion has so strong a *prima facie* connection with the absence of autonomic response. If one does not take this drastic step, then there seems to be only one alternative. This is to propose that, whilst the absence of autonomic response is what suggests the delusional proposition as a candidate for belief via abductive inference, because this belief is not endorsed by ventromedial patients (as it shouldn't be, given its implausibility) but is endorsed by Capgras patients, there must be an additional cognitive deficit in the Capgras patients. This second deficit can be described as some form of impairment of belief evaluation processes. The delusional proposition arises (as a candidate for belief) via a legitimate abductive inference; nevertheless it should not be accepted as a belief because there is so much evidence against it (the sheer implausibility of the belief, the spouse's protests, the spouse's appearance, urgings from friends and clinicians that the belief is false, etc.). It has been suggested that this second deficit, impaired belief evaluation, is associated with pathology of some region in right lateral prefrontal cortex (Coltheart, 2007, 2010; Coltheart, Langdon & McKay, 2007; Langdon, McKay & Coltheart, 2008).

This is consistent with the two-step conception of the normal processes of belief formation involving abductive inference proposed by the philosopher of science Norwood Hanson: "Norwood R. Hanson (1965) differentiates between two aspects in the rational process of hypothesis selection: first, reasons for accepting a hypothesis, and second, reasons for entertaining a hypothesis in the first place. While the former highlights the problem of logical coherence, the latter stresses pragmatic relevance" (Wirth, 1998, p. 2)

The theory of delusion proposed by James and Maher was a one-factor theory, the one factor being the presence of some form of unusual perceptual phenomenon. But if the relevant unusual perceptual phenomenon in Capgras delusion is the absence of autonomic responding to familiar faces, then a one-factor theory will not work, because some people who show this absence are not delusional. So the James-Maher theory must

be embellished by adding a second factor, defective belief evaluation, if it is to provide a satisfactory explanation for Capgras delusion. Coltheart (2007) has discussed evidence that this second deficit is associated with damage to right lateral prefrontal cortex.

This two-factor cognitive neuropsychiatric approach (Langdon & Coltheart, 2001; Davies, Coltheart, Langdon & Breen, 2001) is not intended to apply only to Capgras delusion. It is meant to apply to all forms of monothematic delusion – or at least to all of those which plainly depend upon some form of neuropsychological deficit. The general form of this approach in relation to any monothematic delusion is as follows:

> *Step 1:* Try to discover (or at least hypothesize) some form of neuropsychological impairment in the patient which would generate some abnormal datum D, involving perceptual or affective processing, for which the patient will seek an explanation via abductive reasoning processes;
> *Step 2:* Demonstrate that the delusional belief held by the patient is a proposition P that is a legitimate abductive inference from D, the criterion for legitimacy being that it is the case that if P were true then D would indeed be expected;
> *Step 3:* Then try to discover a patient or patients having the same impairment (the impairment mentioned in (1) above) as the delusional patient, but without any delusion. This form of demonstration is what is needed to justify the claim that a second factor is required when monothematic delusions are being explained;
> *Step 4:* Seek evidence of pathology of right lateral prefrontal cortex in the delusional patient.

Since the impairment mentioned in *Step 1* above is what determines the content of the delusional belief, and different monothematic delusions have different content, it will be the case according to this approach that Factor 1 will vary from delusion to delusion. And since the impairment mentioned in *Step 4* above is what determines the failure to reject the delusional belief, and this failure is evident in all patients with monothematic delusion, the approach predicts that Factor 2 will be present regardless of the content of the delusional belief; there will in all cases be an impairment of belief evaluation

processes. Current evidence suggests that this impairment will be associated with right lateral prefrontal cortex pathology, but the belief evaluation system itself must involve a number of different cognitive subcomponents, and damage to any one of these subcomponents might disrupt inputs into the critical functioning of the right lateral prefrontal region. These different subcomponents will, of course, be realized in the brain as a neural circuit involving various brain regions only one of which might be located in right lateral prefrontal cortex.

We'll now briefly consider some other types of monothematic delusion and discuss the applicability to them of the two-factor theory, as embodied in steps (1) through (4) above.

Fregoli delusion

This is the belief that people known to me are following me around, but in disguise: that is why I don't recognize them. *Step 1*: It has been speculated by Ramachandran and Blakeslee (1998) that what causes this delusion is that the autonomic nervous system is *hyper*-responsive to faces, so that a strong autonomic response is evoked even by unfamiliar faces: that is the datum D. *Step 2*: If the apparent strangers following me were actually familiar people, that is a proposition P which if true would explain D: so the abductive inference from D to P is a legitimate one. *Step 3*: but the patient with left-hemisphere brain damage who experienced the faces of strangers as highly familiar (Vuilleumier, Mohr, Valenza, Wetzel, & Landis, 2003) was not delusional; so if the Ramachandran-Blakeslee hypothesis is to be retained it needs to be embellished by postulating a second deficit, a belief-evaluation impairment that is present in Fregoli delusion but absent in the case reported by Vuilleumier et al. (2003). *Step 4*: in Fregoli delusion, there should be abnormality of right lateral prefrontal cortex. This two-factor account of Fregoli delusion is of course largely speculative at present, but is perfectly testable: for example, if the pattern of SCR responses to familiar versus unfamiliar faces was found to be not significantly different between Fregoli patients and controls – that is, if in both groups SCRs were relatively weak to unfamiliar faces while being relatively strong to familiar faces - that would directly falsify this account.

Cotard delusion

Patients with this delusion express the belief that they are dead. *Step 1*: It has been speculated by Ramachandran and Blakeslee (1998) that what causes this delusion is that the autonomic nervous system is severely under-responsive to any form of stimulus, so that strong autonomic responses are never evoked: that is the datum D. *Step 2*: If I were dead, that is a proposition P which if true would explain D: so it is a legitimate abductive inference. *Step 3*: consider the neuropsychological disorder known as "pure autonomic failure" in which SCRs to stimuli which would normally evoke such responses (such as unexpected sudden touches or loud noises) are absent (see e.g. Magnifico, Misra, Murray & Mathias, 1998). Despite this generalized absence of autonomic response, such patients are not delusional. *Step 4*: in Cotard delusion, there should be abnormality of right lateral prefrontal cortex. Like the two-factor account of Fregoli delusion, this account of Cotard delusion is largely speculative at present, but it too is perfectly testable: for example, if SCRs to a variety of stimuli that would normally be arousing were measured in patients with Cotard delusion and found to be clearly evoked by such stimuli, that would directly falsify this account. Evidence consistent with the account comes from findings reported by Young, Robertson, Hellawell, de Pauw and Pentland (1992), whose patient with Cotard delusion showed contusions affecting temporo-parietal areas of the right hemisphere as well as some bilateral damage to the frontal lobe.

Mirrored-self misidentification

A patient with this delusion, when looking into a mirror, will express the belief that the person he sees in the mirror is not the patient, but some stranger. Two cases of this delusion, FE and TH, were studied in detail by Breen, Caine, Coltheart et al. (2000), and interpreted in terms of the two factor account. *Step 1:* the answer here was different for the two patients, though the consequence for their beliefs was the same. FE was shown on neuropsychological testing to have impaired face processing. TH was shown to have

mirror agnosia. For him, a mirror was a window (or a hole in the wall). For example, when he was looking into a mirror and an object was held up over his shoulder so that he could only see it in the mirror, and he was asked to touch it, he did not reach back over his shoulder: he repeatedly reached into or around the mirror to try to touch the object. *Step 2:* Re FE: because of his impaired face perception, the visual representation of the face in the mirror that he constructs will not match the long-term-stored visual representation he has of his own face or any other known face: this is a datum D. How might it be explained? The abductively-inferred proposition P "That is a stranger" would explain it, since if P were true D would follow. Re TH: because of his treatment of mirrors as windows, anyone he sees in a mirror must be in a different part of space than he is. Here the datum D is: "I am looking at someone in another room", and the proposition P is "This person can't be me, it's someone else, because the person is in another room". *Step 3*: there are many reported cases of impaired face perception who do not believe that the person they see when they look in the mirror is someone else, and there are cases of mirror agnosia who are likewise not delusional (Binkofski, Buccino, Dohle et al., 1999). *Step 4:* in mirrored-self misidentification, there should be abnormality of right lateral prefrontal cortex. Neuropsychological testing of FE and TH revealed that they were unimpaired on tests of left-hemisphere function but impaired on tests of right-hemisphere function (these tests did not allow precise within-hemisphere localization).

Somatoparaphrenia

This term refers to patients with paralysed left limbs (the paralysis being due to damage to the right cerebral hemisphere) who express the belief that their left limbs are not their own, but belong to someone else: this other person might be the neuropsychological examiner, or it might be someone known to the patient, who may not even be present in the room. *Step 1:* the patient registers the datum D that the limb on the left cannot be voluntarily moved, which prompts the question: why not? *Step 2:* if the limb belonged to someone else, then the patient would not be able voluntarily to move it. So if the proposition P "This is not my limb" were true the datum D "I can't move this limb" would be expected. Hence here P is a legitimate abductive inference from D. *Step 3:*

many patients with paralysed left limbs after right hemisphere damage are not delusional about limb ownership. When questioned, they will acknowledge that the left limb is their left limb and that the reason they can't move it is that they have suffered brain damage. *Step 4:* patients with paralysed left limbs will have right hemisphere damage whether or not they are somatoparaphrenic. Amongst such patients, it is predicted that this damage will extend to right prefrontal cortex only for those with somatoparaphrenia.

One complication we need to face up to here is that somatoparaphrenia is not the only delusion associated with paralysis. Some patients with left-sided paralysis have the delusion, not that their left arm belongs to someone else, but that their left arm is not paralysed: this is anosognosia for hemiplegia, discussed below. Our suggestion, elaborated further when we discuss anosognosia below, is that hemiplegic patients with somatoparaphrenia do possess the datum D "I can't move this arm" because, for example, they have intact somatosensory and motor feedback from that arm and so can detect that the arm does not move when they try to move it, whereas hemiplegic patients with the other delusion (anosognosia for hemiplegia) do not have such feedback intact, so that when they seek to move their arm and fail they have no direct evidence that their arm did not move. This is discussed further below.

Alien control delusion

People with this delusion express the belief that other people can control the movements of their bodies, against their wills. Mellor (1970) provided the example of a 29-year-old short-hand typist who described her actions as follows: ''When I reach my hand for the comb it is my hand and arm which move, and my fingers pick up the pen, but I don't control them … I sit there watching them move, and they are quite independent, what they do is nothing to do with me … I am just a puppet who is manipulated by cosmic strings. When the strings are pulled my body moves and I cannot prevent it" (pp. 17-18).

Frith (1992) offered a one-factor account of this delusion. He suggested that it arose through an impairment of the self-monitoring process that is responsible for our sense

of agency over our own movements. When I form an intention to execute some action, I create a representation of the sensory feedback that the execution of this action will create. Then I act, and receive sensory feedback, and compare the received and expected feedback. If these cancel out, that is the signal that the action was executed under my control, not someone else's.

Our argument is that this is an important component of the alien control delusion, but that a one-factor account is insufficient. *Step1:* Any failure of the monitoring system that prevents this cancelling-out process from occurring will generate an abnormal datum D – perceived movement without cancellation and hence without a sense of agency – that requires explanation. *Step 2:* if it were the case that the limb movement was caused by someone other than the patient, the patient could not in advance have computed the appropriate expected sensory feedback, and so no matching of expected and received feedback could be achieved. Thus if the proposition P "Someone else caused my arm to move" were true, the datum D (no cancellation) would be expected. So again P is a legitimate abductive inference from D. *Step 3:* In the condition known as haptic deafferentation, the patient gets no sensory feedback from any actions performed (Fourneret, Paillard, Lamarre, Cole & Jeannerod, 2002); but the patient studied by Fourneret and colleagues did not present with a delusion of alien control So if inability to match expected and received sensory feedback is implicated in the delusion of alien control, a one-factor theory is not sufficient: an additional factor – impaired belief evaluation – needs to be added. *Step 4:* The delusion of alien control is particularly associated with schizophrenia, and there is abundant evidence of frontal and right-hemisphere pathology in schizophrenia. What's needed though is specific assessment via brain imaging of the integrity or otherwise of right lateral prefrontal cortex in patients specifically exhibiting the symptom of alien control delusion.

SOME DIFFICULTIES FOR THIS TWO-FACTOR THEORY OF MONOTHEMATIC DELUSION.

The specificity of delusions

If all patients with monothematic delusions, regardless of the content of the delusion, are suffering from an impairment of a belief evaluation system, why don't they show more general effects of this impairment? Odd ideas occur to all of us all of the time; we prevent these from becoming odd beliefs via the operation of our belief evaluation systems. Shouldn't anyone with an impairment of belief evaluation develop a wide range of odd beliefs, rather than just one?

We propose the following answer to this question. It seems clear that in delusional conditions the belief evaluation system is impaired rather than abolished. For example, consider the fact that patients who have suffered a right hemisphere stroke and consequent left hemiplegia sometimes deny that their left limbs are their own (somatoparaphrenia). Administration of cold water to the left external auditory canal (a standard neurological technique for assessing vestibular function) can result in temporary abolition of this delusion: for some hours after this administration, patients will agree that their paralysed left limbs are their own, before eventually reverting to the delusion of somatoparaphrenia (Bisiach, Rusconi & Vallar, 1991; Rode, Charles, Perenin & Vighetto, 1992). Brain imaging studies have shown that cold caloric left vestibular stimulation produces right hemisphere activation (for a review of these studies see Coltheart, 2007, p 1055-1056), and even activation of right dorsolateral prefrontal cortex (Fasold, von Brevern, Kuhberg et al., 2002). Thus one might take the effect of left vestibular stimulation on patients with the delusion of somatoparaphrenia as evidence that the right-hemisphere belief evaluation system is not abolished (if it were, right-hemisphere activation could not improve its functioning) but only impaired.

Given this, perhaps odd ideas that sporadically present themselves can be rejected by even an impaired belief evaluation system. In contrast, the idea that prompts a monothematic delusion is persistently present: *every time* the Capgras patient sees his wife, there will be a mismatch between the expected and the obtained autonomic response, so there is a continuous ongoing presence of the abnormal datum that is explained by the Capgras abductive inference, and the effect of that datum can be continuously resisted only if the belief evaluation system is fully intact.

The intermittency of delusions

Coltheart (2007, pp 1053-1054) described two people with Capgras delusion in whom the delusion waxed and waned; on some occasions, these people identified their family members correctly, while on other occasions claimed that they were strangers or impostors. There are other reports (e.g. David, 1999; Sharp et al., 1996) of the same kind of waxing and waning of delusional belief. If monothematic delusions are caused by permanent neuropsychological damage, why aren't the delusions themselves permanent?

One way in which this might be explained is by having recourse again to the idea that in cases of monothematic delusion the belief evaluation system is impaired but not abolished. If so, then if sufficiently strong evidence contradicting the delusion is supplied, this might be able to outweigh the ever-present abnormal datum that is explained by the abductive inference leading to the delusional belief.

This argument works as follows. It is characteristic that, when someone expresses a delusional belief, concerned family members, friends and clinicians will try to persuade that person of the falsity of the belief; and sometimes such attempts at persuasion can be tactful yet incisive. One might ask a person with Capgras delusion: If this isn't your wife, how is it that she knows so many details about your past life? How is it that everyone else thinks she is your wife? How is it that she looks so much like your wife? Such questions can lead the patient to suspiciousness and even violence (Bourget & Whitehurst, 2004). But they may also lead the patient to doubt and then even reject the belief via use of the impaired but not abolished belief evaluation system. So the delusional belief is abandoned. Now those around the deluded person no longer feel any need to continue to provide evidence contradicting the delusional belief. But the abnormal datum that is explained by the abductive inference that yields the delusional belief is still being presented to the patient. So the delusional belief returns.

How could Cognitive-Behavioral Therapy (CBT) be effective in treating delusions if delusions are produced by neuropsychological deficits?

If, as has been argued in the previous two points, the belief evaluation system of a person with a monothematic delusion is impaired but not abolished, and therefore is still capable of responding to sufficiently strong or persistent evidence in favour of or contradictory to any belief, then there is nothing mysterious about finding that CBT can be effective in the treatment of delusion (see e.g. Landa, Silverstein, Schwartz & Savitz, 2006; O'Connor, Stip, Pelissier, Aardema, Guay et al., 2007) even if delusions result from neuropsychological damage. This is because the very essence of this therapeutic technique is to seek to introduce to the patient hypotheses that are alternatives to the delusional belief, and for patient and cognitive-behavioural therapist collaboratively to consider evidence that might support such alternatives to the delusional belief:

"Delusions are appropriate targets for a collaborative formulation approach. One commonly used technique to start the formulation process is known as 'peripheral questioning'. The clinician begins by asking a series of peripheral questions about the person's belief system, with the goal of understanding how the patient arrived at his or her convictions (e.g., 'How could others control your thoughts? What mechanism would they use?'). Peripheral questioning is linked with graded reality testing, which in turn can lead to the introduction of doubt and the generation of alternative hypotheses. Education about real-world issues can help patients understand the factual assumptions made to support their belief systems (e.g., 'Can microchips really be inserted without your knowledge when you are asleep?'). Such ideas can be explored with appropriate homework exercises (e.g., 'Shall we find out—perhaps on the Internet—what we can about the use of microchips in operations? Also we could check about regulations concerning such operations')." (Turkington, Kingdon & Weiden, 2006, p. 368)

Why does Capgras delusion occur only for individuals with whom the deluded person is *highly* familiar?

The autonomic nervous system responds more strongly to familiar than to unfamiliar faces even when the familiar faces are not those of highly familiar persons such as family members. But patients with Capgras delusion are typically not delusional about the identities of mere acquaintances, even though it will be the case that the faces of such individuals will generate weaker autonomic responses than they once did. Why does this discrepancy not result in the deluded person believing that these mere acquaintances are also strangers or impostors? All we can suggest here is that in such cases the discrepancy between expected and actual autonomic response will be small, and perhaps it is only when this discrepancy is very large that the stranger/impostor hypothesis is generated.

What is the intended scope of this theory of delusion?

All of the delusions we have discussed so far have clearly demonstrated or hypothesized organic and neuropsychological bases. But this is not so for other kinds of monothematic delusions, including some of the most common kinds of delusions such as delusions of persecution or reference. Is the two-factor theory of monothematic delusion meant to apply to such delusions? Only future work can answer this question; but two points are worth making here.

The first point is that some caution is needed in claiming that apparently non-neuropsychological delusions are in fact non-neuropsychological. Erotomania (sometimes called de Clerambault's syndrome) is the belief that someone of great fame or social status is in love with the deluded person but refuses to acknowledge this. People with this delusion hold strongly to the belief and are uninfluenced by any counterevidence (they may even agree that they and the object of the delusion have never met). It is hard to imagine what Factor 1 (some neuropsychological anomaly that gives rise to the delusional idea in the first place) could possibly be, so it is natural to think that this must be a non-neuropsychological delusion. And yet in a review of 29 cases of erotomania (Anderson, Camp & Filley, 1998) a number of cases were identified which provided varying degrees of evidence of a neuropsychological insult that seems to have precipitated the delusion. And there are even proposals that delusions of

reference arise through an impairment of the mesolimbic dopamine system that makes some environmental events appear much more salient than they should (Kapur, 2003): this could serve as Factor 1 in a two-factor neuropsychological account of delusions of reference. So: can we really be sure that there *are* any kinds of monothematic delusions that are not a consequence of some kind of neuropsychological damage?

The second point is that the two-factor account of delusional belief is not intrinsically committed to the idea that these factors stem from neuropsychological damage. The role of Factor 1 is to explain what initially suggested the delusional idea in the first place. It might be possible, for some monothematic delusions, to identify a plausible candidate for Factor 1 that does not involve neuropsychological damage. Consider the alien abduction delusion, for example: the belief that you have been abducted by beings from another planet and subsequently returned to Earth. McNally and Clancy (2005) studied ten people who had such a belief. All ten provided reports that the abductions happened after they woke up and experienced paralysis, odd sounds and sensations, and the feeling of other creatures present in their bedrooms. These sensations are not caused by neuropsychological damage: they are the symptoms of sleep paralysis and hypnopompic hallucinations and occur in a substantial proportion of the general (non-deluded) population. Given that if someone were actually being abducted by beings from another planet these are sensations that might be expected, it is not implausible to attribute the generation of the alien abduction idea to abductive inference concerning these sensations: that is, on this account Factor 1 is the sleep paralysis and hypnopompic hallucinations, which are not due to specific neuropsychological impairments.

But many people who experience sleep paralysis and hypnopompic hallucinations do not adopt the alien abduction belief. So we might argue that for this belief to be adopted some second factor must be present. This may not involve neuropsychological damage either, since there's no reason to believe that all people holding the alien abduction belief have any specific neuropsychological impairment. Furthermore, McNally and Clancy (2005, p. 120) note that not all individuals experiencing sleep paralysis seek explanations for it: some simply shrug it off as inexplicable. They then go on to say:

"It is unclear why some people opt for an alien abduction explanation . . . Our abductees did, however, entertain a wide range of 'New Age' beliefs (e.g. astral projection, foretelling the future) that might have made them especially prone to endorse an alien encounter interpretation of their sleep paralysis episodes."

We can use this observation to propose a Factor 2 that does not involve neuropsychological damage – namely, a predisposition to accept 'New Age' beliefs that is present to varying degrees in the general population. Thus we have a potential "non-neuropsychological-deficit" account of the alien abduction delusion. If (a) you experience sleep paralysis and hypnopompic hallucination, this can give rise, via abductive inference, to the thought of alien abduction and (b) you will reject this thought– you will not accept it as a belief – except when your belief system is such that this candidate belief is compatible with many other pre-existing things that you believe.

The problem of anosognosia

Anosognosia is a general term referring to conditions in which a person has some kind of impairment but denies this: see McGlynn and Schacter (1989) for a comprehensive review. Anosognosia occurs in various forms. In Anton's syndrome, the patient is blind because of damage to visual cortex, but asserts that he or she can see. In anosognosia for hemiplegia, the patient is paralysed on one side of the body (because of damage to motor regions of the contralateral hemisphere) but asserts that he or she can voluntarily move these paralysed limbs.

Anosognosic conditions do have the properties that we think of as characteristic of delusions. The belief is monothematic. It is firmly held. It is resistant to counter-evidence even though very clear counter-evidence is available (i.e. evidence of the presence of an impairment). And, just like patients with the forms of delusion we have already discussed, anosognosic patients will respond to counter-evidence by confabulating (i.e. seeking to explain the evidence away, often bizarrely).

But there is one crucial way in which anosognosic conditions differ from the other delusional conditions we have discussed. In the latter conditions, what the deluded people believe of themselves is something that is true of very few, if any, people. Not many people (if any) have had their wives replaced by impostors, or are dead yet can walk, or are susceptible to control of the movements of their bodies by other persons. That is why the beliefs held by people with Capgras delusion, Cotard delusion and the delusion of alien control are labelled "bizarre". Anosognosic conditions are the opposite. Almost all people can see (though not people with Anton's syndrome). Almost all people can voluntarily move all of their limbs (though not people with anosognosia for hemiplegia). So there is nothing at all bizarre about the contents of the delusional beliefs expressed by people with anosognosic conditions: as Davies et al. (2005, p. 226) say, here "The delusional belief is not new and exotic, but old and commonplace".

What could Factor 1 be in this delusion? What could prompt the idea, in a person with left hemiplegia, that "I can move my left arm"? Davies et al. (2005) have discussed in detail the nature of anosognosia and the possibility of offering a two-factor account of it. They argued (p. 228) that all that is required of Factor 1 here is "that it should be a neuropsychological anomaly that impairs the patient's awareness of his or her paralysis. Somatosensory loss and unilateral neglect would be candidates, as would damage to intentional-preparatory systems . . . or even a specific memory deficit that may be associated with damage to the right temporal lobe." Now, what's unusual here in terms of the two-deficit theory is that the hypothesised first deficit is not serving its usual function – which is explaining where the idea that becomes a delusion comes from in the first place. None of the possible first factors listed here (somatosensory loss, unilateral neglect, damage to intentional-preparatory systems) can be seen as plausibly generating any datum which, via abductive inference, would yield the proposition "I can move my left arm". But we need to offer an account of where that proposition comes from; and perhaps we can say here that this proposition is not something that is *introduced* by the patient's impairment but instead is a proposition that the patient has *always* believed (because of course it has always been true, up until the occurrence of the brain damage). The problem for these delusional patients then is not the presence of

some new idea, but the failure to reject some old idea, an idea which ought now to be abandoned in the light of the patients' acquired impairment; and this failure to revise the old belief arises because the new datum, which would lead to the abductive inference (correct in this case), "Accident or injury has caused impairment", is unavailable because, for example, of somatosensory loss. Patients cannot notice the absence of sensory feedback; they do not detect the abnormal datum (relative to their pre-existing belief system) that would lead to an abductive inference concerning their paralysis, or other impairment. Of course, they should nevertheless abandon the pre-existing belief that they are not paralysed, since they can see that their arms do not move when they are asked by an examiner to move them. That should prevent the adoption and persistence of the no-paralysis idea as a continuing belief: but this will not happen in patients where Factor 2 is also present, and of course all patients with left hemiplegia will have right-hemisphere damage which in some patients may extend to right lateral prefrontal cortex.

How are polythematic delusions to be explained?

It seems to us that the two-factor theory of monothematic delusion does a reasonable job in accounting for such delusions. But as we have noted, some patients exhibit polythematic delusions, i.e., they express a variety of delusional beliefs that are more or less unrelated to each other. How is polythematic delusion to be accounted for?

All we can do here is to offer several speculative answers to this question, answers that might be followed up in future research. We'll suggest three such answers.

Firstly, polythematic delusions might arise because multiple first factors are present. This is not inconceivable in the context of schizophrenia, where neurochemical imbalances and neural disconnectivities might affect a range of different functional systems, consistent with the evidence of a range of cognitive deficits in schizophrenia.

Secondly, polythematic delusions possibly arise when Factor 2 is particularly drastically impaired (or even abolished), as might occur in cases of dementia. We have argued

above that all sorts of odd ideas occur to people but can be prevented from being adopted as beliefs even when the belief evaluation system is impaired as long as it still has any capacity to function. But when this system is severely impaired or even abolished, there will be nothing to prevent various odd ideas from being adopted and retained as beliefs: so a polythematic delusional condition will develop.

Thirdly, polythematic delusions might arise when the abnormal datum is relatively ambiguous with regard to the possible abductive inferences that might be made – that is, when several propositions are generated by abductive inference all of which possess the desired property that if the proposition were true the datum generated by Factor 1 would be expected. In this case, the job of a belief evaluation system is to choose just one of these propositions for adoption as a belief. A severely impaired belief evaluation system may not be capable of selecting just one from a set of candidate-beliefs and rejecting the others in that set, so many or all of them may be adopted as beliefs, and over time such a patient will accumulate multiple different unrelated delusional beliefs.

CONCLUSION

Delusions are important to both the clinician and the scientist. We began by discussing the diagnostic manual most widely used by clinicians concerned with delusions, the *DSM-IV-TR*, focusing in particular on the definition of delusion that it provides. This definition clearly assists the work of the clinician, but it has not been as helpful to scientists, since it does not stand up very well to scientific or conceptual scrutiny: we considered six different objections that have been made to it. We then went on to describe the prevalence of delusion in various psychiatric and neuropathological syndromes (noting the extreme etiological heterogeneity: delusions are not uncommon in schizophrenia, dementia, Huntington's Disease, Parkinson's Disease and Traumatic Brain Injury) and to describe the various forms of delusional belief that occur (heterogeneity is even more extreme here: firstly there is the distinction between polythematic and monothematic delusional cases, and even within the category of monothematic delusion very many different delusional themes may be discerned). The cognitive-neuropsychiatric approach responds to the occurrence of such heterogeneity

by focusing not on psychiatrically or neuropathologically defined syndromes, but instead on specific symptoms – that is, on specific delusions.

We described a two-factor theory of delusional belief which aims to offer cognitive-level explanations of specific monothematic delusions such as Capgras delusion, alien control delusion, etc. The task of this theory in relation to any delusion is to offer answers to two questions: what prompted the initial thought which forms the content of this particular delusion, and why was that thought adopted as a belief when in the case of delusional belief there exists so much evidence contradicting that belief and when the belief itself is, as in some kinds of delusion, so bizarre? We considered six different kinds of monothematic delusion and showed that for each of these the two-factor theory offers plausible answers to both questions.

Various potential difficulties currently existing for this two-factor cognitive-neuropsychiatric account of monothematic delusional beliefs were then discussed, and possible solutions proposed. We concluded with a few remarks concerning possible ways of understanding polythematic delusional conditions.

FUTURE ISSUES AND UNRESOLVED QUESTIONS

1. What is the role of motivation in delusional belief? Is the presence of delusional belief sometimes due to motivational factors?
2. What is the appropriate scope of the two-factor theory of delusional belief described in this chapter?
3. How is the genesis of polythematic delusions to be explained?
4. For at least some of the monothematic delusions we have considered, the first factor that we posit can be thought of as a prediction error (for example, one predicts that the sight of someone who looks just like one's wife will be accompanied by a response of the autonomic nervous system, but this prediction fails in patients with Capgras delusion). Prediction error is a key concept in associative learning theory, and there is now considerable knowledge of the neural systems underlying the cognitive processing of prediction error (Corlett, Honey & Fletcher, 2007). Might the first factor for *all* forms

of monothematic delusion be conceptualized as the triggering of a delusional idea as an attempt to explain a prediction error? If that were so, it would establish important relationships between much work in the area of associative learning theory and much neurophysiological and pharmacological knowledge about the brain.

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