

**Delusions and Belief Formation:  
A Cognitive Neuropsychiatric Approach**

**Vaughan Bell**

School of Psychology, Cardiff University

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## Summary

There is no accepted definition of belief and it is likely that the everyday use of the term does not represent a single neuropsychological entity. Nevertheless, cognitive neuropsychiatry is likely to be useful in understanding belief-related phenomena, as it does not necessarily require the focus of study to be a unitary construct. The label 'delusion' is likely to be an umbrella term for a variety of pathologies that lead a person to make an unlikely belief-claim or to have an unlikely belief attributed to them on the basis of their behaviour, in tandem with the person experiencing significant distress and / or causing social stress. Social network analyses and case studies suggested that the traditional psychopathological boundaries of delusion are influenced by socio-cultural developments and that the diagnostic criteria need revising. Despite the central role of anomalous perceptual experience in many delusion formation models, it is unclear whether it is a necessary condition. A new, valid measure of perceptual anomalies, the Cardiff Anomalous Perceptions Scale (CAPS), was developed, and a study of delusional patients suggested that pathological levels of anomalous experience are not necessary for delusion formation. A principal components analysis suggested three factors underlying anomalous experience in the general population: 'clinical psychosis', 'chemosensation' and 'temporal lobe experience'. A study using transcranial magnetic stimulation indicated that disrupting the left lateral temporal cortex in healthy participants can alter processes related to magical thinking, suggesting these areas play a causal role in delusion formation. To investigate the determinants of pragmatically pathological beliefs, as opposed to simply 'magical' ones, participants with religious beliefs (Christians and Pagans) were compared to non-religious controls and delusional patients. Pagans reported similar levels of anomalous experience to psychotic patients, but were no more distressed than the general population, suggesting distress is the more important factor in delusion formation.

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## **Preface**

A recent review paper on persecutory delusions contained a section entitled “What must an adequate theory of persecutory delusions achieve?” (Bentall et al., 2001). Although the paper was focused on a subset of delusions only, the six given criteria are applicable to all attempts to explain delusional phenomena:

1. The phenomena to be explained should be clearly described and defined, and be reliably measurable.
2. The psychological processes invoked in order to explain the phenomena should be clearly defined and reliably measurable.
3. Research should support the hypothesised causal relationships between psychological constructs and psychological phenomena.
4. Empirical tests of models should involve behavioural and physiological data, as well as questionnaire and self-report measures.
5. Psychological models should explain how phenomena change over time.
6. Adequate models will explain the aetiology of psychopathology, by reference to either biological or environmental variables.

Although the authors admit that many of the criteria have still to be met, it is notable that they recommend a wide-ranging approach, aiming for a comprehensive explanation that cuts across several levels of explanation.

This thesis has attempted to abide by these recommendations by approaching the problem of delusions and belief using the techniques of cognitive neuropsychiatry, an eclectic tradition

stemming from cognitive neuropsychology, by which pathology is explained within normal models of functioning and conclusions are drawn from pathological cases to better specify the normal models. This makes reference to both biological and psychological levels of explanation, allowing for a thorough approach to understanding these complex phenomena.

The thesis initially approaches the problem by comprehensively reviewing and integrating the relevant literature on both normal belief and delusions, including evidence from philosophy of mind, philosophy of psychiatry, descriptive psychopathology, social psychology, cognitive psychology and neuropsychology. Although this consists of a more extensive review than is common for doctoral theses, an integrated approach to the literature has been neglected in the past, and, consequently, the first three chapters attempt to make amends for this omission.

Moreover, as the review makes clear, there are currently many gaps in the understanding of delusions and belief, from conceptual issues of definition to mechanisms of pathology and normal function. Therefore, the methodology used in the empirical chapters also aims to be comprehensive, tackling the problem at a number of levels, in an attempt to advance the understanding of delusions and belief in line with the six criteria.

## **Reference**

Bentall RP, Corcoran R, Howard R, Blackwood N, Kinderman P. (2001) Persecutory delusions: a review and theoretical integration. *Clinical Psychology Review*, 21, 1143-92



## **Chapter One**

### **Approaches to the Neuropsychology of Normal Belief**

Elements of this chapter are in publication as:

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Bell, V., Ellis, H. & Halligan, P. (2003) Neuropsychology, delusions and modularity: The curious problem of belief. *Proceedings of the British Psychological Society*, 11 (2), 174

## **1.1 Chapter outline**

Sections:

- 1.2 Why a neuropsychology of belief is necessary
- 1.3 Philosophy of mind and the neuropsychology of belief project: The possibility and boundaries of a neuropsychology of belief.
- 1.4 Approaches to the neuropsychology of belief
- 1.5 What should a cognitive model of belief formation include ?
- 1.6 Candidate neuropsychological models of belief formation
- 1.7 Chapter summary and conclusion
- 1.8 References

As the first of three literature review chapters, this will focus on neuropsychological methodologies and models of normal belief. The chapter will initially discuss the debate in the philosophy of mind over the nature and structure of belief, and the implications this has for a neuropsychological account of belief formation. Subsequently, the discussion will focus on current methodologies in belief research, before finally reviewing candidate neuropsychological models of belief formation

The following chapter (Chapter 2) focuses on reliability and validity of the clinical concept of 'delusion' and builds on arguments presented here to identify philosophical approaches to delusions compatible with neuropsychological explanations.

The third chapter will review current empirical models of delusions, with further chapters reporting empirical investigations, stemming from identified gaps in the field.

## **1.2 Why a neuropsychology of belief is necessary**

Belief is one of the most commonly used and consistently unexplained aspects of contemporary psychology. Bertrand Russell claimed it was “the central problem in the analysis of mind” (Russell, 1921, p231), and yet few attempts have been made to explain the structure of belief itself.

This is despite the fact that belief is understood to form a central part of our psychological explanations of behaviour. From the day-to-day discussion of other people’s intentions and convictions (often called, somewhat disparagingly, ‘folk psychology’) to the academic theories of thought and action that would otherwise wither and die were it not for the assumption that beliefs are the indivisible components of mental life.

Important areas of human science and medicine also rely on the concept of belief, psychiatry being the most relevant of these areas to this thesis. The idea that a belief can be pathological is of central importance in psychiatry, and delusions are typically identified as the ‘sine qua non’ (essential element) of psychosis. This relies on the assumption that delusions are different from normal beliefs, although as Harper (2004) has noted, there is usually little supporting evidence presented about normal beliefs, seriously detracting from an understanding of what delusions are, and how they are formed, maintained and resolved.

Unlike most other areas of psychology, cognitive neuropsychology typically avoids formal discussion of belief. This is likely due to a number of reasons, not least because researchers have had their hands full with less nebulous processes such as memory, attention, language and perception, even free will and intentionality seem to get more consideration.

When free will and intentionality seem less troublesome to neuropsychology than the concept of belief, we may be forgiven for asking exactly what is so difficult about belief? After all, we often discuss what we believe but rarely feel the need to discuss how we generate our own actions.

This difficulty is one of the most hotly-debated issues in contemporary philosophy of mind and the first part of this chapter will discuss the implications of this debate for a cognitive neuropsychiatry of belief.

### **1.3 Philosophy of mind and the neuropsychology of belief project: The possibility and boundaries of a neuropsychology of belief.**

The simple statement “I believe that...” belies the large number of possible interpretations of what is actually happening in the mind and brain when someone makes such a claim. Perhaps, one of the most remarkable things about belief is simply that we can use such straight-forward statements in everyday life without completely overloading the listener with the cognitive effort of interpreting what is said. While the number of possible interpretations may cause surprisingly little distraction for the casual listener, it causes a great deal of consternation for the cognitive scientist who needs to first define a process to be able to understand how it may operate and be supported by the neural systems of the brain.

To bring the issues into stark relief, we can start with the most striking view: an understanding of the neuropsychology of belief may not even be possible. This, it is argued, is not because we do not have the conceptual tools, or the appropriate technologies of investigation, or even the inclination, but because the very idea of ‘belief’ is redundant, no more useful than talk of an excess of ‘black bile’ causing depression, or ‘vital force’ being the essence of all living

things. This is, of course, only one position (known as eliminative materialism or eliminativism; Churchland, 1981; 1999), but the fact that the issue remains unresolved should perhaps give us cause for thought.

The issue at stake is not whether we find the concept of belief useful in everyday communication, but whether this everyday understanding of belief is scientifically valid and should be used as the basis for any sort of scientific investigation or theory, especially those that seek to link belief to brain states. The problem is widely discussed, largely among philosophers of mind rather than psychologists, perhaps who would rather not throw any stones lest their house turns out to be made of rather more glass than they realised.

It is worth noting when reviewing these theories, that it is often difficult to make a clear distinction between questions of whether a neuropsychology of belief is possible, and what the likely structure of belief might be. Although these questions may be conceptually distinct, in practice, the latter tends to have considerable implications for the former. Because of this, these questions are tackled together in the section below.

In this vein, Baker (1987) has identified the four major views on the 'correctness' of the everyday understanding of belief and how it may relate to underlying neural processes. In addition, dispositional accounts of belief have also seen a revival in recent years, and each of these five approaches is outlined below. Although compatible with several of these approaches, a further question over whether beliefs are discrete entities also has implications for any neurological basis for belief and so will also be discussed.

### **1.3.1 Our common-sense understanding of belief is correct: The representational approach**

The common-sense view of belief tends to equate beliefs with explicitly held propositions. Here, such propositions are available as representations in memory and consulted when an appropriate situation arises. Among philosophers who have argued for beliefs as representations, Fodor (1975) is a particularly well known exponent. Controversially, he has argued that a fundamental ‘language of thought’ – independent of the actual spoken language of the believer – underlies the representation of belief (and other mental states). A discussion of his ‘language of thought’ hypothesis is outside the scope of this chapter, although his syntactic description of belief is not without its critics, and the strength of the passion it engenders can be seen from a riposte by Still and Costall who went as far as to describe Fodor’s theories as “where one tries to keep a reasonably straight face while presenting the absurd consequences of the scheme as exciting theoretical revelations” (Still and Costall, 1991, p2).

Nevertheless, the ‘belief as representation’ hypothesis does not necessarily entail subscribing to an underlying language of thought. Both Armstrong (1973) and Dretske (1988) have argued that beliefs may be stored as semantic maps, an idea which seems a good deal less speculative now, in light of recent advances in understanding how neural systems might encode even high-level information topographically or in multi-dimensional arrays (Lloyd, 2000; 2002) than it may have seemed when first introduced.

### **1.3.2 Our common-sense understanding of belief may not be entirely correct, but it is close enough to make some useful predictions: Stich's (1983) approach.**

This view argues that we will eventually reject the idea of belief as we use it now, but that there may be a correlation between what we take to be a belief when someone says "I believe that snow is white" and how a future theory of psychology will explain this behaviour. In a way, this is a 'missing link' argument which suggests that we talk about circumscribed beliefs and particular brain states, but are missing an important conceptual link which will change our understanding of how the two are connected and will cause us to rethink our model of belief (or even eliminate it). We can perhaps draw an analogy between how we now understand hemi-spatial neglect in terms of an attentional deficit to one side of space; and how, through the ages, the same behaviour was undoubtedly explained as partial blindness. The traditional explanation may be no longer sustainable under rigorous scientific investigation, but some of the practical implications may be similar. Most notably, Stich (1983) has argued for this particular understanding of belief (with his 'panglossian' approach) and whether a project linking them with neural underpinnings will be successful.

### **1.3.3 Our common-sense understanding of belief is entirely incorrect and will be superseded: The eliminativist approach.**

Known as eliminativism, this view, most notably proposed by Churchland (1981; 1999), argues that the concept of belief is similar to obsolete theories of times past, such as the four humours theory of medicine, or the phlogiston theory of combustion. In these cases, science has not provided us with a more detailed account of these theories, but completely rejected them as valid scientific concepts to be replaced by entirely different accounts. Churchland argues that our common-sense concept of belief is similar, in that as we discover more about neuroscience and the brain, the inevitable conclusion will be to reject the belief hypothesis in

its entirety. Although Churchland's account might make grim reading for anyone wishing to use the concept of belief in any sort of explanatory framework, one implication of this view is that our current concept of belief will be replaced by a number of better specified neuropsychological theories.

#### **1.3.4 Our common-sense understanding of belief is incorrect, but treating people, animals and even computers as if they had beliefs, is often a successful strategy: The interpretational approach.**

Dennett (1999) and Baker (1987) are both eliminativists in that they argue that beliefs are not adequately reducible to their neural underpinnings, but they do not go as far as rejecting the concept of belief as a predictive device. Baker (1987, p150) gives the example of playing a computer at chess. While few people would agree that the computer held beliefs, treating the computer as if it did (e.g. that the computer believes that taking the opposition's queen will give it a considerable advantage) is likely to be a successful and predictive strategy. In this understanding of belief, called 'interpretationism', or in Dennett's terminology, taking the 'the intentional stance', belief-based explanations of mind and behaviour are at a different level of explanation and are not reducible to those based on fundamental neuroscience, although both may be explanatory at their own level.

#### **1.3.5 Dispositional approaches to belief**

In addition to Baker's (1987) four approaches to belief, other authors have argued that beliefs are best understood as dispositions to act in a certain way in certain circumstances, and argue against the common-sense, representational view of belief on the implausibility of its consequences. If you were asked "do you believe tigers wear pink pyjamas ?", you would say (presumably) you do not, despite the fact that you may never have thought about this situation



before. You are also likely to believe that there are more than two people in a duet, as well as believing that there are more than three people in a duet, more than four... and so on, ad infinitum. Proponents of the dispositional view of belief (such as Marcus, 1990) claim that such examples show that beliefs cannot simply be representations of 'facts' in the brain, as it would be impossible to have an infinite number of beliefs stored in finite neural structures. They claim that beliefs are therefore dispositional and only ascribable to observable behaviour in others.

Initially, this seems like a behaviourist approach to belief, and, unsurprisingly, was initially championed by philosophers such as Gilbert Ryle (1949), who were sympathetic to many of the goals of behaviourist psychology. Others (such as Schwitzgebel, 2002) have argued for a more liberal and less strictly behaviourist interpretation of the dispositional account, where dispositions could include non-observable behaviour and responses, such as emotional reactions or cognitive reorganisation. This approach would also suggest our current conception of belief is realised as multiple neuropsychological systems, as dispositions could be understood as the likely engagement of other neural systems when belief-based action or reasoning is used.

### **1.3.6 Are beliefs discrete entities ?**

The views expressed above can be thought of as discussions over the internal structure of belief and / or its supervenience on neural systems. A further issue concerns whether it makes sense to conceive of beliefs as independent entities (i.e. circumscribed propositions), or whether beliefs only exist as coherent entities when conceptualised as part of a network of other beliefs. The former position is known as atomism, the latter holism, and the distinction becomes clear when the issue arises of whether beliefs can be considered identical for any

given referent. For example, Price (1934; 1969), an atomist, conceives beliefs as single propositions, so two people would be considered to hold the same belief if they both assent to the same belief sentence (e.g. “snow is white”). Alternatively, proponents of the holist view (Davidson, 1973, 1984; Quine and Ullian, 1970) argue that beliefs can only be understood in terms of their relation to other beliefs. For example, if two people express the belief that “snow is white” but one believes it is made of star-dust while the other believes it is frozen water, a holist would argue that they are not expressing the same belief, as they have radically different conceptions about the nature of snow.

This issue is important for neuropsychology, as it potentially defines the link between individuals’ belief claims and how they relate to the underlying neuropsychological processes which support them. For example, an atomist would expect that similar neural activation would occur for an identical task that relied on the same belief that  $p$ , regardless of whether additional beliefs relating to  $p$  are accepted or rejected over time. In contrast, a holist might expect brain activity to be radically different if and when new beliefs relating to  $p$  are acquired, as  $p$  can only be understood in the context of its interconnectedness with other beliefs. Therefore, a holist view is more likely to support a multi-factorial neuropsychological account of belief, as multiple processes or multiple sites of neural activation might be involved by nature of the fact that activation of any particular belief would involve the activation of related beliefs.

This tradition might see belief as simply a linguistic label for a number of disparate cognitive processes, suggesting that the neural underpinnings of what we understand someone to be doing when they say “I believe that...” to be many and various, and best explained as a

complex system of more fundamental neuropsychological processes (Horgan and Woodward, 1985).

### **1.3.7 Philosophical approaches to belief: Conclusions**

One dominant theme which arises from these theories is that there is unlikely to be a unitary belief formation process that could be explained by a monolithic neuropsychological model. While eliminativists would argue that any such model will eventually become so fragmented as to make any talk of belief redundant, there is no doubt that whichever theory one subscribes to, there are many potential processes that could lead to a belief claim.

Another important conclusion that can be drawn from the above is that the neuropsychological study of belief may be able to address some of these philosophical concerns. As each of these theories can be seen as making predictions about the likely neural involvement in belief processing, these can be compared to experimental data to provide evidence for the ongoing debate in the philosophy of mind. It is to these sorts of empirical approaches to which we now turn.

## **1.4 Approaches to the neuropsychology of belief**

The arguments underpinning the views expressed in the previous section have been debated in the literature in more detail than is appropriate to discuss here, nevertheless, none are entirely without merit. Many of these arguments are explicitly addressed to physicalism, and do not make a distinction between beliefs being explained as neurological phenomenon (based entirely on the biology of the brain) and neuropsychological explanations, which are more likely to argue for an information processing approach, based on models of neural function, rather than a purely physical account.

Although it is by no means clear, it is hoped that neuropsychology could be the ‘conceptual glue’ that will eventually bind the physical and psychological levels of explanation together, potentially solving the ‘problem of belief’ (or at least the problem of how to approach it) in its wake. This section will therefore assess the current tools of cognitive neuropsychology and evaluate them in light of their past successes in tackling the problem of belief.

#### **1.4.1 Functional neuroimaging**

Considering that “theories in the cognitive sciences are largely about the beliefs organisms have” (Fodor, 1981), we might expect that the majority of functional neuroimaging research to have significant implications for locating the neural processes underlying belief. Until recently, however, the explicit study of belief is not something that has seemed particularly to interest functional imaging researchers, with the exception, perhaps, of research into ‘theory of mind’ (the theorised ability to represent another person’s beliefs and intentions) which has been investigated many times in this way.

Indeed, ‘theory of mind’ is often touted in terms of belief (or false belief) yet it is not clear how explicitly these studies tackle the issue. As Dennett (1978) has noted, in many instances predicting others’ behaviour can be achieved without a ‘theory of mind’ and can be completed simply by observing the actual state of the world. Similarly, the relationship between the false belief task and ‘theory of mind’ is unclear, despite the fact that many studies conflate the two (Bloom and German, 2000), making ‘theory of mind’ research a potentially poor candidate on which to base any general theories of belief.

Part of the problem is that belief is a particularly difficult problem to tackle with imaging methods. We can conduct memory experiments in a scanner because there is an activity (remembering) associated with the construct of memory; as yet, however, there is no such identifiable activity as believing. Since functional imaging typically relies on the comparison of task-related activation, belief would seem unsuited for imaging research. Nevertheless, some recent studies have managed to tackle the problem by devising tasks that tap belief indirectly to examine how these interact with other tasks and processes.

One novel and ingenious approach has been taken recently by Goel and Dolan (2003) who conducted an fMRI study using Evans, Handley and Harper's (2001) belief modulated reasoning paradigm. Evans' group found that syllogistic reasoning is impaired when the outcome of a problem is in conflict with an individual's belief, despite being correct in the context of the presented problem (e.g. No addictive things are inexpensive. Some cigarettes are inexpensive. Therefore some cigarettes are not addictive).

Goel and Dolan compared brain activation between syllogisms where the correct answer was in agreement with the participants' beliefs, those which were belief discordant, and a condition using non-belief reasoning (e.g. All A are B; All B are C; therefore All A are C) to elicit activation specifically related to the effect of belief on reasoning. They reported left temporal activation for belief-based reasoning, and ventral medial prefrontal cortex activation when pre-existing beliefs caused reasoning to go awry, whereas successful suppression of belief for successful reasoning was associated with right lateral prefrontal activation. They concluded that belief may affect reasoning through emotional processing mechanisms (known to involve the medial ventral prefrontal cortex), a conclusion not unrelated to much of Frijda's work on the links between emotion and belief (Frijda, Manstead and Bem, 2000).

While Goel and Dolan's study could be seen as neutral with regard to a philosophical theory of belief (belief as representation, disposition etc), a study by Gallagher et al. (2002) specifically tackled belief by attempting to image the 'intentional stance' (Dennett, 1999). In this study, participants were asked to play the game "paper, scissors, stone" against an opponent whose responses were displayed on a computer screen. In one condition, participants were told that they were playing against a human opponent, in the other, the responses were randomly generated by a computer. In fact, in both conditions the responses were randomly generated, but when participants believed they were playing against a human there was significantly greater bilateral anterior paracingulate activation, which, the researchers suggest, shows a specific neural response for taking the 'intentional stance'.

Whilst these are exciting results in themselves, of greater interest is the use of indirect methods (the 'belief modulation' and the 'intentional stance' paradigm respectively) to tackle the cognitive neuropsychology of belief. Although this suggests that (like the executive system) belief might only be successfully tackled through its effect on secondary processes (Burgess, 1997), the imaging of belief is still in its infancy and such studies are best thought of as interesting, albeit exploratory, forays into a developing area.

#### **1.4.2 The lesion method**

The lesion method has proved to be a revolutionary tool in understanding the mind and brain. One of the main assumptions is that the mind, or at least parts of it, are organised as a series of co-operating but encapsulated modules (Fodor, 1983). Brain injury may selectively damage these cognitive modules, so their existence and function may be inferred from the experience, abilities and behaviour of post-injury patients (Shallice, 1988).

Ideally, we would like belief to be organised as a module or a number of modules, so the study of brain injury (or the application of other module-friendly experimental methods) would lead to a full understanding of how the belief-system operates. However, the work of Fodor (1983), on which much of this modularity thesis is based, makes an exception for certain sorts of psychological processes he defines as non-modular and 'central'. He further suggests that belief formation is one of these 'central' processes (particularly with regard to a lack of information encapsulation, see further discussion in Fodor, 2000), and should therefore be regarded as qualitatively different from other processes; and most importantly for our discussion, unassailable by studying the breakdown of cognition after pathology.

Despite Fodor's dire predictions, Bell, Ellis and Halligan (2003) have sounded a note of optimism, pointing out that useful parallels can be drawn between the successful and ongoing research into the executive system and attempts to understand the neuropsychology of belief. They draw inspiration from Burgess' (1997) analysis of methodology in executive function research, who noted that the executive system, as it is currently understood, fulfils all of Fodor's criteria for a 'central' process. Yet substantial progress has been made in adequately defining it (both theoretically and for the pragmatic purposes of measurement and assessment) and, furthermore, in understanding its function and role in thought and behaviour. One of Burgess' most salient points is that there is no direct 'process-behaviour correspondence', so the executive system can only be seen to be working indirectly through the measurement of other cognitive processes with which it integrates. If belief is indeed a central process this may be an important point to remember, as any attempts to find a single point of measurement (or single point of breakdown) may be doomed to failure.

At first sight then, it may seem paradoxical that one avenue that has proved successful is the study of delusions (one of the so-called ‘pathologies of belief’). The application of cognitive neuropsychology to psychiatric symptoms (‘cognitive neuropsychiatry’) attempts to understand normal psychological function by studying psychopathology, and to explain psychiatric symptoms in terms of normal models of neuropsychological function (David and Halligan, 1996; Ellis, 1998; Halligan and David, 2001). Early successes have produced specific and plausible mechanisms for how certain pathological beliefs might arise, most notably for the Capgras delusion (Ellis and Young, 1990; Ellis and Lewis, 2001; Hirstein and Ramachandran, 1997). In this account, damage to an unconscious or covert face recognition pathway is impaired, leaving Capgras sufferers without the appropriate emotional response to familiar faces, potentially explaining why they come to believe familiar people have been replaced by identical-looking impostors.

Unlike physical lesions, however, delusions are not easily quantified or defined. Despite being typically treated as pathological versions of common-sense belief, it is becoming increasingly clear that they are far more multi-faceted than the way they are typically represented in mainstream psychiatry (Bell, Halligan and Ellis, 2003; David, 1999). Although this has obvious disadvantages in terms of agreement between research groups (and even between researchers and clinicians) about the object of enquiry, it does make it unlikely that delusions qualify as the ‘single points of failure’ as criticised previously, and therefore perhaps, makes the seemingly ‘paradoxical progress’ of the cognitive neuropsychiatric study of delusions understandable.

Indeed, variance in pathological presentation is not a new issue to cognitive neuropsychology, where the debate over whether inferences about normal function should be drawn from single



or group studies (Caplan, 1988; Caramazza, 1986) has been of central importance. This debate has been largely over the contribution of these factors to variance in the dependent variable from which inferences are drawn. Despite some polarised opinions, it has become clear that variance in pathology is not necessarily a problem in itself for either approach, as long as the advantages and disadvantages of each methodology are known and inferences are drawn on the basis of converging evidence (Shallice, 1988).

Similarly, the debate over whether symptom or syndrome classifications (Ellis, 1987) should be used as the basis of neuropsychological investigations has been an important influence on current methodology. As the history of neuropsychology shows, awareness of these issues has led to impairments originally thought of as unitary disorders, being better understood in terms of symptom clusters and deficits in cognitive sub-systems (for example, double dissociations in prepositions versus definite articles found in Broca's aphasia; Miceli et al., 1989).

Therefore, delusions can, and have been, successfully investigated with group studies as well as single case approaches, each with the assumption that delusions vary in their pathological presentation and that they may be eventually be shown to be either symptom or syndrome-like (i.e. the expression of pathology in multiple, or even distinct cognitive systems) without jeopardising their validity in the research of normal belief.

#### **1.4.3 Neuropsychological correlates of belief states**

One aspect of belief that seems to have attracted much attention is paranormal belief, not least because of its potential links with religion and psychosis (Persinger and Makarec, 1990; Peters, 2001; Peters et al., 1999) both of which may involve beliefs that (at least at the surface level) share some similarities.

Paranormal belief has also been particularly linked to increased right hemisphere activation, and a reduction in left hemisphere dominance for language. Whilst Crow (1997) has controversially argued that this pattern is associated with the expression of frank psychosis and schizophrenia, it is certainly clear that this asymmetry of activation is also associated with paranormal beliefs that are not accompanied by a psychiatric diagnosis. It has variously been reported that a simple belief in extra-sensory perception (ESP) is associated with measures of increased right hemisphere activation (Brugger et al., 1993a, 1993b) a finding which has also been replicated with measures of paranormal belief and neuropsychological measures such as EEG (Pizagalli et al., 2000), olfactory discrimination (Mohr et al., 2001) and line bisection (Taylor et al., 2002).

Leonhard and Brugger (1998) have argued that this pattern of activation signifies an over-reliance on right hemisphere processes, whose coarse rather than focussed semantic processing may favour the emergence of 'loose' and 'uncommon' associations. They argue that these effects occur on a continuum: in some people they may contribute to novel thought and creativity, and, potentially, psychosis in the most extreme expression of the continuum. It is unlikely, however, that the degree of 'illness' is a simple correlation with degree of hemispheric asymmetry as many other factors (such as socio-cultural factors, individual history and coping styles) may contribute to such cognitive influences, beliefs and experiences being impairing, distressing or disabling rather than considered as normal belief (Bentall, 2003; Johns and van Os, 2001).

One surprising aspect is that work on hemispheric asymmetries and paranormal belief has rarely been linked to the conceptually similar work on temporal lobe disturbance and comparable beliefs and experiences.

Of particular interest is the work of Persinger who has amassed considerable converging evidence that temporal lobe activity, particularly on the right, is associated with paranormal belief (the term 'paranormal' is used here in its widest sense, also to include religious and mystical beliefs). In particular, Persinger (1983) has argued that mystical experience and religious beliefs are the normal consequence of transient activity in the tempero-limbic structures and has subsequently reported that the strength of paranormal beliefs is positively correlated with the amount of activation in these structures. He has reported this correlation in individuals engaged in psychic studies (Persinger and Fisher, 1990), during specific episodes of glossolalia ('speaking in tongues') and transcendental meditation (Persinger, 1984) as well as with a more general measure of paranormal experience (Persinger and Valliant, 1985).

Methodologically, this has often been tackled by developing a psychometric questionnaire based on the phenomenology of temporal lobe disturbance, and subsequently validating it by correlating the psychometric scale score with appropriate neurocognitive measures of temporal lobe disturbance (in this case EEG; Makarec and Persinger, 1985; 1990) or by stimulating the temporal lobes with weak complex magnetic fields to elicit similar experiences (Persinger and Healey, 2002; Cook and Persinger, 1997; although see Granqvist et al., 2005; Persinger and Koren, 2005).

The major implication of these neuropsychological findings is that widely-held religious, mystical or paranormal beliefs are associated with specific patterns of cortical and subcortical

activation. These can be studied by developing standardised psychometric questionnaires which can be the basis of drawing neuropsychological inference, as long as they are well validated by the appropriate application of cognitive neuroscience.

#### **1.4.4 Approaches to the neuropsychology of belief: Conclusions**

The three approaches discussed above (functional neuroimaging, the lesion method, and examining correlates of belief states) are all valid approaches to the neuropsychology of belief. Functional neuroimaging research in this area is still embryonic, however, so a more prudent approach would be to tackle the other areas first (particularly due to the importance of formulating well-specified questions before neuroimaging can be used to draw strong conclusions; Kosslyn, 1999).

Combining these two approaches has particular benefits. The 'lesion method' allows researchers to examine and test the predictions of current theories and definitions in light of individual pathology and group effects, while the use of correlational studies allow broad conclusions to be drawn that do not necessarily need a strict definition of pathology and may be more widely applicable. Finding such neuropsychological correlates of belief states allows for an understanding of trends and patterns of distribution, allowing pathology to be placed in the context of population effects and non-pathological analogues.

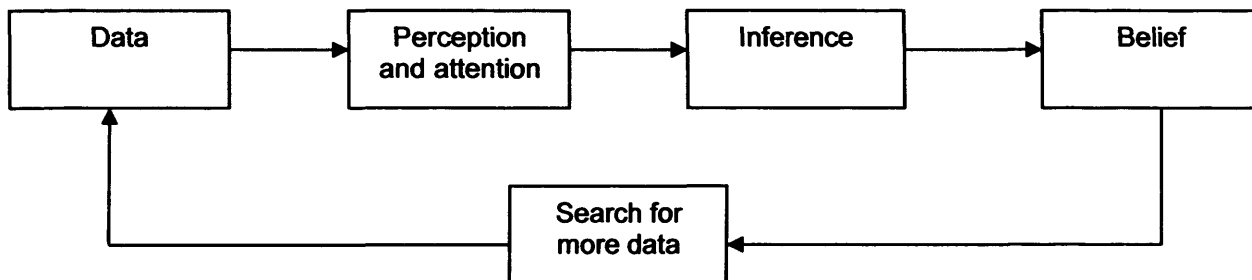
This suggests that the problem needs to be tackled at a number of levels, to combine an understanding of the phenomenology with converging evidence from neuropsychological approaches.

## 1.5 What should a cognitive model of belief formation include ?

Although it is common for psychological models to include belief within wider models of psychological function, formal models of belief are rarely discussed in terms of their necessary components. The following analysis identifies the essential elements of belief formation, viewed from the necessity of a cognitive approach.

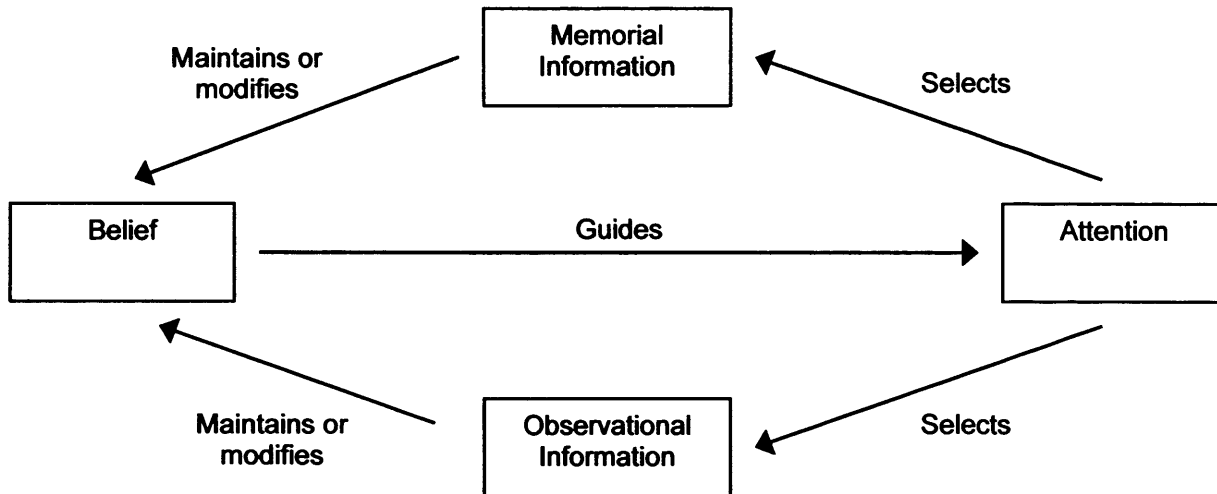
### 1.5.1 A contrast with intuitive / cyclic models of belief formation

Bentall (1990) produced a simple cyclic model (figure 1.1) which captures some of the ‘intuitive’ aspects of belief formation (although Bentall himself admitted that it is a somewhat ad hoc theory - literally conceived on the “back of an envelope”).



*figure 1.1 - Bentall's (1990) "back of an envelope" model of belief formation*

Clore and Gasper (2000) argue for a similarly cyclic model (figure 1.2), although this time in the context of explaining how affective modulation of attention might affect belief formation. In their model, there are two identical cycles running in parallel, one selecting information from perception, and the other from memory, to update and modify belief.



*figure 1.2 - Clore and Gasper's (2000) model of belief formation*

### **1.5.2 Conscious and unconscious processes in belief formation**

There are several reasons why such simple cyclic models might be considered inadequate. Philosophers have long argued that we cannot and do not choose our beliefs (review in Engel, 2002). For example, we cannot decide to believe that it is snowing in Devon unless we have evidence that it is – or more obviously, disbelieve it is raining if we are getting rained on. This suggests that an adequate model of belief formation must involve an explicitly unconscious component, as well as a process of conscious evaluation.

### **1.5.3 The role of the 'web of belief' and the influence of context**

Additionally, as Quine and Ullian (1970) noted (and regardless of their view on the atomism vs holism debate; section 1.3.6), beliefs must also be somehow integrated into our current web or system of belief, which may itself be changed as new beliefs are added (for example, some beliefs may be mutually exclusive). Indeed, the need to account for a 'web of beliefs' in belief formation processes has long been recognised in a diverse range of fields. Philosophically, the importance of belief networks have been stressed many times. This is perhaps stated most

succinctly in *On Certainty*, Wittgenstein's (1975) posthumously-published paper on belief and knowledge [italics in the original]:

141. When we first begin to *believe* anything, what we believe is not a single proposition, it is a whole system of propositions. (Light dawns gradually over the whole.)

142. It is not single axioms that strike me as obvious, it is a system in which consequences and premises give one another *mutual* support.

Apart from eliminative materialists (who argue that all talk of belief is invalid) this view is widely held, despite the fact that the authors hold differing views about other aspects of belief structure or formation. For example, Davidson (1973, 1984), an interpretationist, has been an influential proponent of the view that beliefs can only be understood by relating them to a background of other beliefs and desires; Fodor (1978) draws the same conclusions from his 'language of thought' hypothesis – that beliefs must be necessarily related to and justified by reference to other propositions.

Even Price (1934), an atomist, argues that belief formation must rest on conclusions drawn from pre-existing beliefs. In social psychology, theories of belief networks are central to many theories on the psychology of attitudes (Eagly and Chaiken, 1993). Cognitive dissonance theory, as first proposed by Festinger (1957), has the discrepancy between active beliefs as eliciting a drive to make them coherent, and even many of the re-interpretations are essentially belief coherence models (Harman-Jones and Mills, 2002). Similarly, cognitive balance theory, another foundational model in social psychology, has attitudes and beliefs as necessarily existing in relation to other attitudes (Abelson, 1986; Heider, 1946). Rokeach

(1968) goes as far as making explicit distinctions between highly interconnected 'central beliefs' and poorly connected (and therefore relatively inert) 'peripheral beliefs'.

Indeed, even with the rarity of neuropsychological models of belief formation, this integrative process is seen as crucial. Stone and Young (1997) have strongly argued that belief formation may involve weighing up explanations that are observationally adequate versus those that fit within a person's current belief set. 'Observationally adequate' could be interpreted here in a number of ways, including both experiences of the external world and experiences arising from a person's affective state and emotional reaction to an event, in line with the previously mentioned cognitive dissonance and cognitive balance theories.

#### **1.5.4 Role of affect in belief formation**

The idea that there is an important role for emotion in belief formation is a view which has not been without some longstanding support. David Hume famously considered belief to be 'inert' without emotion and much recent empirical and philosophical work has argued convincingly for an important role for emotions in belief formation and belief based reasoning (Frijda et al., 2000; Evans and Cruse, 2004). Social psychological research increasingly defines 'attitudes' in terms of both cognitive and affective components and propensities of individuals to preferentially favour these components in forming attitudes (Haddock and Zanna, 2000).

What is not clear, is how affect and a structural account of belief might interact. Clore and Gasper (2000) suggested that emotion may have an attention-directing influence, but outline this in terms of their, somewhat limited, model of belief formation (section 1.5.1) Spicer (2004) has attempted to bridge this gap and outlined three potential ways in which emotion



and belief might interact: The ‘hybrid view’ is that emotions *are* beliefs and desires, the umbrella view is that emotions behave like beliefs and desires (i.e. they have a propositional content and intentionality) and the nomological view is that emotions carry beliefs and desires with them (i.e. they correlate – anger is generally accompanied by a belief that someone is to blame). The evidence for which of these ways emotion and belief combine is still unclear, however, despite the abundant evidence for their influence on each other.

### **1.5.5 Confidence and authority in belief formation**

Furthermore, beliefs exist with differing degrees of confidence in their likelihood or validity. A belief formation model should not simply ‘output’ a belief as having been accepted or rejected, but allow for a degree of conviction in the belief statement. Although there is reason to think that ultimately this may not break down into a simple single dimension of belief (Harman, 1988, for example, has convincingly argued that in some cases people may be justified in having a higher degree of confidence in a proposition that they do not believe than in a proposition that they do believe), a tacit acknowledgement that beliefs are not ‘all-or-nothing’ entities would seem to be essential to give any successful model of belief face validity.

Finally, beliefs may also be formed on the basis of testimony rather than direct experience, a type of belief Rokeach (1968) called “authority beliefs”. An acceptable theory of belief must include the ability to hold these sort of ‘second hand’ beliefs, without direct perceptual experience of the subject of the belief.

### **1.5.6 What should a cognitive model of belief formation include? : Conclusions**

Belief formation is a complex process and, as has been noted in the philosophy of mind literature, likely to be supported by a number of processes. Both conscious and unconscious processes are likely to be important, as is the ‘web of belief’ and the acceptance of beliefs with differing degrees of confidence and on the basis of authority.

One aspect which seems particularly important is the role affect in the belief formation process. There is now considerable convergent evidence for this, both from studies of normal belief (Frijda et al., 2000) from evolutionary theorist (Evans and Cruse, 2004) and from studies of pathological belief (sections 3.3; 3.4.3). As will be outlined in the subsequent section, this is an aspect which has only recently been included in neuropsychological models.

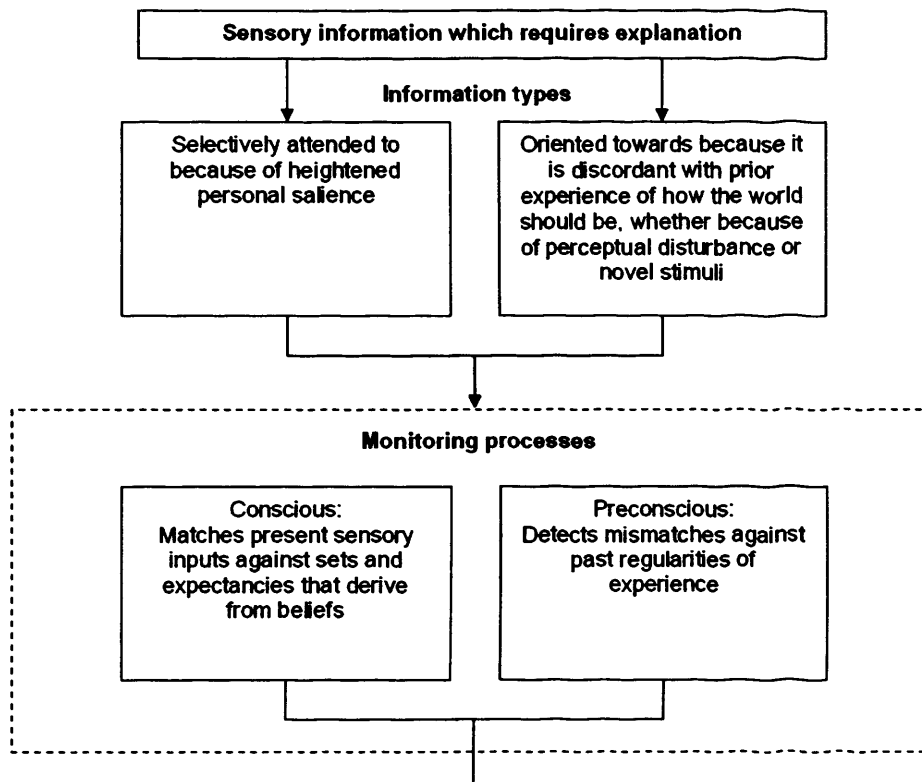
### **1.6 Candidate neuropsychological models of belief formation**

While models of belief formation have been specified before in psychology (e.g. Abelson, 1986; Heider, 1946; Smith et al., 1991), these have typically been created on a common-sense view of belief, without any regard to the neuropsychological mechanisms which might support the process, or whether they are coherent in terms of their relation to supporting neurological structures. There is currently a paucity of detailed neuropsychological models of belief formation, although each is outlined below.

#### **1.6.1 Langdon and Coltheart’s neuropsychological model**

Possibly the most explicitly specified account, derived from a cognitive neuropsychiatric analysis of delusions, is the model from Langdon and Coltheart (2000). As can be seen in figure 1.3, their model takes a three stage approach: the first stage consists of monitoring processes which alerts an individual to information in the environment, which may be novel

or personally relevant. The second stage concerns the generation of hypotheses to explain any information that might be made salient by the earlier monitoring stage; with the final stage involving the evaluation of all possible explanations, a process by which the most rational (or most likely) explanation is accepted as a belief. The monitoring stage acts as a selection system to decide which sensory information is worthy of further consideration.



*figure 1.3 - Langdon and Coltheart's (2000) neuropsychological model of belief formation.*

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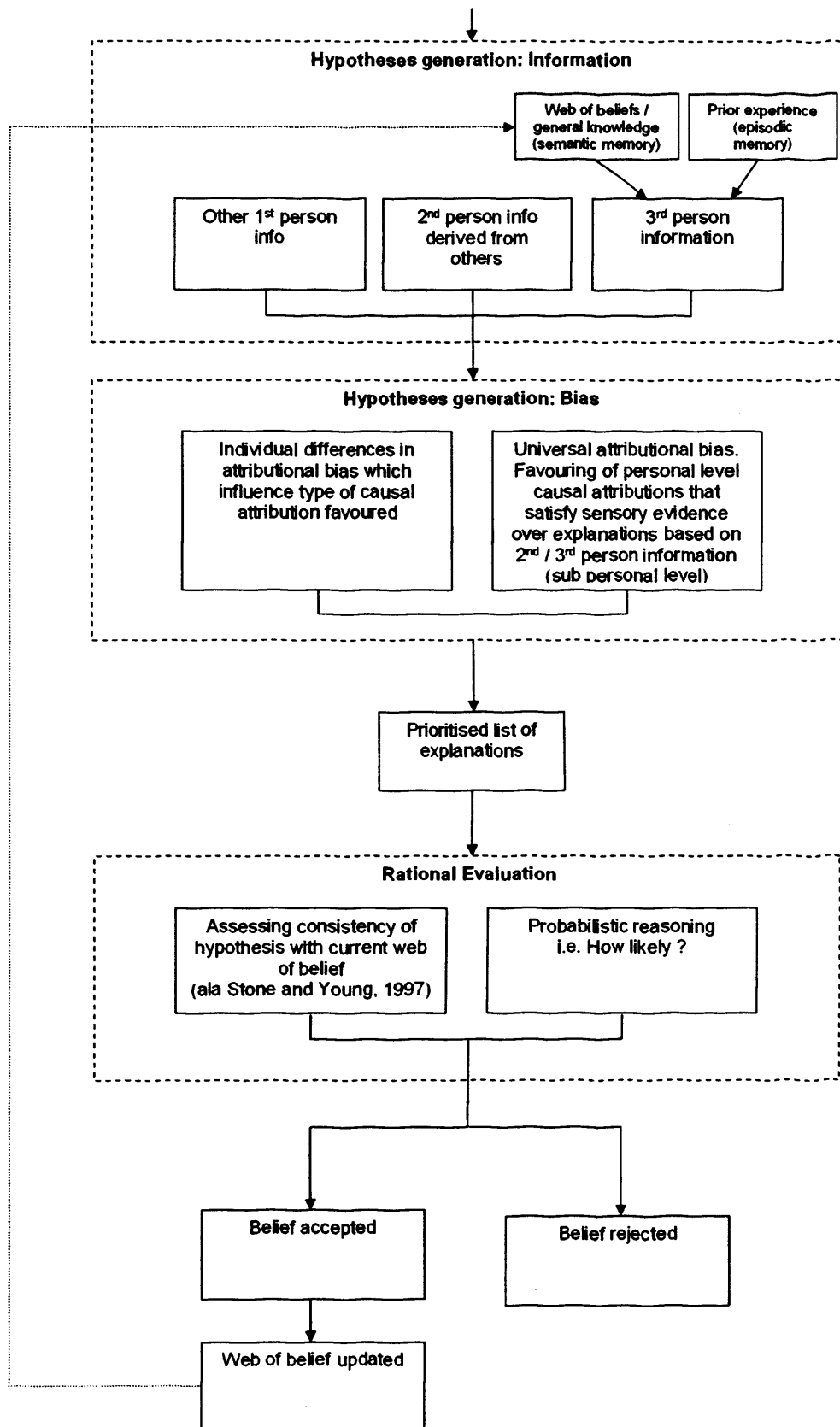


figure 1.3 - Langdon and Coltheart's (2000) neuropsychological model of belief formation  
Diagram created for this thesis.

Langdon and Coltheart are particular about specifying both a conscious and an unconscious component to the monitoring stage, including in their model a conscious process that matches sensory inputs against expectancies that derive from beliefs, as well as an unconscious process that detects mismatches against past regularities of experience. Information that passes the presumed salience threshold might then cause the generation of hypotheses to explain the experience. These hypotheses are drawn from a variety of information sources, including first, second and third person information, as well as episodic memory and a pre-existing 'web of beliefs' (drawing in part from semantic memory). It is at this stage that the hypotheses are weighted so a prioritised 'list' is formed, the weightings being assigned by various sources of bias.

Langdon and Coltheart argue that these may stem from either one of two sources. Their first source is universal influences (although we may be a little suspicious of the use of the term 'universal' here, and most people will probably be happier with the less grand 'sociocultural') such as the favouring of personal over sub-personal level explanations. The other is individual differences in attributional biases, such as those identified by Bentall et al. (1991) in pathological states (such as a tendency to blame negative events on others), or non-pathological biases that may occur in everyday life (Graham and Folkes, 1990; Mezulis et al, 2004). These hypotheses are then subjected to rational evaluation where both probabilistic reasoning and agreement with the current 'web of belief' is used either to accept the belief (and update the web), or to reject it.

## **1.6.2 The Schwitzgebel / Bayne and Pacherie dispositional account**

In contrast to other theories of belief formation outlined in section 1.6, that are either explicitly or implicitly based on a representationalist view of belief (i.e. belief is represented in the brain as circumscribe propositional content; sections 1.3.1; 1.3.6), Bayne and Pacherie (2005) argue for a liberal dispositional model of belief, largely based on the theories of Schwitzgebel (2002; section 1.3.5). Here, beliefs are seen as dispositions to respond in a certain way, and / or experience internal mental events or states (including conscious experiences and affective states).

Bayne and Pacherie assert two central claims: the first is that rationality constraints are thought to be much less of an influence on belief formation (in particular contrast to interpretationist models of belief; section 1.3.4; 1.3.6), in line with psychological research on ‘cognitive distortions’ in decision making and judgment (Gilovich, 1993), the second is that, in accordance with the dispositional approach, beliefs are context dependent and arise as a function of several factors:

(1) the way the long-term memory of the individual is structured, something that depends in turn both on the cognitive organization of the species and on the personal history of the individual, (2) the current external context, and (3) the current motivational and affective set of the individual.

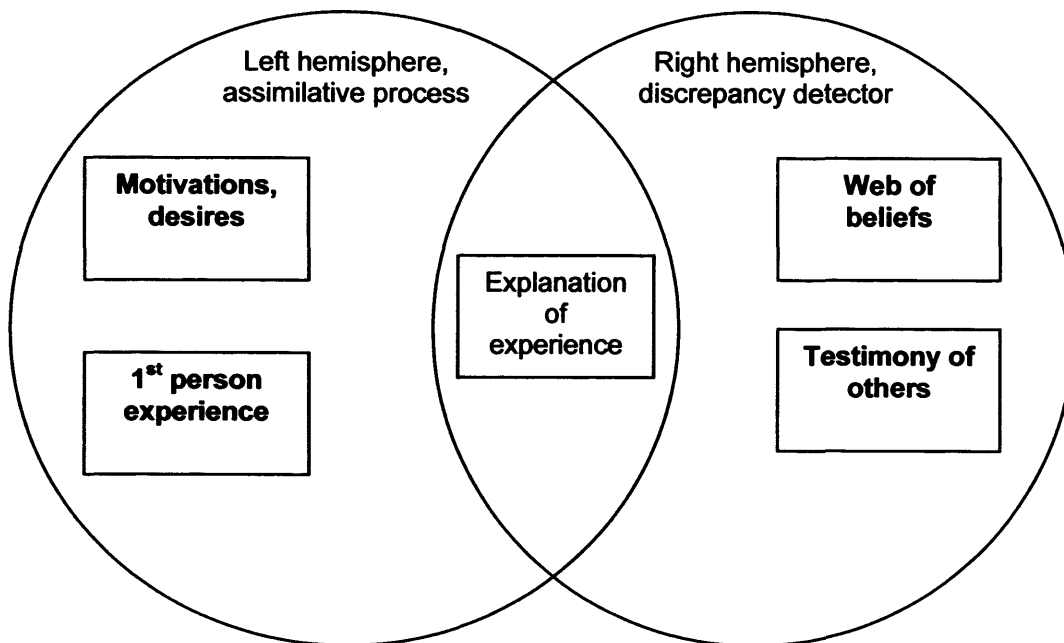
Furthermore, following Schwitzgebel, Bayne and Pacherie argue that “we have dispositional stereotypes for beliefs, specific clusters of behavioral, cognitive, and phenomenal dispositions we associate with given beliefs and expect to be manifested in standard situations.” In other words, belief schemas, which we use both to form and to ascribe beliefs to others.

In contrast to Langdon and Coltheart's (2000; section 1.6.1) 'cold' model of belief formation, this model includes the influence of traditionally 'psychodynamic' factors such as emotion, motivation and past experience of belief formation and ascription. The authors further note that their model meshes well with the understanding of belief used in cognitive behavioural therapy, which relies heavily on the idea that dispositions can be changed through tackling maladaptive schemas and pre-existing assumptions (Hawton, 1989).

Bayne and Pacherie's (2005) model is far from being a well-specified neuropsychological account. For example, it is light on details of how these processes might be implemented. It does have the advantage of being based on a dispositional model of belief, however, which, as noted in section 1.3.5, does not rely on a single monolithic neuropsychological account and is compatible with the idea that belief could be realised by multiple neuropsychological processes.

### **1.6.3 The McKay et al. (2005) hemispheric asymmetry model**

The McKay et al. (2005; figure 1.4) model will be fully explored in section 3.2.3.2, as it is best considered in light of hemispheric asymmetry accounts of delusion formation (section 3.6). The model is intended as a further development of the Langdon and Coltheart account (2001; section 1.6.1) and is ostensibly a model of normal belief derived from breakdowns in normal function after pathology (although, brain injury is the major pathological consideration).



*figure 1.4 McKay et al. (2005) modified two-factor model.  
Diagram created for this thesis.*

It puts at its core the ideas of Ramachandran (1994a; 1994b; 1995; Ramachandran and Blakeslee, 1998), who attempts to explain anosognosia (the unawareness of disability or impairment, usually occurring after brain injury) in terms of the function of the two cerebral hemispheres. The left hemisphere is thought to be involved in the ‘defensive’ or ‘assimilative’ process of filtering or selecting aspects of information to make it best fit into the pre-existing web of beliefs, whereas, in contrast, the right hemisphere is a ‘discrepancy detector’ and re-organises beliefs in response to new information. In this model, a belief is thought to arise from best fitting these competing constraints, through a process not unlike that described by Stone and Young (1997; section 1.5.3).



It is, at first sight, somewhat simpler than Langdon and Coltheart's model and does not attempt to fully integrate into the detailed cognitive framework proposed by them (figure 1.3). The main contribution, however, is to include 'psychodynamic' factors in an explicitly neuropsychological model, which, until recently, have been largely overlooked in previous neuropsychological accounts.

#### **1.6.4 Are these models adequate ?**

At first sight, Langdon and Coltheart's model is the most detailed in terms of specifying the cognitive mechanisms and providing testable hypotheses. Some crucial processes, particularly the use of the 'web of belief' to assimilate and test new beliefs, are left as theoretical black boxes. As discussed earlier, this may be an essential part of any model of belief; potentially both in terms of making any pre-theoretical concept of belief coherent (as the holists would argue) and / or to capture the empirical data concerning the interconnectedness of beliefs and the influence of other beliefs on belief formation.

Similarly, perhaps we have to be a little generous in assuming that emotional effects on belief are adequately explained by the 'attributional biases' process of this model. Whilst affective factors almost certainly alter attribution, relying purely on the cognitive neuropsychiatric approach unduly dispenses with much good work done on normal belief in this area (see Frijda et al., 2000). In the Langdon and Coltheart model, however, beliefs are seen as binary entities to which a person may either assent or dissent. There is no room for varying degrees of conviction or differing degrees of representation, something upon which non-neuropsychological models (particularly the 'attitudinal' models of social psychology; Eagly and Chaiken, 1993) largely focus.

One further problem with this model is its implicit use of a representational account of belief, despite significant conceptual problems with how this would be supported in the neural systems of the brain (section 1.3.5). In light of this, Bayne and Pacherie's (2005) model, although not as well specified, suggests general themes that are more likely to be compatible with both current theories of normal and pathological belief. The fact that their model explicitly includes the influence of emotion and other contextual factors, and attempts to explain belief formation and belief ascription in a single model, makes it a useful starting point. Notably, the model of McKay et al. (2005) takes a similar tack by including psychodynamic factors, although as will be discussed later (section 3.2.3.2), the hemispheric asymmetry account is not well supported by the evidence.

#### **1.6.5 Candidate neuropsychological models of belief formation: Conclusions**

One important conclusion from this comparison is that a cognitive neuropsychiatric approach to belief may lead to useful developments and testable theories. As the only explicitly neuropsychological model of belief of its type, Langdon and Coltheart's (2000) model is well specified, despite the fact that it relies on some assumptions (namely, a representational account of belief) which make it difficult to reconcile with the proposed neural basis. The most compatible with the conceptual considerations and evidence from both normal and pathological belief, is the model of Bayne and Pacherie (2005), although, in itself, it is not a detailed neuropsychological model.

As a more general point, these current models further suggest that the study of delusions, belief pathology and even simply 'anomalous' belief is likely to be a useful and productive approach to understanding normal belief.

## 1.7 Chapter summary and conclusion

As can be seen from the philosophical literature, the everyday concept of belief is used in a wide and varying set of situations; to communicate explicit propositions and implicit behaviour, as well as pre-existing memories and immediately generated conclusions. It is unlikely, therefore, that the everyday concept of belief can be explained by a monolithic neuropsychological model. It is consequently more likely, that belief will break down into a number of disparate neuropsychological processes.

It is also likely, that this framework for belief can be successfully investigated with current methods in cognitive neuropsychiatry. In particular, a cognitive neuropsychiatric approach is appropriate for understanding potentially pathological belief while informing a model of normal belief formation, although parallel investigations into neuropsychological correlates of held-beliefs are likely to provide converging evidence and will be a useful adjunct.

Unfortunately, there are few current neuropsychological models of belief in which to ground empirical research. The most compatible with a neuropsychological approach, and the one that fulfils the most criteria laid out in section 1.5, is the dispositional model of belief described by Bayne and Pacherie (2005; 1.6.2). This, and indeed other models with potential for development, are still embryonic, however, and should be considered as suggesting avenues for further research rather than being definitive models.

## 1.8 References

Abelson, R.P. (1986) Beliefs and like possessions. *Journal for the Theory of Behaviour*, 16, 223-250.

Armstrong, D.M. (1973) *Belief, truth, and knowledge*. Cambridge: Cambridge University Press.

Baker, L.R. (1987) *Saving Belief: A Critique of Physicalism*. Princeton: Princeton University Press.

Bayne, T., Pacherie, E. (2005) In Defence of the Doxastic Conception of Delusions. *Mind and Language*, 20, 163-188.

Bell, V., Ellis, H., Halligan, P. (2003) Neuropsychology, delusions and modularity: The curious problem of belief. *Proceedings of the British Psychological Society*, 11 (2), 174

Bell, V., Halligan, P.W., Ellis, H. (2003) Beliefs about delusions. *The Psychologist*, 16 (8), 418-423.

Bentall, R. P. (1990) The syndromes and symptoms of psychosis. Or why you can't play 'twenty questions' with the concept of schizophrenia and hope to win. In R.P. Bentall P.D.

Slade (eds) (1990) *Reconstructing Schizophrenia*. London: Routledge.

Bentall, R. (2003) *Madness explained: Psychosis and Human Nature*. London: Penguin Books Ltd

Bentall, R. P., Kaney, S., Dewey, M.E. (1991) Paranoia and social reasoning: An attribution theory analysis. *British Journal of Clinical Psychology*, 31, 12-23.

Bloom P, German TP. (2000) Two reasons to abandon the false belief task as a test of theory of mind. *Cognition*, 77 (1), 25-31.

Brugger, P., Gamma, A., Muri, R., Schafer, M., Taylor, K. I. (1993a). Functional hemispheric asymmetry and belief in ESP: towards a "neuropsychology of belief". *Perceptual and Motor Skills*, 77 (3 Pt 2), 1299-1308.

Brugger, P., Regard, M., Landis, T., Cook, N., Krebs, D., Niederberger, J. (1993b). 'Meaningful' patterns in visual noise: effects of lateral stimulation and the observer's belief in ESP. *Psychopathology*, 26 (5-6), 261-265.

Burgess, P. W. (1997). Theory and Methodology in Executive Function Research. In P. Rabbitt (Ed.), *Methodology of Frontal and Executive Function* (pp. 81-116). Hove: Psychology Press.

Caplan, D. (1988) On the role of group studies in neuropsychological and pathophysical research. *Cognitive Neuropsychology*, 5, 535-548.

Caramazza A. (1986) On drawing inferences about the structure of normal cognitive systems from the analysis of patterns of impaired performance: the case for single-patient studies. *Brain and Cognition*, 5, 41-66.

Churchland, P. (1981) Eliminative materialism and the propositional attitudes. *Journal of Philosophy*, 78, 67-90.

Churchland, P. (1999) Current eliminativism. In W. Lycan (ed) *Mind and Cognition 2<sup>nd</sup> edition*. Oxford: Blackwell publishers.

Clore, G.L., Gasper, K.(2000) Some affective influences on beliefs. In N.H. Frijda, A.S.R. Manstead, S. Bem (eds) *Emotions and Beliefs: How Feelings Influence Thoughts*. Cambridge: Cambridge University Press.

Cook, C. M., Persinger, M.A. (1997) Experimental induction of the “sensed presence” in normal subjects and an exceptional subject. *Perceptual and Motor Skills*, 85 (2), 683-93.

Crow, T. J. (1997). Schizophrenia as failure of hemispheric dominance for language. *Trends in Neurosciences*, 20 (8), 339-343.

David, A.S. (1999) On the impossibility of defining delusions. *Philosophy, Psychiatry, and Psychology*, 6 (1), 17-20.

David, A.S., Halligan, P.W. (1996). Editorial. *Cognitive Neuropsychiatry*, 1 (1), 1-3.

Davidson, D. (1973) Radical interpretation. *Dialectica*, 27, 313-328.

Davidson, D. (1984) *Inquiries into truth and interpretation*. Oxford: Clarendon.

Dennett, D. (1978) Beliefs about beliefs. *Behavioral and Brain Sciences*, 1, 568-570.

Dennett, D. (1999) An instrumentalist theory. True believers: The intentional strategy and why it works. In W. Lycan (ed) *Mind and Cognition 2<sup>nd</sup> edition*. Oxford: Blackwell publishers.

Dretske, F. (1988) *Explaining behavior: Reasons in a world of causes*. London: MIT Press.

Eagly, A., Chaiken, S. (1993) *The Psychology of Attitudes*. Orlando: Harcourt Brace and Company.

Ellis, A.W. (1987) Intimations of Modularity, or the Modularity of Mind: Doing Cognitive Neuropsychology Without Syndromes. In Coltheart, M., Sartori, G., Remo, J. (eds) *The Cognitive Neuropsychology of Language*. London: Lawrence Erlbaum.

Ellis, H. D. (1998). Cognitive neuropsychiatry and delusional misidentification syndromes: An exemplary vindication of the new discipline. *Cognitive Neuropsychiatry*, 3(2), 81-90.

Ellis, H.D., Lewis, M.B. (2001). Capgras delusion: a window on face recognition. *Trends in Cognitive Sciences*, 5(4), 149-156.

Ellis, H.D., Young, A.W. (1990). Accounting for delusional misidentifications. *British Journal of Psychiatry*, 157, 239-248.

Engel, P. (2002). Free Believers. In Pessoa, J. Leclerc, A. da Silva de Queiroz, G. Wrigley, M.B. (eds) *Manuscrito, Vol. XXV-Special Number-2002, Mental Causation. Proceedings of the third International Colloquium in Philosophy of Mind*, pp 155-175.

Evans, D., Cruse, P. (2004) *Emotion, Evolution and Rationality*. Oxford: Oxford University Press.

Evans, J. S., Handley, S. J., Harper, C. N. (2001). Necessity, possibility and belief: a study of syllogistic reasoning. *Quarterly Journal of Experimental Psychology A*, 54 (3), 935–958.

Festinger, L. (1957) *A Theory of Cognitive Dissonance*. Stanford: Stanford University Press.

Fodor, J. (1975) *The Language of Thought*. New York: Crowell.

Fodor, J. (1978) Propositional attitudes. *The Monist*, LXI, 4, 501-523.

Fodor, J. (1981) The mind-body problem. *Scientific American*, 244, 114-123.

Fodor, J. (1983) *The Modularity of Mind*. Cambridge Massachusetts: MIT Press.

Fodor, J. (2000) *The Mind Doesn't Work That Way: The Scope and Limits of Computational Biology*. MIT: MIT Press.

Frijda, N. H., Manstead, A. S. R., Bem, S. (2000) *Emotions and beliefs: How feelings influence thoughts*. Cambridge: Cambridge University Press.



Gallagher, H.L., Jack, A.I., Roepstroff, A., Frith, C.D. (2002) Imaging the intentional stance in a competitive game. *Neuroimage*, 16 (3), 814-21.

Gilovich, T. (1993) *How we know what isn't so: The fallibility of human reason in everyday life*. New York: Free Press.

Goel, V., Dolan, R. J., (2003). Explaining modulation of reasoning by belief. *Cognition*, 87, B11-B22.

Graham, S., Folkes, V.S. (1990) *Attributions to achievement, mental health and interpersonal conflict*. Hillsdale, NJ: Lawrence Erlbaum Associates.

Granqvist, P., Fredrikson, M., Unge, P., Hagenfeldt, A., Valind, S., Larhammar, D., Larsson, M. (2005) Sensed presence and mystical experiences are predicted by suggestibility, not by the application of transcranial weak complex magnetic fields. *Neuroscience Letters*, 379, 1-6.

Haddock, G., Zanna, M.P. (2000) Cognition, affect, and the prediction of social attitudes. In W Stroebe, M Hewstone (eds) *European Review of Social Psychology*. Chichester, England: Wiley.

Halligan, P.W., David, A.S. (2001). Cognitive neuropsychiatry: Towards a scientific psychopathology. *Nature Neuroscience Review*, 2, 209-215.

Harman, G. (1988) *Change in View: Principles of Reasoning*. London: Bradford Books.

Harman-Jones, E., Mills, J. (2002) An introduction to cognitive dissonance theory and an overview of current perspectives on the theory. In E. Harman-Jones J. Mills (eds) *Cognitive Dissonance: Progress on a Pivotal Theory in Cognitive Psychology*. Washington: American Psychological Association.

Harper, D.J. (2004) Delusions and Discourse: Moving Beyond the Constraints of the Modernist Paradigm. *Philosophy, Psychiatry and Psychology*, 11 (1): 55-64.

Hawton, K. (1989) *Cognitive Behaviour Therapy for Psychiatric Problems: A Practical Guide*. Oxford: Oxford University Press.

Heider, F. (1946) Attitudes and Cognitive Organization. *Journal of Psychology*, 21, 107-112.

Hirstein, W., Ramachandran, V.S. (1997) Capgras syndrome: a novel probe for understanding the neural representation of the identity and familiarity of persons. *Proceedings of the Royal Society of London B: Biological Sciences*, 264 (1380), 437-44.

Horgan, T., Woodward, J. (1985) Folk psychology is here to stay. *Philosophical Review*, 94 (2), 197-226.

Johns, L.C., van Os, J. (2001) The continuity of psychotic experiences in the general population. *Clinical Psychology Review*, 21, 1125-41.

Kosslyn, SM (1999). If neuroimaging is the answer, what is the question? *Philosophical*

*Transactions of the Royal Society of London, Series B*, 354, 1283-1294

Langdon, R., Coltheart, M. (2000) The cognitive neuropsychology of delusions. In M. Coltheart and M. Davies (eds) *Pathologies of Belief*. Oxford: Blackwell Publishing.

Leonhard, D., Brugger, P. (1998). Creative, paranormal, and delusional thought: a consequence of right hemisphere semantic activation? *Neuropsychiatry, Neuropsychology and Behavioral Neurology*, 11 (4), 177-183.

Lloyd, D. (2000) Terra Cognita: From functional neuroimaging to the map of the mind. *Brain and Mind*, 1, 1-24.

Lloyd, D. (2002) Functional MRI and the study of human consciousness. *Journal of Cognitive Neuroscience*, 14, 818-831.

Makarec, K., Persinger, M.A. (1985) Temporal lobe signs: electroencephalographic validity and enhanced scores in special populations. *Perceptual and Motor Skills*, 60 (3), 831-42.

Makarec, K., Persinger, M.A. (1990) Electroencephalographic validation of a temporal lobe signs inventory in a normal population. *Journal of Research in Personality*, 24 (3), 323-337.

Marcus, R. B. (1990) Some revisionary proposals about belief and believing. *Philosophy and Phenomenological Research*, 50, 132-153.

McKay, R., Langdon, R., Coltheart, M. (2005) "Sleights of mind": Delusions, defences and self-deception. *Cognitive Neuropsychiatry*, 10, 205-326.

Mezulis, A. H., Abramson, L. Y., Hyde, J. S., Hankin, B. L. (2004). Is there a universal positivity bias in attributions?: A meta-analytic review of individual, developmental, and cultural differences in the self-serving attributional bias. *Psychological Bulletin*, 130 (5), 711–747.

Miceli G, Silveri MC, Romani C, Caramazza A. (1989) Variation in the pattern of omissions and substitutions of grammatical morphemes in the spontaneous speech of so-called agrammatic patients. *Brain and Language*, 36 (3), 447-92.

Mohr, C., Rohrenbach, C. M., Laska, M., Brugger, P. (2001). Unilateral olfactory perception and magical ideation. *Schizophrenia Research*, 47 (2-3), 255-264.

Persinger, M. A. (1983). Religious and mystical experiences as artifacts of temporal lobe function: a general hypothesis. *Perceptual and Motor Skills*, 57 (3 Pt 2), 1255-1262.

Persinger, M. A. (1984) Striking EEG profiles from single episodes of glossolalia and transcendental meditation. *Perceptual and Motor Skills*, 58 (1), 127-33.

Persinger, M. A., Fisher, S.D. (1990) Elevated, specific temporal lobe signs in a population engaged in psychic studies. *Perceptual and Motor Skills*, 71 (3), 817-8.

Persinger, M.A., Healey, F. (2002) Experimental facilitation of the sensed presence: possible intercalation between the hemispheres induced by complex magnetic fields. *Journal of Nervous and Mental Disease*, 190 (8), 533-41.

Persinger, M. A., Koren, S. A. (2005) A response to Granqvist et al. "Sensed presence and mystical experiences are predicted by suggestibility, not by the application of transcranial weak magnetic fields". *Neuroscience Letters*, 380, 346-347; author reply 348-350.

Persinger, M.A., Makarec, K. (1990) Exotic beliefs may be substitutes for religious beliefs. *Perceptual and Motor Skills*, 71, 16-18.

Persinger, M.A., Valliant, P.M. (1985) Temporal lobe signs and reports of subjective paranormal experiences in a normal population: a replication. *Perceptual and Motor Skills*, 60 (3), 903-9.

Peters, E. (2001) Are delusions on a continuum ? The case of religious and delusional beliefs. In I. Clarke (ed) *Psychosis and Spirituality: Exploring the New Frontier*. London: Whurr Publishers Ltd.

Peters, E., Day, S., McKenna, J., Orbach, G. (1999) Delusional ideation in religious and psychotic populations. *British Journal of Clinical Psychology*, 38, 83-96.

Pizzagalli, D., Lehmann, D., Gianotti, L., Koenig, T., Tanaka, H., Wackermann, J., Brugger, P. (2000) Brain electric correlates of strong belief in paranormal phenomena: intracerebral EEG source and regional Omega complexity analyses. *Psychiatry Research*, 100, 139-154.

Price, H. H. (1934) Some considerations about belief. *Proceedings of the Aristotelian Society*, 35, 229-52.

Price, H. H. (1969) *Belief*. London: Allen Unwin.

Quine, W.V., Ullian, J.S. (1970) *The Web of Belief*. New York: Random House.

Ramachandran, V. S. (1994a) Phantom limbs, neglect syndromes, repressed memories, and Freudian psychology. *International Review of Neurobiology*, 37, 291-333.

Ramachandran, V. S. (1994b) Phantom limbs, somatoparaphrenic delusions, neglect syndromes, repressed memories and Freudian psychology. In O. Sporns G. Tononi (Eds.), *Neuronal group selection*. San Diego: Academic Press.

Ramachandran, V. S. (1995) Anosognosia in parietal lobe syndrome. *Consciousness Cognition: an International Journal*, 4(1), 22-51.

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

Rokeach, M. (1968) *Beliefs, Attitudes and Values: A Theory of Organization and Change*. San Francisco: Jossey-Bass Inc.

Russell, B. (1921) *The analysis of mind*. New York: McMillan.

Ryle, G. (1949) *The concept of mind*. New York: Barnes Noble.

Schwitzgebel, E. (2002) A phenomenal, dispositional account of belief. *Nous*, 36, 249-275.

Shallice, T. (1988) *From Neuropsychology to Mental Structure*. Cambridge: Cambridge University Press.

Smith, G. F., Benson, P. G., Curley, S. P. (1991) Belief, knowledge, and uncertainty: A cognitive perspective on subjective probability. *Organizational Behavior and Human Decision Processes*, 48, 291-321.

Spicer, F. (2004) Emotional behaviour and the scope of belief-desire explanation. In D. Evans and P. Cruse (eds) *Emotion, Evolution and Rationality*. Oxford: Oxford University Press.

Stich, S. (1983) *From Folk Psychology to Cognitive Science: The Case Against Belief*. Cambridge Massachusetts: MIT Press.

Still, A, Costall, A. (eds) (1991) *Against Cognitivism : Alternative Foundations for Cognitive Psychology*. London: Harvester Wheatsheaf.

Stone, T., Young, A. W. (1997) Delusions and brain injury: The philosophy and psychology of belief. *Mind and Language*, 12, 327-364.

Taylor, K. I., Zach, P., Brugger, P. (2002) Why is magical ideation related to leftward deviation on an implicit line bisection task? *Cortex*, 38, 247-252.

Wittgenstein, L. (1975) *On certainty*. Translated by Denis Paul and G.E.M. Anscombe. Oxford: Blackwell.



## Chapter Two

### Delusions: A Conceptual Review

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Bell, V. (2002) Book review: Pathologies of Belief by M. Coltheart & M. Davies. *Cognitive Neuropsychiatry*, 7 (4), 329-333.

## **2.1 Chapter outline**

Sections:

- 2.2 Diagnostic definition
- 2.3 Reliability of the diagnostic concept
- 2.4 Validity of the diagnostic criteria
- 2.5 The philosophical foundations of delusions – are delusions beliefs?
- 2.6 Chapter conclusion and summary
- 2.7 References

This chapter reviews the current literature on delusions, examining issues of both definition and conceptual coherence. The first part examines the traditional diagnostic approach to delusions and assesses whether the existing clinical definition is reliable and valid. The second part discusses whether delusions are best understood as pathological beliefs, by reviewing theoretical approaches from the philosophical literature and considering whether these are consistent with a neuropsychological approach to belief, as outlined in Chapter 1.

In particular, the chapter will highlight the following issues:

- i) Although the clinical concept, as used, appears reliable for diagnosing delusions in general, shortcomings in the validity of the diagnostic criteria and underlying assumptions suggest further work is needed to refine the conceptual basis of delusions.
- ii) It is unlikely that all delusions take the form of beliefs as traditionally understood. Therefore, neuropsychology should be agnostic as to whether delusions are beliefs (as in the ‘continuum model of psychosis’) or accept that a monolithic model of belief will only partially capture delusional phenomena.

## 2.2 Diagnostic definition

Traditionally, reviews of the delusion literature start by outlining two points of departure.

First, that delusions are the *sine qua non* (essential element) of madness; and second, that the Diagnostic and Statistical Manual of Mental Disorders 4<sup>th</sup> edition (DSM-IV) diagnostic criteria used for characterising delusions are the following:

A false belief based on incorrect inference about external reality that is firmly sustained despite what almost everybody 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). (APA, 1994)

The APA position is based on criteria and assumptions originally articulated by Jaspers (1923), which included the criterion of certainty (a belief held with absolute conviction), incorrigibility (a belief not changeable by compelling counterargument or proof to the contrary) and impossibility or falsity of content (an implausible, bizarre or patently untrue belief). As noted by Berrios (1991), however, Jaspers' work was actually derived from earlier, nineteenth century views of delusions, the main elements of which were:

- a) delusions are symptoms (i.e. indications of underlying disease);
- b) psychopathological symptoms can be analysed both in terms of 'form' and 'content', and the diagnostic criteria for delusions could be derived from this analysis;
- c) delusions are beliefs (ideas or mental contents), and hence are amenable to reality-testing;

- d) there are sources and mechanisms for delusions, that include a person's character, past history, circumstances during development of the delusion, and brain state;
- e) the clinical significance of delusions can be determined by using analysis based on form, content and mechanism. For example, different types of delusions could be indicative of different types of illness.

Although, as will be discussed later, all of these elements have now been challenged, the views expressed above can be considered as the working model generally accepted by contemporary mainstream psychiatry (Berrios, 1991).

Clinically, the detection of delusions has “enormous implications for diagnosis and treatment, as well as complex notions concerning responsibility, prediction of behaviour, etc.” (David, 1999). Yet, as pointed out by many commentators, the usage of the term ‘delusion’ and its distinction from other normal and abnormal beliefs involve a host of practical, semantic and epistemological difficulties (Jones, 1999; Leeser and O’Donohue, 1999; Spitzer, 1990).

The following sections will examine the concept of delusion both in terms of its reliability as a pragmatic construct, and as a valid clinical concept based on a coherent conceptual foundation.

## 2.3 Reliability of the diagnostic concept

### 2.3.1 Diagnosis of non-bizarre delusions

The practical use of any clinical concept relies on the reliability with which diagnosis can be made. There are various techniques available for diagnosing or rating a belief as delusional.

The reliability of such methods have been shown to vary both within methods, as well as when methods are compared with each other. Table 2.1 summarises studies that have examined the inter-rater reliability of the general concept of delusions.

	<i>Inter-rater reliability</i>	
	<i>kappa</i>	<i>R</i>
<i>Structured interview</i>		
World Health Organisation (1979)		
Inter-centre		0.83 – 0.98
Intra-centre		0.61
Andreasen et al. (1982)	0.88	
Endicott et al. (1982)		
NHSI <sup>1</sup> delusions	0.81	
Carpenter et al. <sup>2</sup> widespread delusions	0.28	
DSM-III <sup>3</sup> non-persecutory / jealous delusions	0.59	
RDC <sup>4</sup> non-persecutory / jealous delusions	0.77	
Helmes et al. (1983) nihilistic delusions	0.156	
<i>Vignette assessment</i>		
Junginger et al. (1992)	0.46 – 0.78	
<i>SAPS<sup>5</sup></i>		
Moscarelli et al. (1987)	0.88	
Norman et al. (1996)	0.86	
<i>CASH<sup>6</sup></i>		
Andreasen et al. (1992)	0.64	
<i>PANNS<sup>7</sup></i>		
Bell et al. (1992)	0.93	
Norman et al. (1996)	0.88	
Muller and Wetzel (1998)	0.42 – 0.85	
<i>Mean</i>	0.69	0.76
<i>SD</i>	0.24	0.21

<sup>1</sup>New Haven Schizophrenia Index (Astrachan et al., 1972). <sup>2</sup>Carpenter et al. criteria, part of the WHO international Pilot Study of Schizophrenia (Carpenter et al., 1973). <sup>3</sup>Diagnostic and Statistical Manual of Mental Disorders III (APA, 1980). <sup>4</sup>Research Diagnostic Criteria (Spitzer et al., 1978). <sup>5</sup>Scale for the Assessment of Positive Symptoms (Andreasen, 1984). <sup>6</sup>Comprehensive Assessment of Symptoms and History (Andreasen et al., 1992) <sup>7</sup>Positive and Negative Syndrome Scale (Kay et al., 1987).

table 2.1 Studies of inter-rater reliability for diagnosis or detection of non-bizarre delusion

The studies summarised in table 2.1 suggest that the diagnosis of delusions can be made with an acceptable level of inter-rater reliability (falling in the ‘substantial agreement’ range of 0.61 – 0.80; Landis and Koch, 1977). There are some important caveats, however, particularly with regard to using a clinical interview for diagnosis. Harper (1999) has noted that the World Health Organisation (1979) study used a limited statistical method, had a small sample size and did not rule out the possibility of clinicians discussing their findings before deciding on a diagnosis; whereas the Andreasen et al. (1982) study has a particularly high level of agreement, although the ratings were based on videotaped interviews after raters were given a “careful and systematic training program”, suggesting that the results are unlikely to generalise to typically more haphazard clinical situations.

The detection or diagnosis of delusions by the use of standardised scales seems to produce the most reliable inter-rater results; however, diagnosis by clinical interview, the standard diagnostic procedure, also seems to produce an acceptable level of reliability.

Research studies are now increasingly favouring the use of such scales owing to their greater reliability, although alternative and principled approaches have been used in empirical studies. Garety and Hemsley (1994), for example, eschewed standardised scales to assess the presence of delusions and simply relied on psychiatric diagnosis based on clinical interview, as they were interested in examining the concept of delusions used by clinicians rather than researchers.

### 2.3.2 Diagnosis of bizarre delusions

Although no criteria is provided in the DSM-IV for the defining the bizarreness of delusions, previous editions have considered a bizarre delusion to be “patently absurd with no possible basis in fact” (DSM-III; APA, 1980) or that it “involves a phenomenon that the person’s culture would regard as totally implausible” (DSM-III-R; APA, 1987).

Bizarre delusions, although notoriously difficult to define and quantify, have particular diagnostic significance, both for the positive diagnosis of DSM schizophrenia, and also as exclusion criteria for DSM classifications such as delusional disorder. Table 2.2 summarises reliability studies for the diagnosis of bizarre delusions, using structured interview, vignette assessment and psychometric scales.

	Inter-rater reliability	
	<i>kappa</i>	<i>r</i>
<i>Structured interview</i>		
Endicott et al. (1982)		
Carpenter et al. criteria	0.66	
DSM-III	0.29	
Kendler et al. (1983)	0.27 – 0.30	
Flaum et al. (1991)	0.28 – 0.31	
Goldman et al. (1992)	0.78	
Garety and Hemsley (1994)	0.31	
Nakaya et al. (2002) <sup>1</sup>	0.85 – 0.92	
<i>Vignette assessment</i>		
Junginger et al. (1992)	0.45	
Spitzer et al. (1993)	0.64 – 0.65	
Mojtabai and Nicholson (1995)	0.38 – 0.43	
<i>Psychometric scale</i>		
Eisen et al. (1998)		0.02
<i>Mean</i>	0.50	0.02
<i>SD</i>	0.22	0

<sup>1</sup>Also used Positive and Negative Syndrome Scale (Kay et al., 1987)

table 2.2 Studies of inter-rater reliability of bizarre delusion diagnosis or detection

Although the mean reliability lies within the moderate agreement range (0.41 – 0.6; Landis and Koch, 1977), the studies reporting the most substantial levels of agreement are subject to significant methodological flaws: diagnoses in the Goldman et al. (1992) study were based on “consensus judgment” and involved raters who, according to Spitzer et al. (1993) “participated in weekly discussions of ratings of psychiatric symptoms, including bizarre delusions, for a year and a half before the study”. In turn, the methodology of Spitzer et al. has been criticised, as the raters were either the authors or the authors’ associates, which, as pointed out by Mojtabai and Nicholson (1995), is a serious methodological flaw as “it is not surprising that a homogenous group of raters would attain a higher level of agreement”. To address this issue Mojtabai and Nicholson used a random sample of psychiatrists and obtained far lower levels of inter-rater reliability. Similarly, the high level of diagnostic reliability reported by Nakaya et al. (2002) can be accounted for by the fact that the diagnosis of bizarre delusions was based on the use of both the Positive and Negative Syndrome Scale (Kay et al., 1987) and a review of case notes. Furthermore, the raters practised joint interviews before the study began, and made joint ratings at regular points throughout the testing period.

When these problematic studies are removed from the reliability calculations reported in table 2.2, the mean kappa drops to 0.37 (SD = 0.15). With these drawbacks in mind, only one method (the Carpenter et al. criteria from the study by Endicott et al., 1982) reported acceptable reliability, and it is by no means clear that this study used either a random sample of psychiatrists or prevented pre-rating discussions as criticised previously. These weaknesses suggest, on the basis of diagnostic reliability, that a reliable concept of bizarre delusion has yet to be achieved.



### **2.3.3 Conclusions on reliability**

Despite unacceptable reliability in the diagnosis of bizarre delusions, it seems that the presence of delusions in general, can be diagnosed reliably, both with clinical interview and with standardised scales.

### **2.4 Validity of the diagnostic criteria**

Assuming for the moment that delusions are beliefs (although this view has also been seriously challenged and will be subsequently tackled in section 2.5) the APA diagnostic criteria for a delusion can be broken down into four main criteria, three of which are taken directly from Jaspers' (1923) criteria, plus an additional criterion to allow for cultural variation:

- i) certainty – the belief is held with absolute conviction,
- ii) incorrigibility – the belief is not changeable by compelling counterargument or proof to the contrary,
- iii) impossibility or falsity of content – an implausible, bizarre or patently untrue belief derived from an incorrect inference about external reality,
- iv) culturally atypical – the belief is not one ordinarily accepted by other members of the person's culture or subculture.

Furthermore, as discussed earlier, a delusion may be considered to be bizarre, with some definitions specifically making reference to criterion *iv*, that of cultural atypicality (see section 2.4.3 below).

The application of these diagnostic criteria also relies on a phenomenological distinction between form and content, first introduced into mainstream psychiatry by Jaspers (1923), and still forming an essential part of diagnostic practice (Sims, 2003).

The validity of these criteria is a matter of ongoing debate, largely because it has become clear that the criteria are, in themselves, incoherent, or subject to important and damaging counterexamples.

#### **2.4.1 Certainty and incorrigibility**

Studies on psychiatric patients have shown that delusions vary in intensity and conviction over time, which suggests that certainty and incorrigibility are not necessary components of a delusional belief.

Myin-Germeys et al. (2001) used an experience sampling method and reported that the intensity of delusional conviction can vary over short periods of time (even during a single day). These results have been supported by psychometric measurements of conviction in delusional patients (Garety and Hemsley, 1994; Freeman and Garety, 2004). A large-scale study of 1,136 hospitalised psychiatric patients by Appelbaum et al. (2004) similarly showed that the stability of delusions is highly variable, with the chance of an initially identified delusion still being intact during regular follow-up interviews typically being less than 45%.

Studies which have directly asked about the possibility of being mistaken about a delusional belief have typically found that about 50% of patients will accept the possibility (Garety et al., 1997; 2005). Clinical trials and case studies examining the effect of cognitive behavioural therapy on delusions (e.g. Chadwick et al., 1996; Kuipers et al., 1997; section 4.4.4.4) have

also suggested that incorrigibility is not a necessary feature of delusions, as certainty and conviction can be changed through collaborative therapy. The success of this form of therapy (and hence the corrigibility of delusions) can be seen from a study by Garety et al. (1997) where 50% of people with delusions were judged to have shown a response to therapy.

Furthermore, observers of the scientific community have noted that scientists can hold fixed, incorrigible beliefs about external reality, despite overwhelming evidence to the contrary without being considered clinically delusional (Fort, 1919; Kuhn, 1962). Recently, similar processes have been found in the general population, where individuals report believing in news reports despite being aware of them being subsequently discounted (Lewandowsky et al., 2005). These examples suggest that incorrigibility does not necessarily distinguish between 'normal' and pathological beliefs.

#### **2.4.2 Impossibility or falsity of content**

Simply described, "bizarre delusions are generally impossible, whereas non-bizarre delusions are generally improbable" (Sedler, 1995, p.256). The DSM-IV distinguishes these as follows: a non-bizarre delusion may involve situations that in principle could occur in real life but are thought (by the psychiatrist) to be highly improbable and therefore potentially falsifiable; a bizarre or fantastic belief, however, is considered impossible, and, therefore, is assumed to be one not normally held by others in the culture or society.

The problem with each of these definitions lies not with the differential distinction, but with the absence of agreed operational definitions as to how these criteria are employed clinically. The DSM definition does not specify how an individual psychiatrist might set about establishing the falseness or bizarreness of the belief.

Non-bizarre delusions involve situations and events that could occur in real life, such as believing that one is being followed, infected, poisoned or deceived by another. Therefore, the 'falsifiability' criterion means that psychiatrists are required to make judgements on claims of marital infidelity, persecution or conspiracy in the workplace, where the available relevant evidence is either limited, cannot be ascertained within the confines of the consulting room, or lies beyond the forensic capabilities of the clinician (Jones, 1999). As pointed out by Young (2000, p47), "many of the beliefs considered to be delusions do not meet these criteria (or are not tested against them) in practice".

Accordingly, this falsity criterion has been rightly questioned (Spitzer, 1990). Moreover, it is unclear what level of evidence would be required to consider a belief 'incontrovertibly false' and whether judgements should be based on the 'balance of probabilities' or the more stringent test of 'beyond reasonable doubt'.

In fact, many diagnosed delusions may turn out to be true beliefs in one of two ways: they may be true beliefs incorrectly diagnosed as delusions, or, paradoxically, they may turn out to be true beliefs while still being considered delusional.

The former situation has been reported where a real-life basis for a patient's paranoia was discovered (Mayerhoff et al., 1992). This has been called the 'Martha Mitchell effect' by Maher (1988), after the wife of the US Attorney General who believed there was a conspiracy in the White House and was diagnosed as mentally ill, only to be proved correct (and hence sane) when the Watergate scandal broke.

The latter situation, where delusions turn out to be largely true while still being considered delusions, have also been reported. Cases of delusional jealousy, where the accused partner has genuinely been unfaithful – but not on the grounds that the deluded person claimed – are not uncommon (Sims, 2003). Slightly more curious was a case of delusional hypochondriasis reported by Fulford (1989), where the patient arrived at an Accident and Emergency centre claiming he was mentally ill. As this belief was his only psychiatric symptom, this created a unusual logical paradox for the falsity criterion.

Furthermore, as Fulford (1994) has noted, some beliefs are inherently unfalsifiable; as they may either be value judgements (e.g. “I am a fantastically talented poet”) or not amenable to practical falsification (e.g. “Satan is listening to my thoughts”). In these cases, the falsity criterion is simply inapplicable, despite the fact that grandiose and religious delusions (undoubtedly comprising many such unfalsifiable examples) are considered to be among the most prevalent themes.

### **2.4.3 Cultural atypicality and bizarreness**

The attribution that a delusion is culturally atypical is usually defined in terms of beliefs considered not normally held by other members of a person’s culture or society. This, however, first involves the psychiatrist’s own evaluation as regards the plausibility of the belief, after which it is considered whether the belief is typically shared by others in the community.

The DSM definition clearly assumes that the criterion of abnormality or bizarreness should be obvious, given that the belief is one not ordinarily accepted by other members of a person’s culture or subculture. This is not a reliable strategy: as has been shown earlier, many studies

of psychiatrists show poor inter-rater reliability for the diagnosis of bizarre beliefs. Moreover, most clinicians are not in a position to know or find out whether such beliefs comprise those which are widely accepted.

One method of comparison is the use of large-scale surveys, but most clinical judgements on the prevalence of beliefs in society are not typically informed by empirical evidence. In fact, beliefs in unscientific or parapsychological phenomena are not uncommon (Della Salla, 1999); and were this criterion alone employed as a sufficient condition, many people would at times be classified as delusional (Moor and Tucker, 1979). Large-scale research polls carried out in the UK and North America consistently reveal that significant numbers of people within these societies hold strong (largely unfalsifiable) beliefs about the paranormal. For example, Cox and Cowling (1989) reported that 50% of people believed in thought transference and 25% of people believed in ghosts as spirits of the dead; whereas a recent American survey found that over 50% of people believed in demonic possession and more than 30% believed in visitation from extra-terrestrials (Rice, 2003).

The existence of high levels of conviction in what might be considered abnormal, unscientific or paranormal beliefs raises important questions when justifying the notion of bizarre beliefs on purely conceptual or statistical grounds. As pointed out by French (1992), most beliefs are based upon “personal experiences perhaps supported by reports of trusted others, and the general cultural acceptance that such phenomena are indeed genuine”.

It is also not clear exactly what constitutes ‘cultural’ or ‘sub-cultural’ and how many people need to hold a belief before it is considered normal. One curious concept in this regard is *folie à deux* (Enoch and Ball, 2001), or its more populous cousins *folie à trois*, *folie à quatre* or

even *folie à famille*, where a delusional belief is transmitted from one person (typically known as the ‘primary’, ‘inducer’ or ‘principal’) to another (the ‘secondary’, ‘acceptor’ or ‘associate’). Presumably, at some point, a belief considered to be delusional escapes from the *folie à...* diagnostic category and becomes exempt because of the number of people holding it.

Related work by sociologist Robert Bartholomew (2001) has shown that huge numbers of people can accept false, seemingly bizarre and even impossible beliefs without them fulfilling any of the standard diagnostic criteria for a delusion, particularly as these beliefs tend to resolve after a relatively short period of time, usually when related social tensions have been resolved. Although these beliefs (for example, *koro*, the belief that the genitals are shrinking into the body or have been stolen) are often classified under the rubric of a ‘culture-bound syndrome’, it is clear that the content of such beliefs are not necessarily culturally specific. Neither it seems, is the process of rapid social acceptance of false or unusual beliefs, which has been recorded many times in the West in addition to the cultures that are usually given as exemplars in discussion of ‘culture bound’ pathologies (Bartholomew, 2000).

#### **2.4.4 The form / content distinction in diagnosis**

Following Jaspers (1923), psychopathology has been typically diagnosed on the form (the structural properties) of a mental phenomenon, rather than its content (Sims, 2003). For example, the fact that a perception does not correspond to an external stimulus is considered more diagnostically significant than whether the perception is of a male or female voice. Spence (2001) has argued, based on the Jasperian dichotomy, that delusions arise from the same “underlying biological signal” and that influences of culture are only likely to affect the “surface content”. As has been noted above many of the aspects of form that are thought to

distinguish pathological beliefs from non-pathological ones, do not seem to achieve this task adequately.

There is increasing evidence, however, that the form / content dichotomy may be an artificial distinction and that social and cultural experiences have a significant influence in the development of delusions beyond simply “surface content”. Rhodes and Jakes (2004) reported that almost half the patients interviewed related their current delusional beliefs to metaphors used to understand recent experiences. Furthermore, they proposed that continuing use of these metaphors could maintain the psychotic experience. An earlier study by the same researchers (Rhodes and Jakes, 2000) noted that delusions often relate to life goals and personal concerns. A study by Birchwood et al. (2000) suggested that patients’ beliefs about the power and authority of hallucinated voices (and, interestingly, even their perception of properties such as frequency and volume) were linked to experiences of social subordination in their daily lives. Other research has reported that traumatic experiences during childhood can influence both the form and content of psychotic experiences (review in Read et al., 2004).

This evidence indicates that cultural and social experience can significantly influence clinical factors, such as form, aetiology and prognosis, despite the traditional view that they only affect the content of delusions.

#### **2.4.5 Conclusions on validity**

In light of the conceptual difficulties with the validity of the diagnostic criteria, David (1999) has gone as far as to suggest that “there is no acceptable (rather than accepted) definition of a delusion”. Indeed, it seems that although psychiatrists may be able to reliably decide on



whether a delusion is present, there are difficulties with the current definition as important counter-examples can be found for many of the criteria, suggesting delusions are not a unitary phenomenon (not a result of a unitary “underlying biological signal” as suggested by Spence, 2001), and hence more likely to resemble a syndrome rather than a symptom (Gilleen and David, 2005).

The limits of the current definition still need to be tested and explored to ensure that the diagnostic criteria are relevant to the current social and cultural environment. Cultural and social aspects seem to have been relatively neglected in terms of their direct influence on diagnostic validity, making this a priority for a coherent understanding of the practical application of the delusion concept. As well as providing a significant criticism of the diagnostic criteria, studies showing that cultural factors play a substantial role in multiple aspects of the clinical category may suggest that delusions are not a unitary biological symptom, but a multi-faceted, syndrome-like phenomenon.

It is important to note, however, that a cognitive neuropsychiatric approach is not necessarily hampered by the fact that delusions may not be a unitary symptom. As discussed in section 1.4.2, cognitive neuropsychology has shown that impairments originally thought of as unitary symptoms are now better understood in terms of symptom clusters and deficits in cognitive sub-systems. This suggests that delusions are valid as elements of enquiry, as long as they are not inappropriately reified as indivisible units of cognitive pathology.

One surprising omission in the psychiatric definition of delusions, however, is any criterion that defines pathology in terms of the impact on a person’s life. A person who fulfils the current diagnostic criteria could, in principle, be no trouble to themselves or others, and

presumably never need social or medical assistance. Indeed, this is highlighted by the fact that many beliefs considered normal can fulfil all the diagnostic criteria (section 2.4). This is likely due to a sample bias in psychiatry, where definitions are based on observations of clinical cases. Those clinical cases are, by and large, only going to include people who find themselves distressed or causing social concern.

Work by Peters et al. (1999a) has suggested that members of new religious movements may have equal levels of delusional ideation to psychotic inpatients, but are markedly less distressed. Similarly, Romme and Escher (1993) have challenged the view that auditory hallucinations are pathognomic of mental illness, by reporting on large numbers of voice hearers who find their experiences neither distressing nor socially problematic.

It is clear that further work is needed on the experiences of people who may prove to be counter-examples to many of the assumptions of mainstream clinical psychiatry, and to current theories of delusion formation that associate anomalous perceptual experience with pathology (to be outlined in Chapter 3). This issue is further addressed in an empirical study reported in Chapter 7.

## **2.5 The philosophical foundations of delusions – are delusions beliefs?**

The preceding sections have considered the reliability of diagnosis and the validity of the explicitly stated diagnostic criteria, concluding that although some types of delusions can be diagnosed with acceptable reliability in the clinic, the diagnostic criteria are, at best, subject to significant counter examples and at worst, incoherent – particularly when applied as a catch-all definition.

Following the analysis presented in Chapter 1, one aspect of the diagnostic criteria – that delusions are beliefs – may seem particularly contentious. This assumption is rarely questioned in the empirical literature, with most studies assuming that delusions are beliefs with no regard to the coherence or implications of this theoretical stance. Nevertheless, the status of delusions as beliefs has been questioned in the philosophical literature, both in terms of its folk psychological basis and the notion of scientific realism and objectivity on which it depends. Crucially, such discussions provide a context in which to place the empirical research which will be reviewed in Chapter 3.

The following sections, therefore, will tackle some of the main theoretical approaches to delusions, particularly with regard to the theories of belief as outlined in Chapter 1, as a prelude to discussing the empirical and experimental work in this area.

### **2.5.1 Delusions as ‘speech acts’: The Non-assertoric approaches.**

Berrios (1991) famously described delusions as “empty speech acts”, considering them “not the symbolic expression of anything”. This broad approach has been alternatively christened the ‘non-assertoric’ (Young, 1999) or ‘expressivist’ (Gerrans, 2001) view and has gained a number of supporters.

Berrios bases his conclusion on Price’s (1934) account of belief; i.e. that a proposition can only be considered a belief if it has involved the consideration of alternative propositions and the believer knows that the relevant facts make the accepted belief more likely than the alternative propositions. Since, Berrios argues, delusions do not seem to involve this reality-testing process, they cannot be considered beliefs and are simply linguistic entities. Sass (1994) argued for a similarly non-assertoric view, on the basis that people rarely act on their

delusions and do not seem to have the emotional or affective response to their beliefs that might be expected, if, for example, they genuinely believed their spouse was trying to kill them, or that they had God-like powers.

Neither of these factors consistently distinguish normal from pathological beliefs, however, as can be demonstrated with reference to the literature on normal belief. Maio (2002) has conclusively shown that people can typically produce very few reasons for beliefs in important social values (such as freedom, equality and so on) and can therefore hold such beliefs without fulfilling Price's criteria. The social psychological literature has also shown that people often do not act on their beliefs. Indeed, Eagly and Chaiken (1993, p155) note that "positive correlations of no more than moderate magnitude are probably the best expectation that most social scientists could hold about the relation between attitudes and behaviour".

This also belies that fact that many people do indeed act on their delusions. A recent review on forensic aspects of the Capgras delusion noted that acts of violence were "frequently" reported (Bourget and Whitehurst, 2004), many of them directed towards the misidentified person. A study by Wessely et al. (1993) found that half of the delusional patients surveyed had acted on their delusions, with persecutory themes most likely to predict action. A more recent study on the less obvious behaviour of people with persecutory delusions found that 100% of the sample reported using 'safety behaviours' developed to 'protect' the believer from the threat or persecution in direct response to their beliefs (Freeman et al., 2004).

While these studies suggest (against the non-assertoric view) that delusions are not simply 'empty speech acts', other authors have argued that although delusional claims may not be 'empty', they may result from radical changes in the expression of meaning in language. So,

when a delusional person claims that (for example) “I have magical healing powers”, they are not expressing the same belief as others perceive them to be, because the semantics of their belief-claim have been pathologically altered.

This was a view originally expounded by Jaspers (1923) and has recently been taken up by Campbell (2001). Campbell argues that, although the language of a person with delusions seems to express a belief (e.g. “I am dead”), that belief claim does not express the same content as an identical claim if it were made by a non-deluded person, by virtue of the fact that a deluded person has impaired perceptual and reasoning systems and therefore has different semantic referents when making such claims.

Campbell bases his thesis on the work of interpretationist philosophers such as Davidson (1973; 1984) and Quine (Quine and Ullian, 1970; section 1.3.6) who argued that beliefs can only be ascribed to people with reference to their supporting beliefs and who are assumed to be, at least in part, rational. According to these accounts, there should be a link between knowledge of a term’s meaning, and the use of that term or concept in reasoning. Radically irrational claims, therefore, suggest to Campbell that, for an otherwise rational person, the claim in question must hold a radically different meaning for the speaker.

At first glance, it may seem unlikely that people with delusions use common terms in a fundamentally different way to the general population. However, the difficulty with which internal mental events can be successfully labelled and discussed with others has been highlighted by Wittgenstein’s (1953) ‘private language argument’. Wittgenstein argued that agreement on the labelling of external objects is possible because the errors of individuals can be corrected by (perhaps literally) pointing out the referent. Since internal mental events and

sensations are inaccessible to others, there is no independent criterion for correctness. If this process is thought problematic for the normal range of mental phenomena, the unusual beliefs and experiences of psychosis might be even more difficult to describe adequately by individuals who have only their previous referents upon which to rely.

The non-assertoric position argues, therefore, that although delusions are frequently communicated as belief-claims, they are not beliefs in any recognisable sense. There is a middle position, however, in that some belief-claims expressed by patients may not express beliefs and might more closely resemble metaphor and simile, while others might be more traditionally belief-like (Halligan et al., 1995). It has even been suggested that this belief-resemblance might lie on a continuum, so that some delusional belief-claims are more likely to be genuinely belief-like than others (Bayne and Pacherie, 2004), although no clear criteria have been offered for judging how belief-like a claim might be.

With regards to finding possible neuropsychological underpinnings, the general non-assertoric view suggests that delusions do not reflect pathologies of belief per se, but are better understood as inadequately communicated pathologies of experience and perception. This position is not affected by objections or limitations on a neuropsychological approach to belief (as outlined in Chapter 1), by virtue of the fact that it denies that delusions are beliefs as we would normally understand them. Therefore, a neuropsychology of delusion is compatible with the non-assertoric approach, although it would likely reduce the phenomena to pathologies of perception, semantics, language or communication, rather than a putative neuropsychology of belief.

The major drawback of the most specified non-assertoric accounts (e.g. Campbell, 2001), however, is that they lack empirical support, particularly with regard to their reliance on the assumption of strict rationality as a prerequisite for belief formation and ascription, as it is now widely accepted that strict rationality is not a common feature of human reasoning (Gilovich, 1993).

### **2.5.2 Delusions as negotiated pathology: Social constructionist approaches.**

Because of difficulties with both adequately describing internal mental events and the incoherence of the accepted diagnostic criteria for delusions (see section 2.4) some researchers have criticised what they see as the “naïvely realist view” (Harper, 2004) inherent in the traditional understanding of delusions, and instead argue for delusions as a diagnostic decision that is constructed during doctor-patient discourse, based on a socio-politically influenced interpretation of abnormality.

Georgaca (2000; 2004) has noted that the diagnostic process does not happen ‘in a vacuum’, but instead, it is a discursive processes embedded within a complex system. For example, the person making the diagnosis is a mental health professional located in a system of influence (e.g. mental health legislation, social hierarchy) with the power to infer belief claims from the patient’s discourse. These claims are further transformed into value-laden psychopathological signs and symptoms. Rather than locating the pathology of rationality in the patient, Georgaca argues that the problem lies within a breakdown of understanding between patient and professional.

Discourse analyses of how mental health professionals talk about paranoia supports this position. Harper (1994) found that professionals use flexible criteria when defining paranoia,

allowing them to 'work around' challenges and contradictions in the diagnostic process by using strategies that serve to support their professional and institutional legitimacy. Analysis of discourse between doctors and psychotic patients by McCabe et al. (2002), showed that doctors rarely test the diagnostic criteria for delusions, instead deflecting talk of topics assumed, *a priori*, to be delusional. Further work by Harper (1999) also showed that delusional patients' discourse attempts to satisfy multiple goals: to explain behaviour regarded as problematic so as not to appear wilful or responsible for the negative consequences; to appear a 'good' and motivated patient; and to portray themselves as rational and in control, contrary to the stereotypes of madness.

From a social constructionist perspective, therefore, a delusion is the result of a situated decision that an interpreted belief claim is pathological which emerges from the attempts of various parties to satisfy competing, and sometimes mutually exclusive goals. From this standpoint, although the diagnostic concept specifically and directly refers to a pathology of belief, the label may be (and perhaps, often is) deployed by medical professionals in a pragmatic or arbitrary way, suggesting a dichotomy in how delusion is defined and how the concept is used in clinical practice.

It is important to note, however, that this position does not necessarily deny that delusions are beliefs, just that a diagnosis of delusion may not necessarily denote a belief or involve an objective definition (or even exploration) of pathology. It is also important to note that this position is compatible with the reliability data presented in section 2.3, as socially or politically influenced diagnoses could still be made reliably, as was demonstrated by the use of the diagnostic category "sluggishly progressing schizophrenia" to silence dissidents in the former Soviet Union (Lavretsky, 1998).



Nevertheless, with reference to attempts to derive neuropsychological explanations for delusion, a social constructionist approach would not necessarily deny that certain forms of pathological belief might be explained by a neuropsychological model. However, such an approach would question whether the clinically-deployed diagnostic categories denote an unbiased objective phenomenon, and whether such categories should be the basis of a valid enquiry. From this perspective, any neuropsychological model is likely to fail if it seeks to explain all phenomena that become diagnosed as 'delusions', and therefore may have to accept limitations of scope if it is to be successful.

### **2.5.3 Delusions as a continuum of anomalous mental phenomena: The dimensional view**

In contrast to the traditional understanding of delusions as qualitatively different from normal belief, dimensional approaches consider delusional ideation to exist on a continuum distributed throughout the population, with those diagnosed as delusional being at one of the most extreme ends of the distribution.

Although thought of as a relatively recent idea, the view that delusion-like thinking may exist on a continuum has been put forward many times in the history of psychiatry, with Bleuler (1911) being an early advocate. Perhaps its relative lack of acceptance has been, in part, due to the championing of the idea by people outside mainstream psychiatric thought.

For example, Campbell (1926, p8) offered a strikingly modern view of delusions when he wrote:

The concrete material referred to in the following pages has been chosen to illustrate the role of beliefs in the adaptation of the individual to the stresses of existence, and may serve to show that the delusions of the ill-balanced and the beliefs of the orthodox are more closely akin than is usually recognized. Under special strain the orthodox may lapse from conventional belief into individual delusion, and the delusion of one person, rejected by his contemporaries, may in another group of period become a socially accepted belief.

However, his further classification of the beliefs of non-Western peoples as “primitive” and the beliefs of minority religious groups as “inferior”, perhaps suggest that his virtual omission from the delusion literature is due to embarrassment over his less-than-palatable political opinions.

Taking a similar line on the continuity of sanity and madness, Laing variously argued that delusions are the result of attempting to accommodate conflicting social expectations (Laing, 1960) or a form of cathartic spiritual experience (Laing, 1967). His association with the counter-culture of the time, and his association with the ‘anti-psychiatry movement’ (notably, an association he himself rejected) led to his views being widely rejected by mainstream psychiatry (Clay, 1996).

It was not, perhaps, until psychologists began to advocate and implement quantitative approaches that dimensional views of psychosis became more widely influential. One of the earliest attempts to capture the psychosis-continuum was the inclusion of a psychoticism dimension in Eysenck’s personality scales (Eysenck, 1952; Eysenck and Eysenck, 1976), implying that psychosis could be measured as an aspect of personality and could vary between individuals without necessarily being pathological. Extrapolating from personality-

theory, Eysenck aimed to capture psychosis-proneness on a dimensional construct varying from normality to psychosis. This was subsequently developed into a multidimensional concept of schizotypy (Claridge et al., 1996) based on a factor analysis of various psychosis-proneness scales (Bentall et al., 1989).

Schizotypy and psychoticism, however, are not focused solely (if even mainly) on beliefs, but typically encompass a wide range of idiosyncrasies of thought, experience, preference and perception. Nevertheless, even quantitative dimensional approaches more directly related to delusions, rarely ask about beliefs per se. Although rarely stated explicitly, this ‘continuum model of psychosis’ (Johns and van Os, 2001) is typically agnostic to whether delusions are necessarily beliefs.

For example, despite being frequently cited as an example of how anomalous beliefs are distributed throughout the population (e.g. Johns and van Os, 2001; Lundberg et al., 2004; van Os, 2003), the Peters et al. Delusions Inventory (Peters et al., 1999b; Peters et al., 2005) consists largely of “Do you feel...” rather than “Do you believe...” items, deftly sidestepping the issue of whether delusions are indeed beliefs. Conceptually, it would be quite possible for someone to endorse a large number of items on the PDI without actually assenting to a belief in any of them. Perhaps, to get round this issue, the PDI includes various subscales, one of which allows the respondent to rate conviction in each item’s veracity, although this still remains agnostic as to whether the item content involves a belief (rather than a meta-belief in how veridical the experience is).

In the psychosis continuum framework, delusions are considered non-specific expressions of anomalous beliefs, ideas, memories, feelings, notions or other mental phenomena, and are

therefore compatible with a number of other approaches, including neuropsychology. This is largely by virtue of the fact that attributes of certain experiences and convictions, or the presence of such experiences, can be quantified (largely by self-report measured, avoiding some of the criticisms in social constructionist approaches) and hence correlated with other measures, or used as dependent or independent variables.

#### **2.5.4 Delusions as pathological beliefs: Doxastic views of delusion.**

As mentioned previously, much of the empirical literature (reviewed in Chapter 3) relies on an implicit assumption that delusions are beliefs, usually by attempting to find correlates for whatever is presumed to underlie the diagnosis. This section, however, will examine the literature that provides specific evidence or arguments for delusions being best understood as beliefs.

Surprisingly, considering the amount of literature that has argued against the view that delusions are beliefs, there is little work that explicitly defends this position with reference to an explicit model of normal belief formation. There are, however, plenty of examples where authors assume delusions are beliefs, based, presumably, on little more than *prima facie* evidence.

Theories labelled as ‘empiricist’ approaches to delusion formation are broadly in this camp, and typically argue that delusions stem primarily from pathologies of experience, and that this pathology is a necessary condition for delusion formation (therefore, empiricist approaches include both ‘one-stage’ and ‘two-stage’ theories; section 3.2). For example, Maher (1974; 1988) argues that delusions are the result of pathological experience being interpreted by a normal belief formation process, whereas Ellis and Young (1990) argue that delusions result

from pathological experience being interpreted by an abnormal belief formation process. Both accounts consider delusions to be beliefs *a priori*, an approach best summarised by Davies et al. (2001) who state that “we shall simply assume delusions are beliefs” with no further justification for their position.

Taking a more considered approach, Bentall (1990) was one of the first theorists specifically to attempt an understanding of delusions with reference to a model of normal belief formation. Although, as a general model, it is admittedly lacking in some key areas (see Bell et al., 2006; section 1.5) it has been developed with both clinical and non-clinical belief biases in mind, and is intended to support Bentall’s theories on attribution biases, rather than function as a complete account of belief (Bentall, 2003).

On the basis of the philosophical literature, Bortolotti (2005) similarly defends the position that delusions are beliefs. She argues against Berrios (1991), who contends that Price’s (1934) criteria for belief<sup>1</sup> are not fulfilled by delusions, by suggesting that sensory evidence is indeed factual. She also notes that alternative propositions are often considered by delusional persons (Young, 2000), although they are perhaps rejected, owing to a bias for accepting waking perceptual experiences as veridical (Sackeim, 1998).

She further argues that evidence for socio-cultural influences on the content of delusional beliefs (e.g. Bell et al., 2005; section 4.4) suggests that a conscious process of belief formation is involved. Interestingly, Bortolotti also turns Campbell’s (2001) argument on its head, suggesting that, instead of inferring that a belief-claim has a radically different meaning for the speaker because of its irrationality, the fact that other evidence suggests delusions do

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<sup>1</sup> To recap: a proposition can only be considered a belief if it has involved the consideration of alternative propositions and the believer knows that the relevant facts make the accepted belief more likely than the alternative propositions,

function like beliefs causes us to question the validity of interpretationist theories of belief ascription on which Campbell's own argument is based.

Others have attempted a similar approach, by comparing delusions, overvalued ideas and normal beliefs, on criteria drawn from the empirical and philosophical literature (Jones and Watson, 1997; Jones, 1999). Jones and colleagues conclude that delusions are indeed beliefs, as such comparisons between normal and abnormal belief can be coherently made and empirically tested, something, they argue, that would not be possible if delusions did not have a similar structure to normal belief. A somewhat similar approach was taken by Rhodes and Jakes (2000), who compared delusions to patients' life-goals, and found that delusional themes integrated well into more general concerns and motivations, suggesting they operate similarly to beliefs in this context.

A more abstract approach has been taken by Currie and colleagues (Currie, 2000; Currie and Jureidini, 2001; Currie and Ravenscroft, 2002) who argue that delusions are meta-beliefs (beliefs about other beliefs, sometimes called 'second-order beliefs', in contrast to beliefs about external phenomena, sometimes called 'first-order beliefs'), formed on the basis of misidentified imaginings. According to this account, a breakdown in the ability to separate imagination and belief occurs, where a person imagines a proposition or hypothetical belief, and then, owing to poor metacognition (section 3.5), comes to believe that they believe it (i.e. comes to form a second-order belief about an illusory first-order belief).

Currie's account has been criticised, however, both for a lack of a plausible mechanism by which such a misidentification could occur (Bell, 2002), for inadequately specifying the properties of imaginings and belief, and for having little evidence on which to separate

distinct roles for first- and second-order beliefs in delusional thinking (Bayne and Pacherie, 2005).

Bayne and Pacherie (2005) combine their criticisms of Currie's account with a defence of a belief-based account of delusions. They explicitly deny that all delusions are necessarily beliefs, while aiming to produce a model based on the premise that many delusions will qualify as beliefs. Their model is based on Schwitzgebel's (2002) dispositional account of belief (section 1.3.5) but fleshes out the structure with explicit links to empirically-based psychological theory, such as the likely role of affective modulation and schemas in long-term memory being involved in the mechanisms of belief-based action. They also eschew accounts of belief that emphasize strict rationality constraints in the face of considerable evidence that normal human reasoning is bounded or less-than-optimal in many situations (Gilovich, 1993).

In stark contrast to the Bayne and Pacherie approach is Langdon and Coltheart's (2000) model of belief which aims "to develop a model of the normal cognitive system of belief generation and belief evaluation which can then explain *all* types of delusion" (emphasis in the original; Langdon and Coltheart, 2000, p184). The Langdon and Coltheart model, previously described in section 1.6.1, aims to be explicitly neuropsychological, although it is not clear whether each process can be broken down into independent neuropsychological processes. As discussed in section 1.3 there is no unitary definition for what a folk-psychology belief-claim consists of, or, as discussed earlier in this chapter (sections 2.4; 2.5), what a diagnosis of delusion signifies, suggesting that belief claims may result from partially (or even substantially) different neuropsychological mechanisms. It is unclear, therefore, which parts of the system are necessary or sufficient for the production of belief, and, because

it considers all delusions to be beliefs, whether the model will be successful in explaining all delusions.

Similarly, by taking any of the belief-based approaches to delusion described above, theories hoping to link cognitive-and brain-level explanations are bound by the difficulties and restrictions on making neuropsychological inferences as outlined in Chapter 1. In light of this, the doxastic theories most compatible with a neuropsychological approach are the models of Bayne and Pacherie (2005) and Langdon and Coltheart (2000). A weakness of the Langdon and Coltheart model is that it is likely to be overambitious, in that it considers all delusions to be beliefs, and all beliefs to be unitary phenomena. The Bayne and Pacherie model is more limited in its scope and, therefore, is perhaps more likely to be successful in its own domain. Both, however, allow belief to be explained as a number of interacting subsystems which can produce folk-psychological belief claims.

### **2.5.5 Philosophy section conclusion**

Theories that argue against delusions as beliefs on the basis that the diagnostic process is not an objective method of reifying pathological beliefs (such as the social constructionist approach, section 2.5.2), are not unique to psychosis or even psychiatric illness. The diagnosis of physical illness can be influenced by similar subjective influences (Berg, 1992) and yet successful models of illness and normal function can still be developed from this basis.

Furthermore, as demonstrated in section 2.3, it is possible for trained clinicians to diagnose a delusion with acceptable reliability, although agreement is not perfect and certain classes of delusion may be less reliably classified.



This suggests that much like many higher-level concepts used in the world (e.g. ‘fish’ or ‘flower’) the clinical concept of delusion is used to label a heterogeneous set of phenomena, although, perhaps, with some overlapping core features. There may be various examples, however, that are not easily captured by the orthodox definition, or cannot easily be explained by a single monolithic theory. Perhaps unsurprisingly, this shows striking parallels to theories of normal belief, as outlined in Chapter 1.

Therefore, any successful theory of delusions must either be agnostic as to whether delusions are beliefs, or must explain delusions within a framework that accepts beliefs as heterogeneously represented, or the outcome of heterogeneous neuropsychological processes. In terms of the most useful theoretical frameworks for approaching the neuropsychology of delusion and belief, the ‘continuum model of psychosis’ and the model of Bayne and Pacherie (2005) seem adequate starting points at the current time.

## **2.6 Chapter summary and conclusion**

The diagnostic definition of delusions is subject to some serious flaws and needs refining, particularly in the area of validity. One particular aspect which has seen little in the way of principled empirical testing, is the criterion of socio-cultural atypicality. This implies delusional beliefs cannot form the basis of a cohesive culture or subculture, and should be relatively impervious to social factors, except, perhaps, for trivial or insignificant ‘surface content’ features. These issues will be tackled in Chapter 4.

Because of the lack of conceptual coherence in the diagnostic criteria, a neuropsychological account of delusions will be necessarily based on conclusions drawn from cases where an inexact diagnostic process is used. It is therefore likely that the use of the term ‘delusional

belief', like the use of the term 'belief', does not describe a unitary neuropsychological process. Neuropsychological models must therefore be limited in their scope (not seeking to explain all delusions as beliefs), or accept that phenomena currently labelled as 'delusional' may need to be explained by a number of processes; or they must be agnostic as to whether delusions are beliefs and derive correlates of the general phenomena.

## 2.7 References

American Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorders: DSM-III (3<sup>rd</sup> edn)*. New York: Author.

American Psychiatric Association (1987) *Diagnostic and Statistical Manual of Mental Disorders: DSM-III-R (3<sup>rd</sup> revised edn)*. Washington, DC: Author.

American Psychiatric Association (1994) *Diagnostic and Statistical Manual of Mental Disorders: DSM-IV (4<sup>th</sup> edn)*. Washington, DC: Author.

Andreasen NC (1984) *Scale for the Assessment of Positive Symptoms (SAPS)*. Iowa City: Department of Psychiatry, University of Iowa College of Medicine.

Andreasen NC, Flaum M, Arndt S. (1992) The Comprehensive Assessment of Symptoms and History (CASH). An instrument for assessing diagnosis and psychopathology. *Archives of General Psychiatry*, 49 (8), 615-23.

Andreasen NC, McDonald-Scott P, Grove WM, Keller MB, Shapiro RW, Hirschfeld RM. (1982) Assessment of reliability in multicenter collaborative research with a videotape approach. *American Journal of Psychiatry*, 139 (7), 876-82.

Appelbaum, P.S., Robbins, P.C., Vesselinov, R. (2004) Persistence and stability of delusions over time. *Comprehensive Psychiatry*, 45 (5), 317-24.

Astrachan BM, Harrow M, Adler D, Brauer L, Schwartz A, Schwartz C, Tucker G. (1972) A checklist for the diagnosis of schizophrenia. *British Journal of Psychiatry*, 121(564), 529-39.

Bartholomew, R.E. (2000) *Exotic deviance: Medicalizing cultural idioms from strangeness to illness*. Boulder, Colorado: University Press of Colorado.

Bartholomew, R.E. (2001) *Little green men, meowing nuns and head-hunting panics: A study of mass psychogenic illness and social delusion*. London: McFarland Co.

Bayne, T., Pacherie, E. (2004) *Experience, Belief, and the Interpretive Fold*. *Philosophy, Psychiatry and Psychology*, 11, 81-86.

Bayne, T., Pacherie, E. (2005) In Defence of the Doxastic Conception of Delusions. *Mind and Language*, 20, 163-188.

Bell, V. (2002) Book review: *Pathologies of Belief* by M. Coltheart M. Davies. *Cognitive Neuropsychiatry*, 7 (4), 329-333.

Bell, V., Halligan, P.W., Ellis, H.D. (2006) A Cognitive Neuroscience of Belief. In P.W. Halligan & M. Aylward (eds) *The Power of Belief*. Oxford: Oxford University Press.

Bell, V., Grech, E., Maiden, C., Halligan, P.W., Ellis, H.D. (2005) 'Internet Delusions': A Case Series and Theoretical Integration. *Psychopathology*, 38, 144-150.

Bell M, Milstein R, Beam-Goulet J, Lysaker P, Cicchetti D. (1992) The Positive and Negative Syndrome Scale and the Brief Psychiatric Rating Scale. Reliability, comparability, and predictive validity. *Journal of Nervous and Mental Disease*, 180 (11), 723-8.

Bentall, R. P. (1990) The syndromes and symptoms of psychosis. Or why you can't play 'twenty questions' with the concept of schizophrenia and hope to win. In R.P. Bentall, P.D. Slade (eds) (1990) *Reconstructing Schizophrenia*. London: Routledge.

Bentall, R. P. (2003) *Madness Explained: Psychosis and Human Nature*. London: Penguin Books Ltd.

Bentall RP, Claridge GS, Slade PD (1989) The multidimensional nature of schizotypal traits: a factor analytic study with normal subjects. *British Journal of Clinical Psychology*; 28, 363-375.

Berg, M. (1992) The construction of medical disposals: Medical sociology and medical problem solving in clinical practice. *Sociology of Health and Illness*, 14, 151-180.

Berrios, G. E. (1991) Delusions as "wrong beliefs": A conceptual history. *British Journal of Psychiatry Supplement*, 14, 6-13.

Birchwood M, Meaden A, Trower P, Gilbert P, Plaistow J (2000) The power and omnipotence of voices: subordination and entrapment by voices and significant others. *Psychological Medicine*, 30, 337-44.

Bleuler E. (1911) *Dementia praecox or the group of schizophrenias*. Translated by J. Zinkin. New York: International Universities Press.

Bortolotti, L. (2005) Delusions and the Background of Rationality. *Mind and Language*, 20, 189-208.

Bourget, D., Whitehurst, L. (2004) Capgras syndrome: A review of the neurophysiological correlates and presenting clinical features in cases involving physical violence. *The Canadian Journal of Psychiatry*, 49, 719-725.

Campbell, C.M. (1926) *Delusion and Belief*. Cambridge: Harvard University Press.

Campbell, J. (2001) Rationality, meaning and the analysis of delusion. *Philosophy, Psychiatry and Psychology*, 8, 89-100.

Carpenter WT, Strauss JS, Bartko JJ. (1973) Flexible system for the diagnosis of schizophrenia: report from the WHO International Pilot Study of Schizophrenia. *Science*, 182 (118), 1275-8.

Chadwick, P., Birchwood, M., Trower, P. (1996) *Cognitive therapy for delusions, voices and paranoia*. Chichester, UK: Wiley.

Claridge G, McCreery C, Mason O, Bentall R, Boyle G, Slade P, Popplewell D. (1996) The factor structure of 'schizotypal' traits: a large replication study. *British Journal of Clinical Psychology*, 35, 103-15.

Clay, J. (1996) *R.D. Laing: A Divided Self*. London: Hodder Stoughton.

Cox, D., Cowling, P. (1989) *Are you normal ?* London: Tower Press.

Currie, G. (2000) Imagination, delusion and hallucinations. In M. Coltheart and M. Davies (eds), *Pathologies of Belief*. London: Blackwell.

Currie, G., Jureidini, J. (2001) Delusion, rationality, empathy. *Philosophy, Psychiatry and Psychology*, 8, 159–62.

Currie, G., Ravenscroft, I. (2002) *Recreative Minds*. Oxford: Oxford University Press.

David, A.S. (1999) On the impossibility of defining delusions. *Philosophy, Psychiatry and Psychology*, 6, 17-20.

Davidson, D. (1973) Radical interpretation. *Dialectica*, 27, 313-328.

Davidson, D. (1984) *Inquiries into truth and interpretation*. Oxford: Clarendon.

Davies, M., Coltheart, M., Langdon, R., Breen, N. (2001) Monothematic delusions: Towards a two-factor account. *Philosophy, Psychiatry, and Psychology*, 8, 133-158.

Della Salla, S. (ed) (1999) *Mind myths*. New York: Wiley.

Eagly, A.H., Chaiken, S. (1993) *The Psychology of Attitudes*. Orlando: Harcourt and Brace Ltd.

Eisen, J. L., Phillips, K. A., Baer, L., Beer, D. A., Atala, K. D., Rasmussen, S. A. (1998) The Brown Assessment of Beliefs Scale: reliability and validity. *American Journal of Psychiatry*, 155, 102-108.

Ellis, H. D., Young, A. W. (1990) Accounting for delusional misidentifications. *British Journal of Psychiatry*, 157, 239-248.

Endicott J, Nee J, Fleiss J, Cohen J, Williams JB, Simon R. (1982) Diagnostic criteria for schizophrenia: reliabilities and agreement between systems. *Archives of General Psychiatry*, 39 (8), 884-9.

Enoch, D., Ball, H. (2001) Folie à deux (et Folie à plusieurs). In Enoch, D., Ball, H. *Uncommon psychiatric syndromes (Fourth edition)*. London: Arnold.

Eysenck, HJ. (1952) Schizothymia-cyclothymia as a dimension of personality. *Experimental Journal of Personality*, 20, 345-384.

Eysenck, HJ, Eysenck SBG. (1976) *Psychoticism as a Dimension of Personality*. London: Hodder and Stoughton.

Flaum M, Arndt S, Andreasen NC (1991) The reliability of "bizarre" delusions. *Comprehensive Psychiatry*, 32 (1), 59-65.



Fort, C. (1919) *The Book of the Damned*. Reprinted 1974. Toronto: Dover Publishing Company.

Freeman, D., Garety, P.A. (2004) *Paranoia: The Psychology of Persecutory Delusions*. Hove: Psychology Press.

Freeman, D., Garety, P.A., Fowler, D., Kuipers, E., Bebbington, P.E., Dunn, G. (2004) Why do people with delusions fail to choose more realistic explanations for their experiences? An empirical investigation. *Journal of Consulting and Clinical Psychology*, 72 (4), 671-80.

French, C.C. (1992) Factors underlying belief in the paranormal: Do sheep and goats think differently? *The Psychologist*, 5, 295–299.

Fulford, K.W.M. (1989) *Moral theory and medical practice*. Cambridge: Cambridge University Press.

Fulford, K.W.M. (1994) Value, illness and failure of action: Framework for a philosophical psychopathology of delusions. In G. Graham and G.L. Stephens (eds) *Philosophical Psychopathology*. Cambridge, Massachusetts: MIT Press.

Garety, P., Fowler, D., Kuipers, E., Freeman, D., Dunn, G., Bebbington, P., Hadley, C., Jones, S. (1997) London–East Anglia randomised controlled trial of cognitive-behavioural therapy for psychosis II: Predictors of outcome. *British Journal of Psychiatry*, 171, 420–426.

Garety, P. A., Freeman, D., Jolley, S., Dunn, G., Bebbington, P. E., Fowler, D. G., Kuipers, E., Dudley, R. (2005) Reasoning, emotions, and delusional conviction in psychosis. *Journal of Abnormal Psychology*, 114, 373-384.

Garety, P.A., Hemsley, D.R. (1994) *Delusions: Investigations into the Psychology of Delusional Reasoning*. Oxford: Oxford University Press.

Georgaca, E. (2000) Reality and discourse: a critical analysis of the category of 'delusions'. *British Journal of Medical Psychology*, 73, 227-42.

Georgaca, E. (2004) Factualization and plausibility in delusional discourse. *Philosophy, Psychiatry and Psychology*, 11, 13-23.

Gerrans, P. (2001) Delusions as performance failures. *Cognitive Neuropsychiatry*, 6 (3), 161-173.

Gilleen, J., David, A. S. (2005) The cognitive neuropsychiatry of delusions: from psychopathology to neuropsychology and back again. *Psychological Medicine*, 35, 5-12.

Gilovich, T. (1993) *How we know what isn't so: The fallibility of human reason in everyday life*. New York: Free Press.

Goldman, D., Hien, D.A., Haas, G.L., Sweeney, J.A., Frances, A.J. (1992) Bizarre delusions and DSM-III-R criteria. *American Journal of Psychiatry*, 149, 494-499.

- Halligan PW, Marshall JC, Wade DT. (1995) Unilateral somatoparaphrenia after right hemisphere stroke: a case description. *Cortex*, 31, 173-82.
- Harper, D. J. (1994) The professional construction of 'paranoia' and the discursive use of diagnostic criteria. *British Journal of Medical Psychology*, 67, 131-143.
- Harper, D.J. (1999) *Deconstructing Paranoia: An Analysis of the Discourses Associated with the Concept of Paranoid Delusion*. Doctoral thesis, Manchester Metropolitan University.
- Harper, D.J. (2004) Delusions and Discourse: Moving Beyond the Constraints of the Modernist Paradigm. *Philosophy, Psychiatry and Psychology*, 11, 55-64.
- Helmes E, Landmark J, Kazarian SS. (1983) Inter-rater reliability of twelve diagnostic systems of schizophrenia. *Journal of Nervous and Mental Disease*, 171 (5), 307-11.
- Jaspers, K. (1923) *General Psychopathology (7th edn)*. Translated by J. Hoenig, M. Hamilton. Manchester: Manchester University Press, 1969.
- Johns, L.C., van Os, J. (2001) The continuity of psychotic experiences in the general population. *Clinical Psychology Review*, 21, 1125-41.
- Jones, E. (1999) The phenomenology of abnormal belief: A philosophical and psychiatric inquiry. *Philosophy, Psychiatry and Psychology*, 6, 1-16.

Jones E, Watson JP. (1997) Delusion, the overvalued idea and religious beliefs: a comparative analysis of their characteristics. *British Journal of Psychiatry*, 170, 381-6.

Junginger, J., Barker, S., Coe, D. (1992) Mood theme and bizarreness of delusions in schizophrenia and mood psychosis. *Journal of Abnormal Psychology*, 101, 287–292.

Kay SR, Fiszbein A, Opler LA. (1987) The positive and negative syndrome scale (PANSS) for schizophrenia. *Schizophrenia Bulletin*, 13, 261-276.

Kendler KS, Glazer WM, Morgenstern H. (1983) Dimensions of delusional experience. *American Journal of Psychiatry*, 140 (4), 466-9.

Kuhn, T. (1962) *The Structure of Scientific Revolutions*. University of Chicago Press

Kuipers, E., Garety, P., Fowler, D., Dunn, G., Bebbington, P., Freeman, D., Hadley, C. (1997) London–East Anglia randomised controlled trial of cognitive-behavioural therapy for psychosis: Effects of the treatment phase. *British Journal of Psychiatry*, 171, 319–327.

Laing R.D. (1960) *The divided self: An existential study in sanity and madness*. London: Penguin Books.

Laing, R.D. (1967) *The Politics of Experience and the Bird of Paradise*. London: Penguin Books.

Landis, J., Koch, G. (1977) The measurement of observer agreement for categorical data. *Biometrics*, 33, 159-174.

Langdon, R., Coltheart, M. (2000) The cognitive neuropsychology of delusions. In M. Coltheart and M. Davies (eds) *Pathologies of Belief*. Oxford: Blackwell Publishing.

Lavretsky H. (1998) The Russian concept of schizophrenia: a review of the literature. *Schizophrenia Bulletin*, 24, 537-57.

Leeser, J., O'Donohue, W. (1999) What is a delusion? Epistemological dimensions. *Journal of Abnormal Psychology*, 108 (4), 687-694.

Lewandowsky, S., Stritzke, W.G.K., Oberauer, K., Morales, M. (2005) Memory for Fact, Fiction, and Misinformation: The Iraq War 2003. *Psychological Science*, 16, 190-195.

Lundberg, P., Cantor-Graae, E., Kabakyenga, J., Rukundo, G., Ostergren, P. O. (2004) Prevalence of delusional ideation in a district in southwestern Uganda. *Schizophrenia Research*, 71, 27-34.

Maher, B. (1974) Delusional thinking and perceptual disorder. *Journal of Individual Psychology*, 30, 98-113.

Maher, B. (1988) Anomalous experience and delusional thinking: The logic of explanations. In T.F. Oltmanns and B.A. Maher (eds) *Delusional beliefs*. Chichester: Wiley.



- Maio, G.R. (2002) Values - Truth and meaning. *The Psychologist*, 15, 296-299.
- Mayerhoff D, Pelta E, Valentino C, Chakos M. (1992) Real-life basis for a patient's paranoia. *American Journal of Psychiatry*. 149 (1), 140-1.
- McCabe, R., Heath, C., Burns, T., Priebe, S. (2002) Engagement of patients with psychosis in the consultation: conversation analytic study. *British Medical Journal*, 325, 1148-1151.
- Mojtabai, R., Nicholson, R.A. (1995) Interrater reliability of ratings of delusions and bizarre delusions. *American Journal of Psychiatry*, 152, 1804-1806.
- Moor, J.H., Tucker, G.J. (1979) Delusions: Analysis and criteria. *Comprehensive Psychiatry*, 20, 388-393.
- Moscarelli M, Maffei C, Cesana BM, Boato P, Farma T, Grilli A, Linguardi V, Cazzullo CL. (1987) An international perspective on assessment of negative and positive symptoms in schizophrenia. *American Journal of Psychiatry*, 144 (12), 1595-8.
- Muller MJ, Wetzel H. (1998) Improvement of inter-rater reliability of PANSS items and subscales by a standardized rater training. *Acta Psychiatrica Scandinavica*, 98 (2), 135-9.
- Myin-Germeys, I., Nicolson, N.A., Delespaul, P.A.E.G. (2001) The context of delusional experiences in the daily life of patients with schizophrenia. *Psychological Medicine*, 31, 489-498.

Nakaya, M., Kusumoto, K., Okada, T., Ohmori, K. (2002) Bizarre delusions and DSM-IV schizophrenia. *Psychiatry and Clinical Neurosciences*, 56, 391-395.

Norman, R. M., Malla, A. K., Cortese, L., Diaz, F. (1996) A study of the interrelationship between and comparative interrater reliability of the SAPS, SANS and PANSS. *Schizophrenia Research*, 19, 73-85.

Peters, E., Day, S., McKenna, J., Orbach, G. (1999a) Delusional ideation in religious and psychotic populations. *British Journal of Clinical Psychology*, 38, 83-96.

Peters ER, Joseph S, Day S, Garety P (2005) Measuring delusional ideation: The 21-item Peters et al. delusions inventory (PDI). *Schizophrenia Bulletin*, 30, 1005-1016.

Peters ER, Joseph SA, Garety PA (1999b) Measurement of delusional ideation in the normal population: introducing the PDI (Peters et al. Delusions Inventory). *Schizophrenia Bulletin*, 25, 553-76.

Price, H. H. (1934) Some considerations about belief. *Proceedings of the Aristotelian Society*, 35, 229-52.

Quine, W.V., Ullian, J.S. (1970) *The Web of Belief*. New York: Random House.

Read, J., Goodman, L., Morrison, A.P., Ross, C.A., Aderhold, V. (2004) Childhood trauma, loss and stress. In J. Read, L. Mosher, R.P. Bentall (eds) *Models of Madness*. Hove: Bruner-Routledge.

Rice, T.W. (2003) Believe It or Not: Religious and Other Paranormal Beliefs in the United States. *Journal for the Scientific Study of Religion*, 42 (1), 95-106.

Rhodes, J. E., Jakes, S. (2000) Correspondence between delusions and personal goals: a qualitative analysis. *British Journal of Medical Psychology*, 73, 211-225.

Rhodes, J. E., Jakes, S. (2004) The contribution of metaphor and metonymy to delusions. *Psychology and Psychotherapy: Theory, Research and Practice*, 77, 1-17.

Romme, M., Escher, S. (1994) *Accepting Voices*. London: Mind Publications.

Sackeim HA. (1998) The meaning of insight. In: Amador XF, David, AS, eds. *Insight and Psychosis (1st ed)*. Oxford: Oxford University Press.

Sass, L.A. (1994) *The Paradoxes of Delusion: Wittgenstein, Schreber and the Schizophrenic Mind*. New York: Cornell University Press.

Schwitzgebel, E. (2002) A phenomenal, dispositional account of belief. *Nous*, 36, 249-275.

Sedler, M.J. (1995) Understanding delusions. *The Psychiatric Clinics of North America*, 18, 251-262.

Sims, A. (2003) *Symptoms in the mind: An introduction to descriptive psychopathology (3rd edition)*. Edinburgh: Elsevier Science Ltd.



Spence, S. A. (2001) Computer game delusions. *Journal of the Royal Society of Medicine*, 94, 369.

Spitzer, M. (1990) On defining delusions. *Comprehensive Psychiatry*, 31, 377-397.

Spitzer RL, Endicott J, Robins E. (1978) Research diagnostic criteria: rationale and reliability. *Archives of General Psychiatry*, 35 (6), 773-82.

Spitzer RL, First MB, Kendler KS, Stein DJ (1993) The reliability of three definitions of bizarre delusions. *American Journal of Psychiatry*, 150 (6), 880-4.

van Os, J. (2003) Is there a continuum of psychotic experiences in the general population? *Epidemiologia e psichiatria sociale*, 12, 242-52.

Wessely, S., Buchanan, A., Reed, A., Cutting, J., Everitt, B., Garety, P., Taylor, P. J. (1993) Acting on delusions (1): Prevalence. *British Journal of Psychiatry*, 163, 69-76.

Wittgenstein, L. (1953) *Philosophical Investigations*, translated by G. E. M. Anscombe, 3rd edition, 1967, Oxford: Blackwell.

World Health Organization (1979) *Schizophrenia: An International Follow-up Study*. Chichester: Wiley.

Young, A. W. (1999) Delusions. *The Monist*, 82, 571-589.

Young, A.W. (2000) Wondrous strange: The neuropsychology of abnormal beliefs. In M. Coltheart and M. Davis (eds) *Pathologies of belief* (pp. 47–74). Oxford: Blackwell.

## Chapter Three

### Delusions: Current Models and Empirical Evidence

Elements of this chapter are in publication as:

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Bell, V., Halligan, P.W. & Ellis, H. (2003) Beliefs about delusions. *The Psychologist*, 16(8), 418-423.

Bell, V. (2002) Book review: Pathologies of Belief by M. Coltheart & M. Davies. *Cognitive Neuropsychiatry*, 7 (4), 329-333.

The following presentations have also included material from this chapter:

Bell, V. What is the Role of Affect in Delusion Formation ? *Emotion Research Group, MRC Cognition and Brain Sciences Unit*, November 2004

### **3.1 Chapter outline**

Sections:

- 3.2 One-stage', 'two-stage' and multi-factor models
- 3.3 Motivational and 'defence' models
- 3.4 The 'Institute of Psychiatry' model
- 3.5 The 'metacognitive' or self-monitoring models
- 3.6 Hemispheric asymmetry models
- 3.7 Neuropathological and neurobiological accounts of delusion formation
- 3.8 Connectionist models
- 3.9 Chapter summary and conclusion
- 3.10 References

This chapter reviews the empirical and experimental literature on delusions. In particular, it identifies current models of delusion formation, or traditions in the empirical literature that have produced coherent explanations.

The chapter concludes by drawing on the first three chapters of this thesis to summarise where further research is needed (section 3.9) and how these areas will be addressed in the subsequent empirical chapters.

In particular, the chapter will argue that further research is needed in the following areas:

- i) The validity of the diagnostic criteria for delusions and a better understanding of the phenomenological complexity of delusional beliefs – addressed in Chapter 4.
- ii) A better understanding of the role of perceptual distortions in delusion formation, as perceptual accounts of delusions have been largely dormant. This needs two things:
  - a. an appropriate neutral language measure of anomalous perceptual experience – addressed in Chapter 5.
  - b. a better understanding of neuropsychological factors underlying anomalous perceptual experience, as it is currently conceptualised as a one factor continuum – addressed in Chapters 5 and 6.
- iii) A more complete understanding of what makes a belief pathological, as research has largely focused on beliefs identified by psychiatric diagnosis which has been shown to not necessarily be a marker for pathology – addressed in Chapter 7.

The final chapter of the thesis will evaluate this evidence in light of models of normal belief and delusion formation.

### **3.2 'One-stage', 'two-stage' and 'two-factor' models**

One-stage and two-stage models stem from deficit accounts of cognitive neuropsychology, and argue that delusion formation necessarily involves a pathology of experience (the first stage), but differ over whether a reasoning deficit (the second stage) is also necessary. Two-factor models see both these processes as important, but do not necessarily argue for a sequential first- then second-stage account, but instead, suggest that these factors may operate

in parallel and have differing degrees of influence. Two-factor models are also less wedded to a strict deficit account.

### **3.2.1 Maher and the classic ‘one-stage’ model of delusions**

The hypothesis that delusions are the result of normal reasoning applied to abnormal experience was first put forward by the French psychiatrist de Clerambault (1942; cited in Klee, 2004), although recent criticisms of the one-stage model are typically directed at the better specified theories of Maher (1974; 1988; 1999).

Maher’s approach has been heavily criticised, however, (Davies et al., 2001; Gilleen and David, 2005; Stone and Young, 1997), although it is not clear from reading Maher’s own work, whether these criticisms are aimed at a caricature of Maher’s views, or whether his approach is now so over-inclusive, as to include many of the widely implicated processes in delusion formation under the single banner of ‘perception’.

In Maher’s early work (e.g. Maher, 1974) the explanation for delusion formation is relatively clear-cut: “a delusion is a hypothesis designed to explain unusual perceptual phenomena and developed through the operation of normal cognitive processes”, which he claims are “indistinguishable from [those] employed by non-patients, by scientists, and by people generally”.

In his later work, however, he introduces mechanisms to account for why a person might need to seek an explanation for an anomalous experience, and to explain how perceptions are experienced as anomalous at all, both of which, despite his assertions, encroach on areas traditionally thought to be outside the first ‘perceptual’ stage of delusion formation.

Maher (1988) explains the process of ‘explanation seeking’ by noting that observations which do not match expectations cause a state of alertness and tension, and that the need for an explanation is driven by a desire to reduce tension and to obtain feelings of relief – in effect, a motivational or defence model of delusion formation (see section 3.3). Furthermore, when a hypothesis is formed, cognitive dissonance means that “data that are consistent with the explanation reduce dissonance and are given particular status in the explanation” (Maher, 1988, p20). This seems to suggest the presence of a data-gathering bias, although this, in itself, is still consistent with Maher’s theory, as this type of reasoning bias is commonly present in the general population (Gilovich, 1993; Mezulis et al., 2004). Presumably Maher is suggesting that the bias is neither more nor less strong in delusional patients, although he provides little justification.

In later work, however, Maher (1999) explains the origins of anomalous experience as including:

...a broad range of neuropsychological anomalies. These include, but are not confined to, (a) endogenous neural activation of the feeling of significance normally triggered by pre-conscious recognition of changes in a familiar environment; (b) unrecognized defects in the sensory system, such as undiagnosed hearing loss, or the endogenous activation or inhibition of the central neural representations of sensory input; (c) temporary alterations in the intensity and vividness of sensory input, as in some forms of drug intoxication; (d) neurologically based difficulties in the focusing of attention with consequent difficulty in discriminating between situationally relevant and irrelevant elements of the environment; (e) experienced discrepancies between the willed intent of a response and the actual form

of a response; (f) impairment in the monitoring and calculation of recurring sequential probabilities in environmental events

Most notably, (f) seems unequivocally to describe inferential reasoning, whereas (a) and (e) suggest a post-perceptual breakdown in metacognition (section 3.5) – (a) has been proposed as a metacognitive factor in dual-process models of memory involved in the control of memory retrieval (Koriat, 2000); and (e) in terms of the intention-monitoring system proposed by Blakemore et al. (2002; section 3.5.2). Similarly, (d) could equally describe any number of disorders to the high-level contention scheduling / supervisory attention model (Shallice and Burgess, 1998), which is thought not to have any direct role in perception.

As a more general point, Maher seems to rely on an implicit bottom-up understanding of perception, and leaves little room for the top-down influence of beliefs and concepts on perceptual experience. This top-down influence is now accepted as an essential part of the perceptual process and is supported by significant evidence in both the cognitive (Bruce et al., 2003) and neurocognitive literature (Bly and Kosslyn, 1997).

These analyses suggest that a ‘pure’ one-stage account of delusions is almost certainly untenable, and is best demonstrated by Maher’s increasing need to incorporate post-perceptual processes into his explanation of ‘perceptual disturbance’. Notably, Maher (1974) has stated that his approach may not apply to all delusions, only a subset, although it has never been made clear how this distinction may be applied in practice and no relevant criteria have been offered.



### 3.2.2 'Two-stage' models: The necessity of an additional reasoning deficit

In contrast to 'pure' one stage accounts, two-stage accounts argue that an additional pathology of reasoning is also needed for a delusion to form.

This view has largely been based on an understanding of so called monothematic delusions (particularly those arising after brain injury), and is, therefore, firmly rooted in a cognitive neuropsychological 'deficit' approach. In particular, many of the theoretical developments have been inspired by research into the Capgras delusion, providing strong evidence that, despite the fact that Capgras patients are able to consciously recognise the people they misidentify, they do not produce the same covert emotional response to familiar faces (Ellis et al., 1997; Hirstein and Ramachandran, 1997). This has suggested a mechanism whereby anomalous experience accounts for the *content* of a delusional belief.

It has been noted, however, that virtually identical results have been found in patients with damage to the ventromedial cortex (Tranel et al., 1995) without their becoming deluded, suggesting a second pathological reasoning stage is needed to explain why such an experience results in a delusion in Capgras patients, but not in patients with ventromedial damage (Ellis and Young, 1996; Ellis, 1998; Young, 1999). Davies et al. (2001) extend the argument to cover a number of other monothematic delusions. They describe cases from the literature that suggest that plausible aetiologically relevant anomalous perceptual experiences were present, but where the expected delusions did not occur, again suggesting the necessity of an additional pathology of reasoning.

The difficulty with using these studies as examples, however, is that many infer experience from relatively crude empirical measures. For example, a simple skin-conductance measure

was used to infer a lack of covert recognition in both Capgras patients (Ellis et al., 1997; Hirstein and Ramachandran, 1997) and in patients with ventromedial damage (Tranel et al., 1995), but it does not follow that these experiences are, therefore, identical, or even phenomenologically similar. This makes it difficult to use any examples inferred from such simple physiological measures as evidence against one-stage accounts.

Perhaps, a more telling criticism of the one-stage model (and, therefore, a justification for an additional factor) is the question of “whether it is possible - indeed, whether it is conceptually coherent - to posit that raw perceptual experience contains its own intrinsic thematic content (one-stage) or whether, instead, thematic content is always supplied by a distinct stage of cognitive interpretation (two-stage)” (Klee, 2004). Apart from the compelling evidence for top-down influence in perception, Klee’s point is particularly pertinent in light of research showing that the appraisal of an experience is the most likely mediator of whether a psychosis-like experience presents as illness behaviour (Chadwick and Birchwood, 1994; Gauntlett-Gilbert and Kuipers, 2005), and that cognitive-behavioural therapy can help resolve delusions by reappraising experiences in non-delusional ways (Chadwick et al., 1996).

One notable feature of early two-stage accounts (e.g. Ellis et al., 1997), however, is that they omit any specification of exactly what the second-stage might involve and how it might integrate into a belief formation model – typically relying on non-sequitur references to differences in inferential reasoning and attribution styles between delusional patients and controls (see sections 3.3; 3.4.4).

Perhaps to make up for this shortcoming, Langdon and Coltheart (2000) have put forward a two-stage model that aims to describe these processes in detail, and put them within a

comprehensive model of normal belief formation (section 1.6.1). The authors make the explicit distinction between factors that explain the content of delusions, and those that explain their presence. Perceptual experience, perhaps “nuanced” by attributional bias, is given as a factor that explains the content of an unusual hypothesis, but a deficit in belief-evaluation is given as an explanation for why such a hypothesis is not rejected and is maintained as a delusion.

By relying on perception to provide content, however, they are subject to the same criticisms of one-stage accounts mentioned earlier, and leave little room for the role of top-down influences.

In contrast to other two-stage models, Langdon and Coltheart suggest that in some cases, a single-factor deficit – a belief-evaluation deficit without any perceptual aberration – could lead to a delusion, although more likely to the non-bizarre sort, typically including, “for example, delusions of jealousy, the hypochondrical delusion that you are going to die, or the belief that someone important secretly loves you” (Langdon and Coltheart, 2000, p211). This is considered a minor route to delusion, as the most common result of this process is considered to be paranormal or supernatural ideas that would not be considered clinically relevant. This is perhaps curious, as the most prevalent delusion is the persecutory type (Sartorius et al., 1986), of which common presentations would fulfil Langdon and Coltheart’s criteria for being non-bizarre and not based on obvious perceptual aberration. This suggests that their model may be too focused on bizarre delusions to be a complete account (perhaps, because of the reliance on the relatively rare occurrence of monothematic delusions after brain-injury), despite its obvious advantages in terms of clarity and integration with normal belief formation.

### **3.2.3 'Two-factor' models: Balancing observational adequacy and explanatory conservation**

An alternative approach is taken by two-factor models, that argue that both anomalous perceptual experience and pathological reasoning contribute to delusion formation, but that these do not necessarily occur in series, and may be aspects of a belief-formation system that attempts to reconcile perceptual experience with pre-existing knowledge.

The main thrust of two-factor models stems from an analysis by Stone and Young (1997), who argue that belief formation involves balancing two opponent forms of explanation: explanations that are observationally adequate, with explanations that fit within a person's belief set with the minimum of cognitive reorganisation.

#### *3.2.3.1 Davies et al. (2001) model*

As with two-stage models, the two-factor model of Davies et al. (2001) relies on arguments noting the similarity between anomalous experiences in delusional and non-delusional conditions to justify the need for a second factor. Davies et al. extend the argument from the simple comparison between SCR responses between Capgras patients and the ventromedial damage patients reported by Tranel et al. (1995) to encompass a number of non-delusional examples. They include the delusions of Capgras, Cotard, alien control, denial of limb ownership and mirrored self-misidentification in their analysis, although they admit a failure to find non-delusional experience related to Frégoli syndrome, reduplicative paramnesia and thought insertion. Although perhaps patient MR of Ward et al. (1999), who showed inappropriate familiarity for faces (an experience he described as "seeing film stars everywhere"), and Spence's (1996) highlighting of a proposed continuum between inserted

thoughts in psychosis and everyday spontaneous thoughts, suggests at least two of these gaps could be filled.

While Davies et al. argue that these examples show the need for an additional factor, they also posit two routes to delusion on which such a factor could operate: either by producing an explanation for the existence of an unusual experience, or simply believing what is perceived (see figure 3.1).

Crucially, they argue that the role of a second factor is not to explain why unusual hypotheses are generated, but why they are not rejected in light of previous knowledge and testimony from other people. The two routes might involve different weightings between the first- and second-factor (in the first route, they claim 1<sup>st</sup> person perceptual experience has a much stronger influence), but overall the second factor involves a loss of the ability to reject a candidate belief on the grounds of everything else the patient knows.

The authors do not make any serious attempts to specify the mechanisms of this second factor, except for vague mentions of possible cognitive biases. Nevertheless, they do specify two things which they see as major problems for accounts of the second factor: namely, the monothematicity of some delusions, and the fact that some patients can appreciate how implausible the delusion might sound to others despite believing it themselves. These objections, however, are not likely to be significant obstacles.

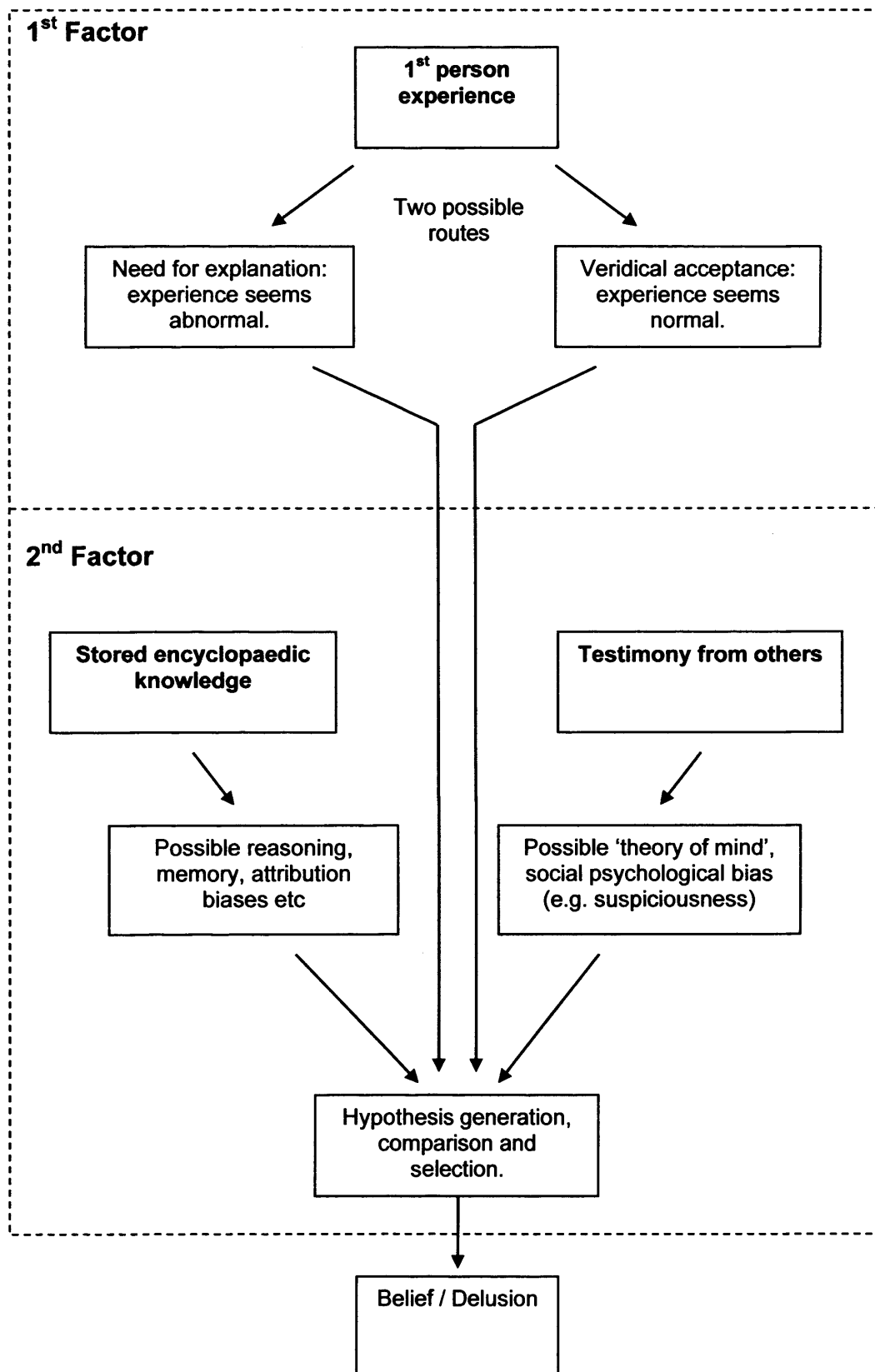


figure 3.1 - Davies et al. (2001) Two-factor model of delusion formation.  
 Diagram created for this thesis.

Davies et al. consider monothematicity to be a problem for any explanation that involves a global pathology of belief formation, as the authors argue that all beliefs should be pathological under such conditions. This is a common fallacy, however, as global influences do not necessarily produce significant effects in all situations, and may only result in a noticeable difference during an interaction with other factors. This is a common finding in psychology and applies equally to delusions (Bell, 2002). It also demonstrates a potential bias from drawing conclusions from pathology that arises largely from brain injury, where more wide-reaching cognitive deficits may be apparent.

Empirical evidence suggests, however, that many delusions normally classified as 'monothematic' (e.g. the Capgras delusion) are most prevalent in paranoid schizophrenia (Forstl et al., 1991), which typically involves polythematic or elaborated delusions. This suggests that many of the cases of 'monothematic' delusions reported in the literature are more apparent than real, owing to sample bias in reporting only the delusion of interest. Furthermore, the whole idea of a monothematic delusion is an unfalsifiable hypothesis (there may always be another delusion that has yet to be found), although admittedly, in practice, patients may only seem preoccupied with a few, rather than many beliefs.

The appreciation of implausibility involves patients having a delusion, and yet realising that others might find the same belief entirely unbelievable. This, however, does not seem to distinguish normal belief from delusional belief in any significant sense. For example, a vegetarian might believe it is wrong to eat meat, but yet realise that others find this implausible. Even beliefs directly derived from perception can be subject to the appreciation of implausibility, as there are a number of ways to justify the differentiation of one's own first person experience from someone else's. Indeed, court cases typically involve conflicting eye

witness reports of the same event where each party might both believe their experience, but appreciate why other people do not, and yet have adequate self-justification for why their belief is the correct one and the other party holds a false belief.

These counter-examples serve to refute the two issues which Davies et al. cite as problems for the second factor, although it is important to note that this does not significantly weaken their own cognitive account. The objections raised by Davies et al. were general points concerning all models that attempt to account for a second stage or factor, and their cognitive account was developed in spite of these issues.

### *3.2.3.2 McKay et al. (2005) model*

Although Davies et al. avoid exploring the mechanism of a second-factor in detail, it is tackled by McKay et al. (2005), who attempt a neuropsychological explanation based on their model of normal belief formation (section 1.6.3). Notably, their account does more than simply specifying a second-factor, it reframes earlier models to take account of motivational factors in belief formation, and denies that the presence of anomalous experience is needed in all cases of delusion. Because of this, they label their model a ‘modified two-factor account’ (see figure 1.4).

McKay et al. note that earlier work considers the first factor to account for the content of a delusion, whereas the second factor accounts for why a certain delusory hypothesis, once generated, is then adopted and maintained. Previously, the first factor was largely considered linked to neuropsychological deficits that cause anomalous perceptual experience. They argue, however, that the first factor should be reconsidered also to include emotional defences, desires and motivations. The second factor might then involve a deficit or bias that



gives undue credence to such evidence, meaning “they would thus be prone to giving undue weight to veridically dubious sensory information, as well as liable to having their belief-formation systems derailed and overridden by their motives”.

The model goes further, however, arguing for a neuropsychological account of this process based on Ramachandran’s work on anosognosia and self-deception (Ramachandran, 1994a; 1994b; 1995; Ramachandran and Blakeslee, 1998). Ramachandran argues that the left hemisphere is involved in the ‘defensive’ process of filtering or selecting aspects of information to make it best fit into the pre-existing web of beliefs; whereas, in contrast, the right hemisphere is a ‘discrepancy detector’ and re-organises beliefs in response to new information. These processes are contradictory, but, in a similar way to Stone and Young’s (1997) suggestion, a belief could be the outcome of the optimal satisfaction of these competing constraints.

Impairment to the right hemisphere, therefore, leaves potential defence processes unchecked, and makes it difficult for individuals to revise their beliefs in accordance with sources of information other than these desires (and presumably) 1<sup>st</sup> person perceptual experience. McKay et al. suggest that this may be the neuropsychological basis of the second-factor, identified by Davies et al. as the loss of the ability to reject a candidate belief on the grounds of everything else the patient knows.

This account is not without problems, however. Perhaps the most obvious is that, although the continuum model of psychosis has suggested that delusions are the extreme end of a distribution of belief (see section 2.5.3), there does not seem to be a clear distribution in hemispheric asymmetry between normal, paranormal and delusional beliefs as might be

expected. Although for at least some sections of psychosis continuum, the relationship does seem to hold (see section 3.6 for a full discussion of this point). It also relies on a somewhat crude whole hemisphere model of delusion and belief formation, which provides little explanatory power when aiming to integrate it into other, more precise, neuropsychological models (section 3.6; Chapter 6).

#### **3.2.4 ‘One-stage’, ‘two-stage’ and ‘two-factor’ models section summary**

One of the most commendable aspects of these models, particularly in the latter ‘two-stage’ and ‘two-factor’ models, is their attempt to explain delusion formation in the framework of a model of normal belief formation. Nevertheless, in contrast to other models of delusion formation, it is notable that these models actually provide little empirical evidence for what the first- or second-factors might involve, and, more particularly, how they may be linked.

Perhaps most striking, is that the role of anomalous experience, thought to be crucial in almost all the models (arguably the modified two-factor model of McKay et al. could account for delusions without significant perceptual distortion), is provided as if it was an explanation, when really it is nothing more than a vague description of clinical phenomena. As can be seen from the rest of this chapter, this is a common factor in almost every model of delusion formation that includes the role of anomalous experience. It is clear that anomalous perceptual experience, and its relation to a potential second factor, needs to be quantified for any empirically based theory to advance.

Moreover, one aspect barely considered by these models is the ‘pragmatic pathology’ of a delusion. In light of evidence from studies showing that approximately 10% of the general population score above the mean of psychotic inpatients on measures of delusional ideation

(Peters et al., 1999a; 2004), it is important to distinguish between beliefs that cause impairment and disability (those that are 'pragmatically pathological'), and those that are simply 'magical' or anomalous. The models outlined in this section only address the formation of anomalous beliefs and have little to say about pragmatic pathology, suggesting they are not complete models of delusion formation (an issue addressed in Chapter 7).

The explicit incorporation of the concepts of emotion, defence and motivation in the McKay et al. model are an innovation for neuropsychological models. As noted in section 3.2.1, this concept has also been implicitly included (although largely ignored by most commentators) in Maher's 'one-stage' account. These concepts have traditionally played an important part in purely cognitive explanations for delusion formation, and will be considered in the following section.

### **3.3 Defence and motivational models**

Motivational models stem from the psychoanalytic concept of 'defence', in that delusions are explained as resulting from attempts to cope with, recover from, or repress, unwanted or painful emotions, albeit, in a maladaptive way. Although this could encompass virtually any negative emotion (Bell, 2003; uses the catch-all label 'inner catastrophe') the theories of Bentall and colleagues have put the concept of self-esteem at the core of this explanation. In contrast to deficit driven models of delusions, motivational models depict delusions as resulting from excessive bias in the belief formation system, rather than catastrophic damage to a particular component.

### **3.3.1 Early defence models**

Although largely eschewing work on psychosis for neurotic disorders, one of Freud's rare forays into explaining delusion was his analysis of Daniel Paul Schreber's account of his own extensive psychotic episode (Schreber, 2000). Freud argued that Schreber's delusions resulted from the 'sublimation' of his personally unacceptable homosexual desire for his psychiatrist into more acceptable, but pathologically bizarre, beliefs (Freud, 1911).

Shortly after, a strikingly modern account of delusion formation was put forward by psychoanalyst Alfred Adler, who argued, in an essay entitled 'Melancholia and Paranoia', that paranoia results from a tendency to blame others for unfortunate events, in an attempt to mask an underlying inferiority complex (Adler, 1914).

More contemporary motivational accounts include Zigler and Glick (1988), who suggested that paranoid schizophrenia may be a form of 'camouflaged depression'; and Roberts (1991), who argued for a similar defence account, based on findings that on a measure of perceived purpose and meaning in life, chronically deluded patients scored more highly than similar patients with remitted delusional symptoms. The delusional patients also showed less depressive and suicidal thinking, leading Roberts to suggest delusions act as a defence against such negative emotions.

### **3.3.2 Bentall and colleagues' model of persecutory delusions**

The theories put forward by Richard Bentall and his collaborators (henceforth called the 'Bentall model') are notable as contemporary 'defence' theories of delusion formation, owing to the considerable amount of empirical investigation they have been subject to, and the

specification of the theory in terms of cognitive psychology, particularly in terms of attributional bias and self-esteem.

The Bentall model is also notable for restricting itself to explaining persecutory delusions, rather than delusions in general, and is not based on a model of normal belief formation. In Langdon and Coltheart's (2000) terms, therefore, the model attempts to explain why a persecutory delusion may exist, but not why that delusion has a particular content (such as why the persecutors are perceived as being neighbours, the CIA, and so on).

The original Bentall model has been revised to take into account evidence from experimental testing. The sections below discuss the original 'early' Bentall model and the more recent revised theory, called the 'Attribution / Self-representation model'.

### *3.3.2.1 The 'early' Bentall model*

The early Bentall model (Bentall et al., 1991; Kaney and Bentall, 1989; Kaney and Bentall, 1992; Lyon et al., 1994), focuses largely on the role of self-serving attribution biases and their extreme expression in patients with persecutory delusions. The self-serving attribution bias is the effect whereby people tend to attribute positive events to their own actions and negative events to external causes (reviewed in Mezulis et al., 2004). Bentall and his collaborators argued that patients with persecutory delusions make excessive external-personal attributions ('it was caused by someone else') for negative events, in an attempt to protect self-esteem from being negatively affected.<sup>1</sup>

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<sup>1</sup> This formulation of the Bentall model, therefore, is largely a restatement of Adler's (1914) account of paranoia (albeit with rigorous empirical testing), although presumably, developed independently, as Adler's work has never been referenced, even in the most recent reviews (Bentall et al., 2001; Bentall, 2003).

Indeed, patients have been reported as showing such attributions using the Attributional Style Questionnaire (Kaney and Bentall, 1989; Candido and Romney, 1990; Lyon et al., 1994; Fear et al., 1996; Sharp et al., 1997), the Social Attributions Questionnaire (Bentall et al., 1991; Young and Bentall, 1997), the Internal, Personal and Situational Attributions Questionnaire (Kinderman and Bentall, 1997), the Attributional Style Structured Interview (Craig et al., 2004) and with content analysis of natural speech (Lee et al., 2004).

These studies, and previous reviews of the literature (Garety and Freeman, 1999; Bentall et al., 2001), support the broad hypothesis that persecutory delusions are associated with external-personal attributions for negative events. More problematic, however, has been the self-esteem component.

The Bentall model predicts that patients with persecutory delusion should have high or normal levels of self-esteem, as their pathological beliefs should be the maladaptive result of a successful defence against low self-esteem. Nevertheless, the findings on self-esteem in such patients have been equivocal. For example, although Candido and Romney (1990) and Lyon et al. (1994) reported high levels of self-esteem in delusional patients, consistent with this aspect of the Bentall model, other studies have reported contradictory findings.

Freeman et al. (1998) reported generally low levels of self-esteem in a group of similarly deluded patients, whereas Ellett et al. (2003) reported that self-esteem was negatively correlated with levels of paranoid ideation in a non-clinical sample. Similarly, Bowins and Shugar (1998) reported that the content of a delusion was congruent with self-esteem, suggesting it was not serving a defensive function.

Non-clinical studies have also suggested evidence against a self-esteem based model of delusions. A recent meta-analysis on the stability of self-esteem across the life span suggested that self-esteem is most stable during adolescence and early adulthood (Trzesniewski et al., 2003), the time when the prevalence of both frank psychosis and sub-clinical psychosis-like experience is at its highest (Gonzalez-Pinto et al., 2004; van Os et al., 2000). Furthermore, the stability of self-esteem seems incompatible with the relative instability of delusions (see section 2.4.1), with Myin-Germeys et al. (2001) reporting that delusional conviction can vary considerably within the span of even a single day.

In response to contradictory evidence such as this, Bentall and colleagues revised the early model to include concepts of implicit and explicit self-esteem, and the concept of actual-self / ideal-self discrepancies.

### *3.3.2.2 Attribution / Self-Representation model*

Bentall et al. (1994) introduced Higgins' (1987) concept of 'self-discrepancies' into their model, where inconsistencies between how one perceives the current self (the 'actual self') and how one conceives of the 'ideal-self' can lead to negative affect, owing to the activation of latent negative beliefs about the self. They further argue that persecutory delusions are the result of trying to keep self-discrepancies to a minimum, by making external personal-attributions, which are neutral with regard to the self, as they attribute negative events to the actions of other people.

One implication, is that latent negative beliefs should be detectable as low self-esteem using implicit measures, even when patients report high or normal self-esteem on explicit measures.

This distinction is also used to explain why the predictions of the 'early' Bentall model, that considered self-esteem as a global or unitary construct, were not fully supported by the empirical evidence (Bentall et al., 2001).

Reliable evidence for a clear difference between implicit and explicit self-esteem in delusional patients has been elusive, however (Garety and Freeman, 1999). Studies using the emotional Stroop task have found evidence that colour naming was slowed for low self-esteem words in both clinical cases (Kinderman, 1994; Lee, 2000) and with high-scoring students on a measure of paranoid ideation (Lee, 2000), although interference was also found for high self-esteem words in patients. Other studies have failed to find an interference effect on the Stroop task for negative words (Bentall and Kaney, 1989; Fear et al., 1996). In contrast, Lyon et al. (1994) did find self-esteem effects in the predicted direction using the Pragmatic Inference Test, although this result was not replicated by Kristev et al. (1999) or Martin and Penn (2002).

A recent related study investigating patients with grandiose delusions failed to find any difference between implicit and explicit self-esteem (Smith et al., 2005), although it is not clear how relevant this is to the strict Bentall model, which aims to account for persecutory delusions only. Finally, a study using the Implicit Attribution Test (Moritz et al., in press) did find low levels of implicit self-esteem in delusional patients, and although the presence of delusions was associated with higher explicit self-esteem than in non-delusional patients, the level was still lower than in healthy controls.

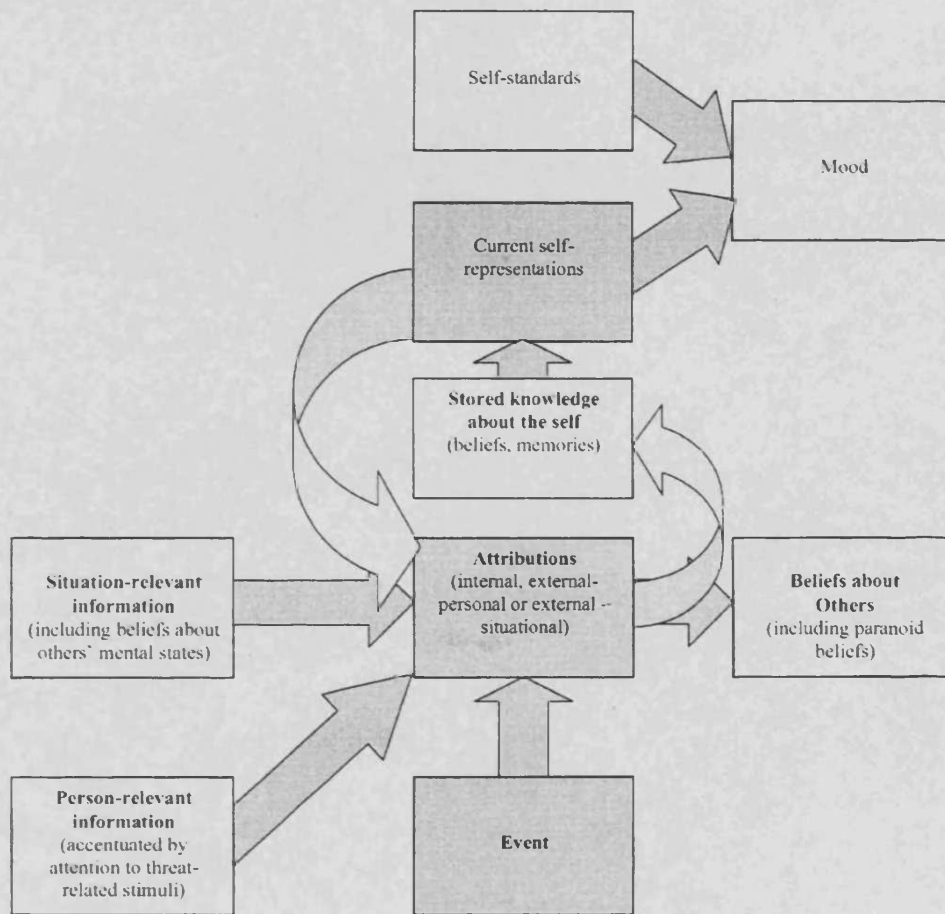
In the most recent review of the model, however, Bentall et al. (2001) have noted evidence suggesting that, as well as the effect of attributions on mood and self-esteem, mood and self-



esteem can also influence attributions. This suggests a cybernetic 'feedback loop' model, in which persistent negative events or persistent low mood or self-esteem lead to a bias for internal attributions for negative events, which consequently leads to a further lowering of self-esteem.

To arrest this downward spiral into 'inner catastrophe', external-personal attributions are made with the intention of stabilising the system. According to Bentall et al., the 'psychologically benign' external-situation attribution, although less pathological, is much more effortful and involves substantially greater cognitive load, meaning external-personal attributions are more common, especially in times of stress. Large numbers of external-personal attributions, therefore, lead to the formation of persecutory delusions.

This model is illustrated by the diagram from Bentall et al. (2001) in figure 3.2, although this does not sufficiently describe the functional flow of the model, and so has been reformulated in figure 3.3.



*figure 3.2 Attribution / Self-Representation cycle.  
Original diagram from Bentall et al. (2001)*

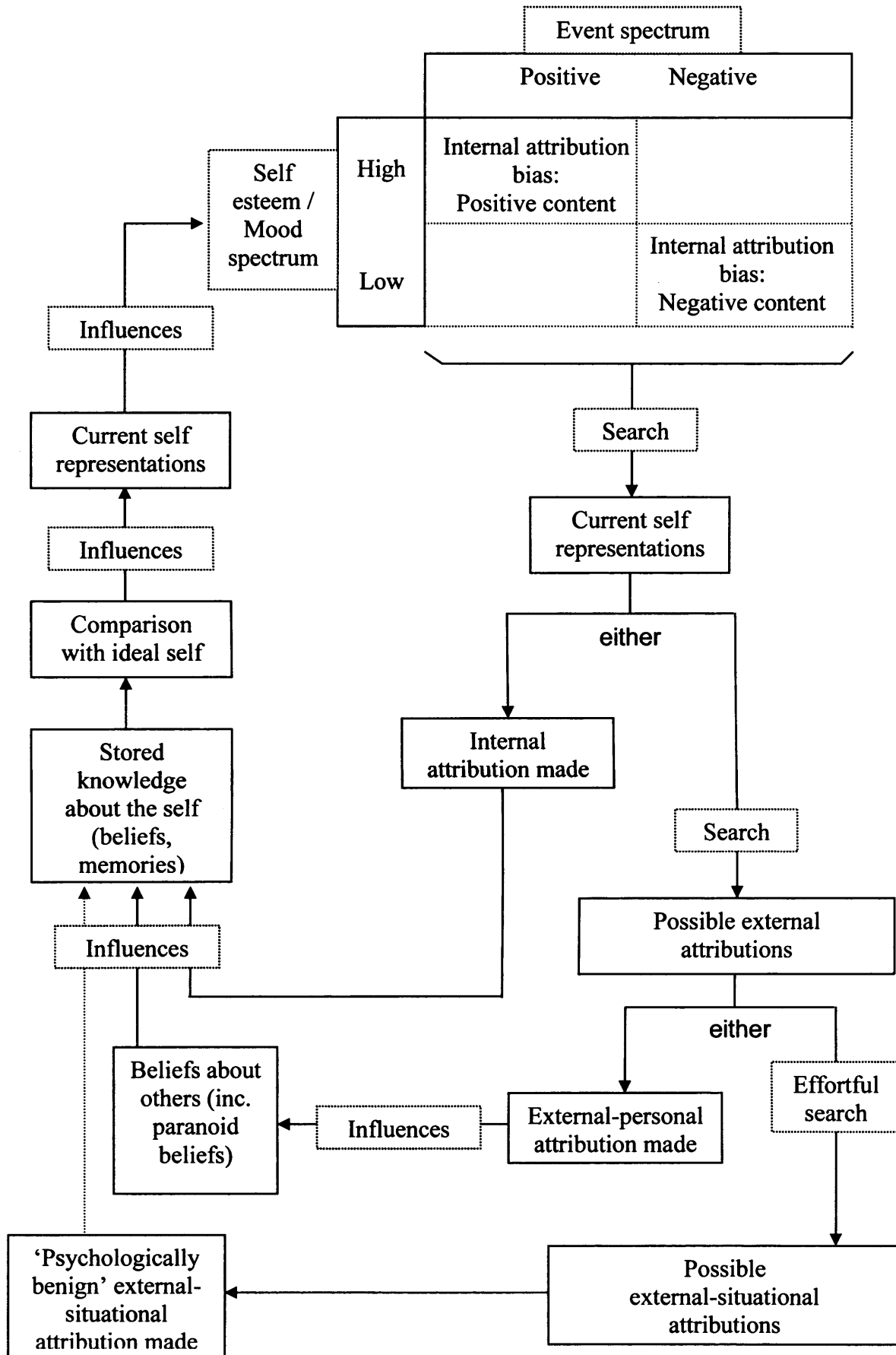


figure 3.3 Alternative representation of Attribution / Self-Representation Cycle  
Diagram created for this thesis.

The inclusion of a dynamic aspect to the model also allows the Bentall model to counter criticisms concerning the contradictory self-esteem evidence (Bentall et al., 2001), as follows:

We have seen that the theory of the attribution–self-representation cycle does not lend itself to precise predictions about self-esteem in paranoid patients. Indeed, we have speculated that some paranoid patients may eventually reach a relatively stable state in which global self-esteem is high, others may reach an equally stable state in which self-esteem is low, and still others may oscillate between periods of high and low self-esteem.

Although this may lead to accusations of the model being untestable, there is some evidence that self-esteem is dynamically affected by attributions in both healthy participants (Kinderman and Bentall, 2000) and delusional patients (Kinderman et al., 2003). The dynamic nature of this model, and the validity of its predictions have yet to be comprehensively tested, however, and so the model must remain speculative until further evidence is gathered.

### **3.3.3 Defence and motivational models section summary**

The most recent version of the Bentall model has yet to be rigorously tested, in light of the shortcomings in empirical support for key aspects of the earlier model, and is notably not a model of belief formation. It relies firmly on a common-sense understanding of belief, and, although not explicitly stated, it is also significant that the Bentall models suggest that delusions are the natural result of a pathological attributions. Here, it seems, the belief formation system, although unspecified, is working as it should (there are no ‘damaged components’ or ‘deficits’), despite input being biased by a compromised general purpose attribution system. Nevertheless, the proposal that patients with persecutory delusions show

an externalising bias is well supported, suggesting that this may be a central factor in delusion formation (Garety et al., 2001).

Because it is not framed within a model of normal belief, however, it is difficult to draw any conclusions about a general model of belief formation from its proposed operation. Although this is a disadvantage for the project of cognitive neuropsychiatry, the model may provide evidence to indicate important attributional influences that are involved in (and potentially bias) belief-based behaviour.

Bentall's model also highlights the role of emotion in delusion formation, something that the 'one-stage' and 'two-stage' models of delusions almost entirely omit (see sections 3.2.1 and 3.2.2). The evidence for the role of self-esteem is mixed, however, and some authors have even questioned the concept itself. One recent review noted that measures of self-esteem predict few behaviours reliably (Baumeister et al., 2003), suggesting the validity of the construct could be questionable.

Although the Bentall model was not originally neuropsychological, it is noteworthy that the most recent developments of the 'two-factor' models (particularly the McKay et al. model; see section 3.2.3.2), now include 'defence' and 'motivational' aspects as key components. Furthermore, recent functional imaging studies inspired by Bentall's model (Blackwood et al., 2000; 2001; 2003; 2004) have suggested they are compatible with a neuropsychological approach, and that identifiable neural circuits are emerging that might support the key attributional biases. This suggests that the Bentall model has the potential to be integrated with a neuropsychological model of belief. Although this has not been attempted as yet, as it

is doubtful whether either model is sufficiently specified to make anything except speculative guesses as to the likely points of contact.

### **3.4 The 'Institute of Psychiatry' (IoP) model**

A coherent model of delusion formation has emerged from researchers based at the Institute of Psychiatry (Kings College London), most recently outlined in Freeman et al. (2002) and Freeman and Garety (2004). While Freeman and Garety focus on persecutory delusions, it is clear that the model is more widely applicable and can be considered a general model of delusion formation in many respects.

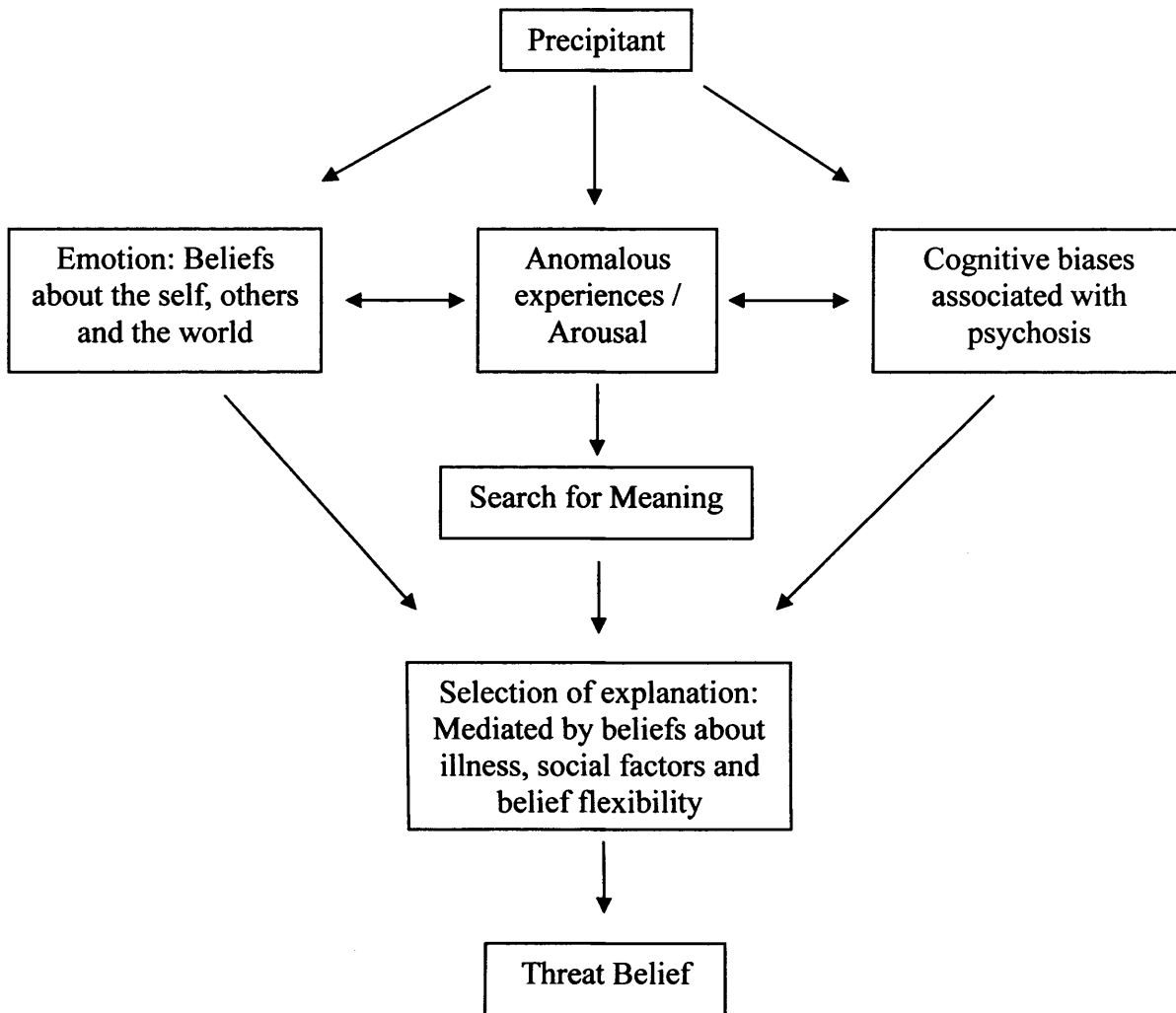
In contrast to 'defence' models, the IoP model attempts to argue for a direct influence of emotion on the likely presence and content of delusions. Furthermore, the model explicitly addresses two different aspects of the presence of delusions (in effect, being two different, but overlapping, models). These are formation – why a delusion exists in the first place; and maintenance – why a delusion is not rejected over time.

#### **3.4.1 Institute of Psychiatry delusion formation model**

The model, summarised in figure 3.4, is based on 'stress-vulnerability' or 'stress-diathesis' approaches that argue that psychosis is caused by an interaction between pre-existing vulnerabilities (including genetic, epigenetic and intrauterine factors) and stressful life events.

##### *3.4.1.1 Precipitants*

Particularly if occurring in an individual with a history or predisposition to depression or long-term anxiety, a precipitant event, such as stress, trauma or drug use, could lead to an significant increase in anomalous experience, arousal, or emotion.



*figure 3.4 Delusion formation model from Freeman and Garety (2004).*

### *3.4.1.2 Anomalous experience, arousal, emotion*

Anomalous experience could result directly from the event (for example, drug use causing perceptual distortions) or from an increase in arousal and anxiety associated with the event.

This could also cause up-regulation of cognitive biases known to be associated with psychosis and vulnerability to psychosis, which are often exacerbated by emotional disturbance.

Mechanisms given for the production of anomalous experience include the ‘weaking of memories of input regularities’ leading to dysfunctional perceptual filtering, described by

Hemsley (1994), abnormal perceptual bias and discrimination, described by Frith (1992), and the experience of aberrant salience, described by Kapur (2003). This is thought to have a reciprocal effect on arousal and information processing, as per Gray's (1982) 'behavioural inhibition model', where novel or unexpected stimuli are linked to arousal, behavioural inhibition, and increased attention to the environment.

#### *3.4.1.3 Search for meaning*

The presence of anomalous experience, abnormal arousal or recent events, will trigger a search for meaning, as described by Maher (1988), leading to an individual, in an attempt at explanation, forming hypotheses that will cause 'relief' owing to a reduction in dissonance.

The anomalous experience may also be included in either the 'event' or as data for the explanation.

Pre-existing beliefs about the self, the world and others are drawn upon to explain recent experiences. The availability and weighting of these beliefs in the reasoning process could be influenced by the current emotional state, ongoing anomalous experiences and cognitive biases associated with psychosis, such as a 'jumping to conclusions' reasoning bias (Garety and Hemsley, 1994; section 3.4.4), attributional biases (Bentall et al., 2001; section 3.3), and 'theory of mind' problems (Frith, 1992; section 3.5.1.1). Notably, it is argued that the emotional state contributes 'directly' to the theme of the delusion (Freeman and Garety, 2004; section 3.4.3), rather than in a 'defence' manner, as in the Bentall model.

Social factors also play a role in the model, including isolation, genuine hostility and victimisation (which patients diagnosed with, for example, schizophrenia are known to suffer at a far higher rate than the general population; Fitzgerald et al., 2005).



Furthermore, the final explanation is likely to be influenced by the individual's beliefs about mental illness and madness (e.g. a person finding the idea of 'going mad' very distressing may be much more likely to entertain an external cause for anomalous experience), social factors (such as isolation and genuine hostility in their environment) and poor belief flexibility (Freeman et al., 2004; Hurn et al., 2002).

### 3.4.2 Institute of Psychiatry delusion maintenance model

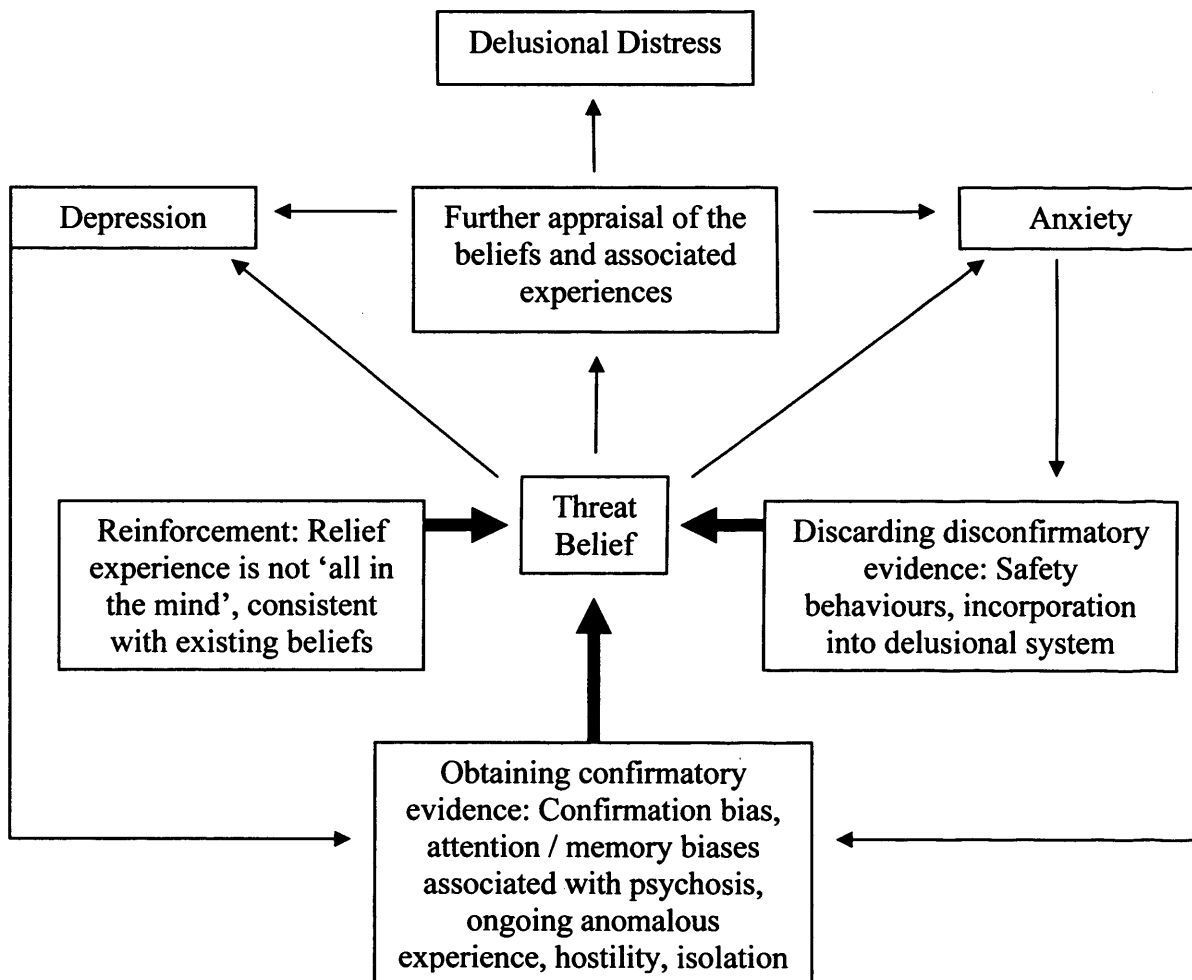


figure 3.5 Delusion maintenance model from Freeman and Garety (2004).

The model that deals with delusion maintenance (outlined in figure 3.5) can be seen to overlap with the formation model, particularly with regard to processes thought to be involved in the 'search for meaning' (section 3.4.1.3).

The model considers two main delusion maintenance processes as key:

- i) the obtaining of confirmatory evidence
- ii) the discarding of disconfirmatory evidence

And also considers the delusion to be reinforced by the 'relief' achieved by accepting an explanation for anomalous experience, as suggested by Maher (1988; section 3.2.1).

#### *3.4.2.1 Confirmatory evidence biases*

Belief confirmation bias, where positive evidence is favoured in belief-based reasoning, is a common cognitive bias in the general population (Gilovich, 1993) and this is known to be exaggerated in people high in delusional ideation (Freeman et al., 2005). This may also include a 'jumping to conclusions' reasoning style (Garety and Hemsley, 1994; section 3.4.4).

Attentional biases may also be involved, that produce a tendency to preferentially process delusion congruent stimuli. Attentional bias studies have typically focused on threat-related stimuli and persecutory delusions, and have found effects using the Stroop task (Bentall and Kaney, 1989; Fear et al., 1996; Kinderman et al., 2003), visual scan path analysis (Phillips et al., 2000), face perception tasks (Green and Phillips, 2004) and a self-relevance decision task (Blackwood et al., 2000). Other studies that have looked at psychosis not classified by theme, or in those with high schizotypy, have also found biases in attentional filtering using

techniques such as latent inhibition or pre-pulse inhibition paradigms (Abel et al., 2004; Gray et al., 2002; Gray and Snowden, in press, for a review).

According to the IoP model, the ongoing emotional state is likely to contribute to the theme of the delusional belief, in that the cognitive content of emotions will have been expressed in the delusion (section 3.4.3) and, in turn, the content of the delusions will contribute to the maintenance and exacerbation of the emotion. This is likely to involve emotional memory biases, that may increase the likelihood of retrieving emotion congruent memories and decrease the chances of retrieving incongruent or neutral material (a process reviewed in relation to delusions by Gibbs and David, 2003). Similarly, trauma-driven intrusive memories and the ongoing contribution of emotion to anomalous experience may also provide further confirmatory evidence for the delusion (Steel et al., 2005).

#### *3.4.2.2 Disconfirmatory evidence biases*

The IoP model suggests that a major way in which disconfirmatory evidence is avoided is by the use of safety behaviours, which serve to decrease anxiety, by either avoiding the perceived source of anxiety, or by completing some behaviour which is thought to ‘ward off’ or protect against the delusional threat. Because these behaviours are perceived as preventing the threat from occurring, the belief is never shown to be false. Freeman et al. (2001) found 100% of patients with persecutory delusions reported some form of safety behaviour, suggesting that this form of bias is a common feature of psychosis. This is one aspect, however, which may be specific to persecutory delusions rather than a delusions in general, owing to the association of safety behaviours with anxiety (Salkovskis, 1991).

One further mechanism, given by Freeman and Garety (2004) is a process of assimilation (not unlike the concept used in McKay et al's model; section 3.2.3.2) whereby disconfirmatory evidence is incorporated into the delusional system (the evidence suggests that "they are playing with my mind / teasing me / they will get me some day" etc).

#### *3.4.2.3 Explanation based reinforcement*

The IoP model also includes Maher's (1988) suggestion that delusional beliefs will be reinforced by the 'relief' provided when an anomalous experience is explained after the 'search for meaning' (discussed in section 3.2.1).

#### **3.4.3 Is there evidence for a 'direct' influence of emotion on delusions?**

There are now many studies which have found links between emotional disturbance and delusional or psychotic phenomena. For example, significant negative affect has been found in patients in the prodromal phases of paranoid schizophrenia (Gourzis et al., 2002), in psychotic outpatients (Steer et al., 2003) and has been associated with the intensity of delusional conviction (Garety et al., 2005; Myin-Germneys et al., 2001). Conversely, a study of the general population found that 10.7% of those reporting feelings of worthlessness or guilt had delusions (Ohayon and Schatzberg, 2002).

One of the key aspects of the IoP model is that, in contrast to 'defence' accounts, specific emotions may directly contribute to the formation, theme and maintenance of delusional beliefs (see table 3.1).

<i>Emotions</i>	<i>Main theme of emotion</i>	<i>Main theme of delusion</i>
Anxiety	Anticipation of physical, social, or psychological threat	Reference ('People are watching me') Persecution ('People are saying negative things behind my back to get at me')
Depression	Loss, low self-esteem, guilt, shame	Guilt ('I've brought ruin to my family') Persecution ('I'm being persecuted because of what I've done in the past') Catastrophe ('The world is going to end and it's all my fault')
Anger	Deliberately wronged, frustration at not reaching goal	Persecution ('People are doing things to annoy me')
Happiness	Success, achievement, high self-esteem	Grandiose ('I've got special talents and am related to a famous person')
Disgust	Finding something offensive, revulsion, dislike.	Persecutory ('My food is being poisoned') Hypochondriacal ('My insides are rotting') Appearance ('My body is ugly and misshapen')
Jealousy	Fear of losing another's affection	Jealousy ('My wife is sleeping with other men in our bed while I lie asleep')

*table 3.1 The themes of emotions and delusions (from Freeman and Garety, 2004).*

From this analysis it certainly seems plausible that emotion directly contributes to the theme of a delusion, although Freeman and Garety's reasoning for excluding religious delusions, on the grounds that they are "secondary elaborations of psychotic experience or other delusions" (p32), seems a little trite, considering many would consider mystical experiences, or mystically themed delusions, to be valid self-supporting categories distinct from the normal range of emotion (Clarke, 2001). Similarly, there are a number of delusional beliefs that may not be adequately covered by this emotion-equivalence analysis, such as delusions of control (Hohwy and Rosenberg, 2005), or more obscure phenomena, like lycanthropic delusions (Garlipp et al., 2004).

A further criticism, similarly aimed at one- and two-stage models (that argue experience can provide content), is that it is difficult to make exact equivalences between a particular emotion and a particular belief (Klee, 2004; section 3.2.2). Some theorists working on normal belief, however, see emotion as focusing belief, even on quite specific topics. Clore and Gasper (2000) give three processes by which emotion can focus belief, emotion as attention (a selective attention bias), changing goal focus (via positive feed-back loop) and emotion as information. Indeed, this final process is typically given as an explanation for Capgras delusion (Ellis et al., 1997) and is a component of Damasio's (albeit controversial) 'somatic marker hypothesis' (Damasio, 1996). This evidence, and a wide range of other studies in the literature on normal belief (see Frijda et al., 2000), suggests that a direct influence of emotion is quite plausible.

Perhaps more contentious, is Freeman and Garety's (2004) claim that their explanation of the role of emotion excludes 'defence' approaches, championed by Bentall (section 3.3). For example, a recent study from the same group, found no difference in implicit and explicit self-esteem in patients with grandiose delusions, something which the authors claim shows that delusions are a direct representation of the emotional state of the patient, rather than a defence against negative affect (Smith et al., 2005).

Their wider model, however, contains one element in particular which is suggestive of a defence account, namely their inclusion of Maher's (1988) hypothesis that delusions serve as 'relief' from tension or dissonance caused by unexplained anomalous experience. As discussed in section 3.2.1, this is, to all intents and purposes, a 'defence' account of delusion formation, although in the IoP model it might be better considered a component, rather than an account in itself. How this fits with aspects of their model that suggest that delusions can

'feedback' to increase negative affect has not been explained, although it is likely that these may be opponent processes, and that each has a varying influence depending on the theme and content of a particular delusion.

#### **3.4.4 How much do 'jumping to conclusions' biases contribute to delusion formation?**

One of the first systematic attempts to uncover a specific cognitive bias associated with delusions resulted in the finding that delusional patients seem to show a 'jumping to conclusions' reasoning bias (Huq et al., 1988), where they make decisions based on less data than healthy or psychiatric controls. This was followed up by a number of similar studies and replications (reviewed in Garety and Hemsley, 1994; 1999) that suggested that delusional patients make decisions in probabilistic reasoning experiments based on less data than do others (the 'data-gathering bias'), but are also more likely to abandon hypotheses to form new ones on the basis of less evidence.

Nevertheless, recent studies have suggested that this bias is not as straightforward as it first seemed and there may not be a clear link with delusions per se. Although a study by Garety et al. (2005) did find a link with delusions, other studies by Mortimer et al. (1996) and Menon et al. (2002) reported that this bias was shown equally in deluded and non-deluded patients diagnosed with schizophrenia, and studies by Peters et al (1999b) and Peters and Garety (in press) reported the bias in remitted delusional patients, suggesting that it may be a trait rather than a state factor. It is now looking increasingly likely that this bias is related to propensity to delusional ideation or magical thinking, rather than frank psychosis. Indeed, a study on non-clinical participants found that those high in delusional ideation were more likely to show such a bias (Linney et al., 1998), and a recent well-controlled clinical study showed that

although non-deluded patients with schizophrenia also displayed the bias, it was more pronounced in delusional patients (Moritz and Woodward, 2005).

Some studies have found no difference between deluded patients and other psychiatric patients (Fear and Healey, 1997; Young and Bentall, 1997; Peters and Garety, in press), leading Peters and Garety (in press) to suggest that this bias could stem from a lack of confidence and / or difficulty with decision making related to underlying depression and anxiety, rather than to any specific diagnosis.

One further problem is with the interpretation of these results, which are difficult to reconcile with the current conception of delusions. The data-gathering bias seems consistent with the traditional view of delusions as being based on magical thinking or apophenia (section 6.3), but the fact that patients seem to abandon hypotheses on the basis of less data, seems inconsistent with the traditional view that delusions are fixed beliefs. One answer may be that delusions are not as fixed as they are traditionally portrayed (section 2.4.1), although the related concept of poor belief flexibility has been supported by some experimental evidence (Freeman et al., 2004; Hurn et al., 2002) and has been linked to jumping-to-conclusions biases in a recent study (Garety et al., 2005).

It is notable that a pattern of results similar to the data-gathering and jumping-to-conclusions biases was found by Burgess and Shallice (1996) in (presumably) non-delusional patients with acquired frontal lobe injury who showed a greater tendency to guess and were more likely to abandon a correct rule once it was attained on the Brixton test. In light of the link between the diagnosis of schizophrenia and the data-gathering biases, rather than delusions per se (Mortimer et al., 1996; Moritz and Woodward, 2005), it is perhaps likely, that this bias



reflects the performance of the executive system. Indeed, executive function has been found to be consistently impaired in people diagnosed with schizophrenia (Velligan and Bow-Thomas, 1999), and attenuated in those high in schizotypy (Dinn et al., 2002), suggesting a plausible (albeit untested) hypothesis.

### **3.4.5 Institute of Psychiatry model section summary**

The IoP model is firmly grounded in clinical practice and is not framed within a model of normal belief formation, nor can it be easily adapted into one. Its main strength, however, is that it uses well specified cognitive models to account for each of the components, including the often neglected aspect of ‘anomalous experience’. It, like the Davies et al. (2001) model (section 3.2.3.1), makes a distinction between factors that explain why a delusion is formed and those that explain why it is not rejected.

Impressively, it also makes the distinction, highlighted in the work of Langdon and Coltheart (2000), between factors that explain why a delusion is formed and those that explain why it has a particular content, proposing that the direct role of emotion explains belief content. In spite of their claims, however, it does not avoid a ‘defence’ component, suggesting that, as in arguments presented in recent revisions of neuropsychological models (McKay et al., 2005; section 3.2.3.2), rejecting this component may not be wise, despite problems with theories that cite this process as a central pillar of delusion formation (Bentall et al., 2001; section 3.3.2).

Although the IoP model gives mechanisms for anomalous experience, again it is notable that the phenomenology of anomalous experience is barely explored, and little empirical evidence is given for an empirical link between anomalous experience and other factors. One further

weakness is that the theories are presented as a purely cognitive account, with little reference to neuropsychological evidence that could provide useful parameters and limits for this theory.

### **3.5 Metacognitive models of delusion formation**

Metacognitive models argue, in essence, that delusions arise from a dysfunction in the self-monitoring of internal mental phenomena. Two main models are associated with this tradition, the first, most associated with Chris Frith and Sarah-Jayne Blakemore, is based on a cybernetic model of action control and monitoring, the second is strongly related to the 'reality discrimination' and 'source monitoring' literature, the latter of which largely derives from the work of Marcia Johnson.

#### **3.5.1 Early Frith 'metarepresentation' model**

Frith (1992) argued that impaired metarepresentation (the ability to reflect on, monitor or represent other mental states) is the key deficit in schizophrenia, leading to disorders of willed action (causing negative symptoms), disorders in monitoring the intentions of others (causing paranoid delusions, delusions of reference and formal thought disorder) and disorders of self-monitoring (delusions of alien control, passivity symptoms and auditory hallucinations).

When re-described as specific cognitive processes, 'theory of mind' deficits are proposed as the basis for paranoid delusions and delusions of reference, and a deficit in the self-monitoring of intention underlies delusions of alien control and passivity symptoms (such as thought insertion and thought echo).

### *3.5.1.1 Delusions of persecution, reference and the role of theory of mind impairments*

Theory of mind is typically defined as the ability to represent one's own and others' mental states. Frith (1992) argues that an impairment in the purported theory of mind module may lead to the pathological misrepresentation of others' intentions, leading to faulty inferences and ultimately to delusions of persecution or reference.

Frith predicts that patients with these delusions should show a relative impairment in theory of mind in comparison to healthy controls, and not a total deficit, as holding a belief about the intentions of others, albeit a delusional one, requires some intact function. In contrast, delusions of alien control should not be associated with theory of mind dysfunction, although it is predicted that affected patients will still show a metarepresentation impairment in the area of action monitoring.

The evidence for impaired theory of mind is equivocal, however. Early studies did find impairments related to persecutory delusions with vignettes involving 'hinting' by the characters, false belief stories and mental state jokes (Corcoran et al., 1995; Corcoran et al., 1997; Frith and Corcoran, 1996). Studies examining the ability to infer beliefs and intentions of another speaker in a conversation have found both positive (Tenyi et al., 2002) and negative results (McCabe et al., 2004), whereas other studies using false belief stories and picture sequencing tasks have not found a clear relationship with persecutory delusions (Doody et al., 1998; Drury et al., 1998; Sarfati et al., 1997).

Langdon and Coltheart (1999) found an association between poor theory of mind and high schizotypy, but contended that this shows evidence against the Frith hypothesis, as all participants were from a non-clinical population; although it could be argued that they simply

showed a correlation with attenuated delusional phenomena, as is common practice in the psychosis continuum literature (section 2.5.3).

In contrast to the deficit models, Abu-Akel (1999) has argued that delusions may arise from “hyper theory of mind” function, where affected persons over-attribute intentions and mental states to others. It is interesting to note that many of the experimental tests of theory of mind rely on tasks that are scored for correct attribution of mental states, making it virtually impossible to infer why these failures are occurring, and whether they are due to up- or down-regulation of the relevant cognitive abilities. One of the sole clues is given by Blakemore et al. (2003), who found that patients with persecutory delusions were more likely to attribute intentionality to the movement of shapes, suggesting a degree of over-attribution rather than under-attribution.

The ambiguous evidence leaves a theory of mind account as a poor candidate for an adequate theory, even of specific (namely, persecutory), delusions. A further drawback is its lack of ability to account for why a belief might be persecutory, distressing, or even a delusion at all, given that a break-down in understanding others states of minds could simply lead to non-psychotic confusion.

#### *3.5.1.2 Delusions of alien control, passivity symptoms and dysfunctional self-monitoring*

Frith (1992) argued that delusions of alien control, and phenomena usually classified as delusions such as thought insertion, withdrawal and broadcasting, are better understood as abnormal experiences. These are explained in terms of an action-monitoring system that involves two stages:

I am essentially describing two steps in a central monitoring system. First, the relationship between actions and external events are monitored in order to distinguish between events caused by our own actions and by external agencies. This enables us to know about the causes of events. Second, intentions are monitored in order to distinguish between our own goals and plans (willed actions) and actions that are in response to external events (stimulus-driven actions). (p81)

Frith considers thoughts to be, for all intents and purposes, actions, and, therefore, subject to the same explanation. It is proposed that thoughts and willed-actions are recognised as such by being accompanied by a feeling of effort or intendedness, mediated by “corollary discharge” or “re-afferance copy”, which provides a label by which they can be recognised as self-generated. In this account, thoughts and actions are willed phenomena, but are not recognised as such, producing the conscious experience of their being caused by external agencies.

Evidence that abnormal self-monitoring is present in people with passivity symptoms is presented by Frith from action monitoring experiments (Frith and Done, 1989; Malenka et al., 1982) and has been supported by more recent studies using a paradigm where participants’ voices are fed back to them, either distorted or non-distorted in form. Notably, patients with delusions are more likely to attribute their own distorted voice to external agents (Cahill et al., 1996; Johns et al., 2001).

As a complete account of delusions, however, the Frith model has been criticised on several fronts for being either incoherent or incomplete. Perhaps the most damaging criticism has come from Thorton (2002), who noted that the model implies an infinite regress of ownership.

If actions are recognised as self-generated by an accompanying 'sense of effort', the model still leaves unexplained how the 'sense of effort' is recognised as self-generated. Thorton argues that the Frith model has simply re-arranged the problem without solving it in any significant sense. Gallagher (2004) made a similar criticism when he noted the concept of 'intention to think' also requires an infinite regress in any causal model, as the intention would also need intention, and so on.

Stephens and Graham (2000) observed that the Frith model does not explain why the subject of an inserted thought would take his thoughts or actions to be controlled by an outside agent, rather than supposing that it was simply unintended or stimulus-driven. Furthermore, they argue that thought insertion is poorly accounted for, as it involves more than the experience of being controlled by another: it involves the impression that a thought occurring in a person's own stream of consciousness is actually someone else's thinking.

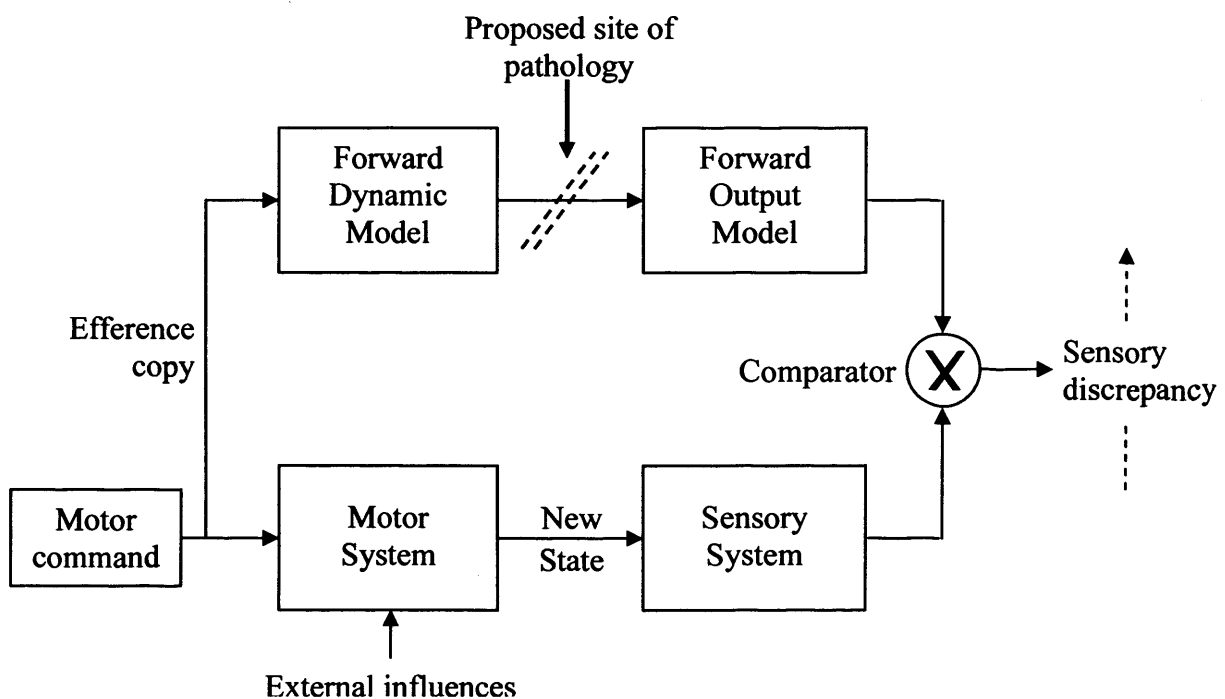
The model could also be taken to task for treating thoughts and actions as analogous, for which no evidence is provided by Frith (although a similar view is referenced; Feinberg, 1979), and, as with Frith's theory of mind approach to persecutory delusions, little explanation is given as to why any such experiences would result in delusional beliefs, as opposed to disordered perceptual experiences.

The model is explicitly neuropsychological, however, and as Gilleen and David (2005) have noted, it benefits from a wealth of data from investigations into the normal operation of action and theory of mind. It has been largely in response to these data, that the model has been revised and updated in a better specified form.

### 3.5.2 Blakemore and colleagues' awareness of action model

Blakemore and colleagues attempt to account for the same types of psychotic phenomena as the earlier Frith model, but explain them within a theory of action production that uses the better specified concept of a 'forward model'. This is thought to use information about intentions to distinguish between self-generated and externally caused actions, and allows for correction of actions based on comparisons between likely and desired outcomes, before the action is completed.

Blakemore and colleagues (Blakemore, 2003; Blakemore and Frith, 2003a; Blakemore and Frith, 2003b; Blakemore et al., 2000; Blakemore et al., 2002) base their explanation on the model of motor control from Miall et al. (1993; see figure 3.6), who suggested that motor commands are sent both to the motor system, to initiate action, and as an additional 'efference copy', to the forward model for comparison.



*figure 3.6 Blakemore and colleagues model of alien control and passivity delusions  
Diagram from Blakemore (2003)*

The forward model is thought to consist of two components; the 'forward dynamic model', that compares the predicted outcome of a motor command to the desired outcome; and the 'forward output model', that compares the predicted consequences of the motor command to the actual consequences of the movement.

Discrepancies from this comparison can be used to determine whether a movement is externally caused (large discrepancy), or self-generated (small or insignificant discrepancy). Blakemore and colleagues argue that delusions of passivity and alien control occur because there is an impairment in transferring information between the forward dynamic model to the forward output model, leading to a sensory discrepancy, and, therefore, the experience of movement being externally caused, despite it being originally initiated as a normal self-generated action.

One of the advantages of this model over Frith's previous version, is that it leads to a number of testable behavioural and neuropsychological predictions. The first is that patients with delusions of alien control and passivity should be less able to distinguish between self-generated and externally generated actions. In an ingenious study by Blakemore et al. (2000), based on the fact that self-tickling is generally less intense than tickling from others, patients with delusions of control and passivity, healthy controls and psychiatric controls, were asked to rate self-generated and externally-generated tactile sensations on various dimensions. Patients with delusions of control and passivity reported no significant difference between how tickly, intense and pleasant the sensations seemed. In contrast, self-generated sensations were rated as less intense by the other participants, suggesting the forward model was



detecting low discrepancy and so ‘damping down’ the intensity of self-produced movements in controls, but not passivity symptom patients.

The model is still subject to many of the same criticisms as the original Frith model, however. Although the problem of ‘infinite regress’, with self-generated actions being recognised by an accompanying ‘sense of effort’ is not applicable, it is still not clear how well the model accounts for thought insertion and related phenomena, as it still relies on thoughts being considered a type of action. In defending this position, Blakemore and Frith (2003b) are unable to provide any empirical evidence, and simply cite other authors as precedence for their views.

Moreover, a legitimate question is: ‘why do we need a forward model of thought at all?’ since the forward model has been hypothesised to solve problems that would not apply to internal thoughts. Even talking of comparing the desired, predicted and actual outcome of a thought sounds highly counterintuitive.

Regardless of the implausibility of the mechanism, the question could be solved empirically. Ford and Mathalon (2004) have suggested, based on findings from electrophysiological experiments, that internal thoughts produce corollary discharge and that this is disrupted in schizophrenia. This, perhaps, is evidence that thoughts may be explainable within the Blakemore model. Ford and Mathalon noted, however, that it is possible to interpret their data in alternative ways, so the case for a forward model for thought, and hence the applicability of the Blakemore model to thought insertion, remains weak.

Furthermore, the Blakemore model does not attempt to explain why such experiences result in delusional beliefs at all, rather than simply abnormal experiences. Blakemore et al. (2002) avoid the issue by simply stating that “In parallel, the patient’s belief system is faulty so that he interprets this abnormal sensation in an irrational way”.

### **3.5.3 Source monitoring, reality monitoring and other metacognitive theories**

Several other theorists have considered the ability to monitor mental states to be the key to understanding delusions. Although all maintain influences from their source disciplines, the main theme remains the same, namely that delusions and false beliefs arise from problems in distinguishing internally generated mental events from veridical experiences.

Currie and his collaborators (Currie, 2000; Currie and Jureidini, 2001; Currie and Ravenscroft, 2002; section 2.5.4) have argued that a potential point of failure might be in the monitoring of imaginative states. They suggested that cognitive biases or deficits could lead individuals to misidentify their imaginings as beliefs, and, therefore, accept imagined ideas as delusions. Currie’s theory has been criticised on conceptual grounds, however (Bayne and Pacherie, 2005; Bell, 2002; section 2.5.4), mainly on the basis that there is little *a priori* reason to suppose that imaginings could be misidentified as beliefs, as it is not clear that they share any cognitive similarities. Currie’s proposal could be significantly boosted by supporting evidence, but so far, no empirical work has been published directly testing the implications of his hypothesis.

A related cognitive account which has been tested, however, has been put forward by Johnson (1988) and Johnson and Raye (1998; 2000), who have suggested that delusions can be explained within the ‘source monitoring framework’ that seeks to explain how veridical

perceptions and self-generated phenomena (typically imagery) are distinguished in memory. Johnson's theory argues that this judgement is based on attributes of the item being retrieved, including, for example, sensory detail; embeddedness in spatial and temporal context; embeddedness in supporting memories and beliefs, and associated memories for the metacognitive operations involved in the producing the event or memory. Applying the framework specifically to delusions, Johnson and Raye (2000, p60) argue that:

Positive symptoms of schizophrenia, like the confabulations of frontal patients, can be viewed as a product of normal source monitoring processes applied to unusual input (for example, vivid perceptual or strong affective features from imagination) or dysfunctional processes applied to usual input (for example, lower criteria) or both.

Empirical studies have shown that source monitoring impairments are associated with the presence of delusions (Brébion et al., 1999; 2000; 2002; Keefe et al., 1999; 2002; Moritz et al., 2005), although it must be noted that these studies typically lump together delusions and hallucinations as 'reality distortion', or have found similar effects for each. Nonetheless, this does suggest that memory distortions may be an important factor in delusion formation.

Theoretically, Johnson echoes two-factor theories of delusion formation (section 3.2.3) which allow for the possibility of delusions arising from either 'unusual input' or cognitive distortions. Interestingly, Johnson has argued that these processes may be based in 'two reflective subsystems' that monitor each other (Johnson, 1991), each of which, she argues, may be disproportionately represented in the left and right hemispheres (Johnson, 1997). She further argues that disruption to communication between these systems could lead to

significant memory distortions, thereby proposing a similar account to McKay et al (2005; section 3.2.3.2).

#### **3.5.4 Metacognitive models of delusions section summary**

Despite the theoretical objections to metacognitive models, mainly aimed at the Frith and Blakemore models, their main disadvantage is that they work better as explanations of anomalous experience rather than delusions per se, owing to the fact that they almost entirely avoid the issue of belief formation and why such an experience would end up being interpreted in a delusional manner (the purported 'second factor'). Gold and Hohwy (2000, p160) make a similar point when they suggest that the delusions that the Frith and Blakemore models choose to explain "are best explained as disorders of experience rather than disorders of belief, desire or reasoning".

The fact that these models have almost nothing to say about normal belief formation, and only a limited amount to say about delusions in general, suggests they are best thought of as specific explanations of certain psychotic experiences. Their utility, therefore, needs to rest on their continued survival through empirical testing.

The more general metacognitive accounts do seem more widely applicable, although, again, decisive empirical evidence is still lacking. The fact that, in Johnson's account at least, there is a strong equivalence between this approach and other, independently developed, theories of delusion formation, seems to provide some conceptual support.

One conceptual quirk of Johnson's approach, however, is that it is largely an extrapolation of work on confabulation, and often makes the equivalence between confabulation and delusion

with little supporting evidence. Johnson and Raye (2000, p36) argued that the main difference between the phenomena is aetiology, and that “as evidence accumulates that patients with psychotic delusions may have brain pathology in some or related brain areas as do confabulating organic brain-damaged patients or abnormalities in one of more neurotransmitter systems... an etiology-based distinction between delusions and confabulations becomes questionable.” Hirstein’s (2005, p18) analysis of confabulation seems to do nothing except muddy the waters when he makes the distinction by stating that delusions are “false or ill-grounded beliefs”, whereas confabulations are “false or ill-grounded claims”. None of this seems based on an understanding of the possible structure of belief, or a principled distinction between belief and memory, making it difficult to see how valid such comparisons are likely to be.

Either way, metacognitive accounts that seek to explain either delusions or confabulation do not provide, or even imply, a model of normal belief formation. The fact that they are grounded in neuropsychological models (albeit, of other processes, such as theory of mind, motor control or memory) could allow conclusions to be drawn as to areas of impairment that commonly result in pathological beliefs. From the analysis above, however, these models are more likely to explain anomalous experience, rather than give answers to the more troubling questions of why such an experience is interpreted in a delusional manner, and why an initially adopted belief is not rejected over time.

### **3.6 Hemispheric asymmetry model of delusions**

Functional asymmetries of the cortical hemispheres have been proposed as an explanation for delusions, with dysfunction to the right hemisphere being particularly implicated. This has been supported by evidence that other phenomena on the psychosis continuum, such as

unusual experiences and paranormal beliefs, are associated with relatively greater activation in the right than left hemisphere.

### **3.6.1 Clinical and non-clinical studies of hemispheric asymmetry**

It has long been known that right hemisphere damage is associated with a range of pathologies of belief. A review of the anosognosia literature by Pia et al. (2004; using literature going back as far as 1938) suggested that most cases are associated with right-hemisphere or bilateral damage. Delusional misidentification syndromes have also been frequently associated with right hemisphere dysfunction (Bourget and Whitehurst, 2004; Edelstyn and Oyebode, 1999; Ellis, 1994) as have 'content specific' or monodelusional disorders in general (Malloy and Richardson, 1994).

Tim Crow has gone as far as suggesting that schizophrenia may be the evolutionary price humans have paid for the development of lateralised language skills (reviews in Berlim et al., 2003; Crow, 1997), on the basis of evidence that people diagnosed with schizophrenia typically show a reduction in the normal pattern of cerebral dominance for language (although this hypothesis is controversial and has been criticised for not taking into account the diversity of the symptoms classified as 'schizophrenic'; Brune, 2004).

The evidence for hemispheric asymmetry is most clear for non- or sub-clinical manifestations of the psychosis continuum. As can be seen from table 3.2, studies using a number of psychosis continuum scales, and measures of hemispheric asymmetry, have shown a relatively greater activation in the right than the left hemisphere.

<i>Continuum Type</i>	<i>Study</i>
Paranormal beliefs	<ul style="list-style-type: none"> <li>▪ EEG (Pizzagalli et al., 2000)</li> <li>▪ Lateralised priming (Pizzagalli et al., 2001)</li> </ul>
Magical ideation	<ul style="list-style-type: none"> <li>▪ Lexical decision (Leonhard and Brugger, 1998)</li> <li>▪ Odour detection (Mohr et al., 2001)</li> <li>▪ Line bisection (Taylor et al., 2002)</li> </ul>
Schizotypy	<ul style="list-style-type: none"> <li>▪ Lexical decision (Kravetz et al., 1998)</li> <li>▪ Dichotic listening (Weinstein and Graves, 2002)</li> </ul>
'Mystical' experience / sensed presence	<ul style="list-style-type: none"> <li>▪ EEG (Makerec and Persinger, 1985)</li> <li>▪ Temporal lobe signs (Persinger and Makerec, 1987)</li> <li>▪ Applied complex magnetic field (Richards et al., 1993)</li> </ul>

*table 3.2 Studies showing bias for right-hemisphere activation for psychosis-like phenomena*

Leonhard and Brugger (1998) have argued that this indicates an over-reliance on right hemisphere processes, whose coarse rather than focussed semantic processing may favour the emergence of 'loose' and 'uncommon' associations. Such associations may lead to anomalous beliefs and experiences, but also to higher levels of creative thinking (Schuldberg et al., 1988; Weinstein and Graves, 2002), and it is argued, in their extreme form, frank delusional belief (Leonhard and Brugger, 1998).

In light of the continuum model of psychosis (section 2.5.3), it might be expected, therefore, that the pattern of hemispheric asymmetry would also be on a continuum, with the most extreme forms of right hemisphere bias being found in delusional patients.

This pattern does not seem to hold, however, as reviews of the functional imaging literature suggest. A series of positron emission tomography (PET) and single photon emission computed tomography (SPECT) studies have linked increased activation in left-sided areas (typically, left frontal or temporal) to 'reality distortion symptoms', including both delusions

and hallucinations (Ebmeier et al., 1993; Kaplan et al., 1993; Liddle et al., 1992; Sabri et al., 1997). A review of the wider functional imaging literature on schizophrenia suggests that left-sided dysfunction is most prevalent (Gur and Chin, 1999) and a study of volumetric changes in psychosis, both longitudinally and in cross-section, suggested differences in grey matter volume could occur in both hemispheres (Pantelis et al., 2004). The only functional magnetic resonance imaging (fMRI) study that has specifically concerned itself with the cognitive neuroscience of persecutory delusions (Blackwood et al., 2004) found increases in activation on both sides of the hemispheric divide.

It is possible that the contradiction between the clinical and non-clinical studies might be attributed to methodology. The non-clinical studies have typically used fairly crude behavioural measures that allow the researchers to infer neuropsychological function, whereas the clinical studies have used relatively sophisticated neuroimaging methods that are much more likely to pick up fine grained distinctions in activation, rather than whole hemisphere averages. Even on the most generous reading, however, the results from the clinical studies (that typically suggest increased left hemisphere activation) and the results from non-clinical studies (that typically suggest the reverse) do not seem to support a simple hemispheric asymmetry account.

### **3.6.2 Hemispheric asymmetry section summary**

It is clear that as a straightforward account of delusions, a simple hemispheric asymmetry model is inadequate. Most relevant to this, somewhat basic, model of delusion formation, is the more detailed model put forward by McKay et al. (2005; section 3.2.3.2), who argue that the right hemisphere is a discrepancy detector, that when damaged, is likely to lead to the adoption of abnormal beliefs.



The model of McKay et al. has been developed mainly with reference to cases of delusion arising after brain injury, where it is relatively clear which hemisphere is dysfunctional, as there is usually physical neuropathology to be observed. It is much harder to relate the findings from people with psychosis who have not suffered brain injury, and even more difficult to integrate the findings from those, supposedly on the psychosis continuum, who do not have clinically significant beliefs or experiences.

The evidence from *some* of the clinical neuroimaging studies could be interpreted as providing some support for the McKay et al. model. One problem, however, is that it is not clear whether increased blood flow suggests impairment, compensation, or adaptation. This problem dogs the interpretation of data from the non-clinical population as well, as these opposite findings could also be interpreted in support of the McKay et al. model, again, depending on how one interprets the pattern of asymmetry (in terms of impairment, compensation, or adaptation). It is not possible, however, easily to integrate both sets of findings, as McKay et al. leave no room for a 'threshold of pathology', that might allow for a change in the balance of hemispheric function.

One possibility is that the McKay et al. model specifically addresses delusions arising from brain injury, which, considering the diverse presentation and aetiology of delusions (Gilleen and David, 2005; section 2.4), is a more likely state of affairs. Nevertheless, what is most clear from this discussion, is that the cognitive neuroscience of delusions needs to be better specified, particularly with regard to the non-clinical aspects of the psychosis continuum. It is also clear that this approach is not a model of belief formation in itself, but understanding how neuropsychological mechanisms on the sub-hemisphere scale are involved in some of the

implicated mechanisms, such as anomalous experience and magical thinking, may help integrate the clinical and non-clinical findings in this area.

### **3.7 Neuropathological and neurobiological accounts of delusion formation**

Stemming from the medical tradition, physical aspects of the brain have often been quantified in an attempt to make a functional link with the presence of delusions. Two main approaches have emerged: neuropathological investigations into structural and functional changes in the gross anatomy of the brain; and neurobiological studies into associations with neurotransmitter function and cellular transmission.

The majority of such studies have not tackled delusions specifically and are typically based on patient groups classified by higher-level diagnostic categories such as schizophrenia or psychosis. Although some more recent studies have attempted to specifically look at the neuroscience of delusional phenomena, many of the conclusions remain speculative when drawn from other, less focused studies.

#### **3.7.1 Delusions and the pathophysiology of gross anatomy**

Findings from structural imaging studies of schizophrenia or psychosis in general suggest that several structures may show abnormal features. In particular, ventricular enlargement, temporal lobe volume reduction (particularly in the medial temporal lobe areas and the superior temporal gyrus); and structural abnormalities of the corpus callosum, and prefrontal cortex white and grey matter, have been identified as reliable findings in reviews of the structural imaging literature (McCarley et al., 1999; Shenton et al. 2001; Wolkin and Rusinek, 2002). A recent review of diffusion tensor imaging studies in schizophrenia (Kubicki et al., in press) indicated that abnormalities in the white matter tracts connecting temporal and frontal

areas were the most common findings, again suggesting that these areas may be important in the pathogenesis of psychosis.

Reviews of the literature on psychosis after brain-injury or neurological disease have also implicated temporal and frontal regions, including when associated with cerebrovascular accident (Starkstein et al., 1992), tumour (Lisanby et al., 1998) and traumatic brain injury (Fujii and Ahmed, 2002).

Studies that have examined 'reality distortion' symptoms in people diagnosed with schizophrenia have found similar results. PET studies have found increased activation in the lateral prefrontal cortex, ventral striatal area, superior temporal gyrus and parahippocampal areas (Liddle et al., 1992), or in the case of a study by Kaplan et al. (1993), increased left temporal activity. In contrast, a SPECT study conducted by Ebmeier et al. (1993) found decreased activation in left temporal area, although an increase in activation in the left striatal area.

Only recently have studies looked specifically at delusions, however. Also using SPECT, and also finding a result inconsistent with most other psychosis studies, Sabri et al. (1997) found decreased activation in left frontal and medial temporal areas related to the delusions score on the Positive and Negative Syndrome Scale (Kay et al., 1987).

An fMRI study by Blackwood et al. (2004) examined self-attribution and 'threat-to-self' processing in a group of inpatients with persecutory delusions and a group of healthy controls. Although the 'threat-to-self' experiment found no significant differences between the deluded and non-deluded groups (in either the behavioural or imaging measures), in determining self-

relevance, the deluded subjects showed significantly less activation in the rostral-ventral aspect of the anterior cingulate, and significantly greater activation in the posterior cingulate gyrus.

A study by Spence et al. (1997) used PET, and focused on patients with passivity delusions, and compared them with healthy controls and deluded patients without passivity symptoms. Increased activation in the right inferior parietal lobule and cingulate gyrus was associated with the presence of passivity delusions in a movement task, independent of diagnosis, suggesting a dysfunction in motor control of movements in extra-personal space and top-down executive control.

A further study focusing on passivity delusions used volumetric analyses, and found that the right inferior parietal lobule and the left dorsolateral and medial prefrontal areas were reduced in patients with passivity delusions (Maruff et al., 2005). This again suggests a dysfunction of executive and motor control, although it is interesting to note that, while the PET study of Spence et al. (1997) found increased activation in this area, a volumetric approach found decreased size – a finding difficult to interpret when a clear understanding of how pathology relates to a combination of functional and structural changes is still somewhat lacking.

Nevertheless, this unclear relationship has also been found in studies of the temporal lobes and their relation to ‘reality distortion’ symptoms. Clinical studies have typically found reductions in left lateral temporal lobe volume in the schizophrenia spectrum disorders (Dickey et al., 1999; McDonald et al., 2004), whereas functional neuroimaging studies have often revealed increased regional cerebral blood flow in the left temporal areas, particularly

the superior temporal gyrus, in patients with ‘reality distortion’ symptoms (see Blackwood et al., 2001, for a review).

Interestingly, a recent volumetric study by Sumich et al. (2005), similarly focusing on ‘unreality symptoms’, found that the volume of two adjacent areas on the left temporal lobe were correlated in different directions. Severity of both hallucinations and delusions were negatively correlated with the volume of the left Heschl’s gyrus, whereas delusions alone were *positively* correlated with the volume of the left planum temporale. This indicates that the relevant functional networks in the brain need to be specified on quite a fine level, as well on the level of larger scale structures, such as those connected by large white matter tracts (as highlighted by the diffusion tensor imaging studies of Kubicki et al., in press).

In contrast, there is a surprising amount of evidence linking pathology in specific areas of the brain and delusions in dementia. The relevant studies are summarised in table 3.3 and show that frontal and temporal impairments are consistently linked to the presence of delusional phenomena, as has been found in previous studies on non-dementia psychosis and delusions.

Sultzer et al. (2003) and Shanks and Venneri (2004) have further noted that several studies (marked \* in the table) have shown that frontal pathology seems specifically associated with what Sultzer et al. (2003) called ‘factual’ delusions, defined as delusions which are “less emotionally charged” and “about current activities or fixed false beliefs regarding the environment”. This raises the possibility of linking not only the presence of delusions, but also specific belief contents, to a functional description of the brain.

<i>Reference</i>	<i>Type</i>	<i>Method</i>	<i>Results</i>	<i>Delusion details</i>
Starkstein et al. (1994)	AD	SPECT	↓ left and right temporal lobes	Various
Mentis et al. (1995)	AD	PET	↓ bilateral orbitofrontal and cingulate areas, left medial temporal. ↑ bilateral superior temporal and inferior parietal	Delusional misidentification
Hirono et al. (1998)	AD	PET	↓ left medial occipital ↑ left inferior temporal gyrus	Various
Staff et al. (1999) *	AD	SPET	↓ right frontal and limbic	Various
Venneri et al. (2000)	AD	SPECT	↓ right DLPFC	'Autobiographical'
Staff et al (2000) *	AD	SPECT	↓ right DLPFC	'Autobiographical'
Fukuhara et al. (2001)	AD	SPECT	↓ right middle posterior parietal	Theft
Breen et al. (2001)	FOD	CT / MRI / neuropsych testing	CT / MRI inconclusive. neuropsych testing showed 'right hemisphere dysfunction'	Mirror self-misidentification
Shanks and Venneri (2002)	AD	SPECT	↓ right fronto-parietal	Animistic delusions about soft toys
Geroldi et al. (2002)	AD	structural CT	↓ left frontal horn ↑ right temporal horns	Various
Sultzer et al. (2003) *	AD		↓ right superior dorsolateral and inferior frontal pole; ↑ lateral orbitofrontal Additional bivariate analysis: ↓ bilateral prefrontal and anterior cingulate ↑ left middle temporal gyrus	Various

↓ reduced activation or size; ↑ increased activation or size; DLPFC = dorsolateral prefrontal cortex; AD = Alzheimer's dementia; FOD = focal onset dementia; \* Studies showed particular relationship between 'factual' delusions ("about current activities or fixed false beliefs regarding the environment") and frontal impairments.

*table 3.3 Association between circumscribed brain regions and delusions in dementia studies*

Although this evidence is far from being integrated into a useful or comprehensive pathophysiological account, it does suggest that it may be possible to refine theories so both the presence and specific content of delusions can be linked to impairments to specific brain areas or neural networks.

The fact that the conclusions drawn from this work are only on the scale of large brain areas (often implicating whole lobes), further highlights the urgent need for additional work in this

area, particularly in light of the necessity to refine the somewhat oversimplified hemispheric asymmetry approach (section 3.6).

### **3.7.2 Neurobiological accounts of delusion formation**

The 'dopamine hypothesis of psychosis' was an early and popular explanation for the presence of delusions, largely based on findings that early antipsychotic medications blocked dopamine receptors, and that amphetamine abuse could lead to schizophrenia-like psychoses (Healy, 2002).

This theory is now thought to be incomplete as an adequate account of psychosis, as subsequent studies have shown significant counter-evidence, such as unreliable correlations between the occupancy of dopamine receptors and the clinical benefit of antipsychotics; and unreliable correlations between dopamine receptor density and psychotic symptoms in untreated patients (reviewed in Jones and Pilowsky, 2002).

Similarly, evidence that drugs such as ketamine and phencyclidine (PCP), both N-methyl D-aspartate antagonist (NMDA) antagonists, cause psychosis which is not reversed by antipsychotics (Krystal et al., 1999) and that there is a tendency for low densities of gamma-aminobutyric acid (GABA) axon terminals in the prefrontal cortex of the post-mortem brains of people diagnosed with schizophrenia (Woo et al., 1998), suggests that dopamine is not necessarily the sole neurobiological factor.

It is important to note that this evidence still suggests a significant role for dopamine in the neurobiology of psychosis. Nevertheless, work in this area is in a similar state to research

concerning gross anatomy (section 3.7.1), in that only recently has work focused on delusions per se, rather than on the more diffuse concepts of schizophrenia or psychosis.

Kriekhaus et al. (1992) argue that delusions are caused by information processing errors during the declarative memory consolidation process. This, they claim, is particularly due to excess dopamine, causing modulatory neurons in the hippocampus that project to the parietal-temporal-occipital association cortex, to become hyperactive. The proposed upshot of this process is that inappropriate connections become strengthened, leading to false declarative memories and, consequently, delusional beliefs.

Later, both Spitzer (1995) and Kapur (2003) attempted to make a similar connection between the neurobiological and cognitive effects of dopamine, and the phenomenology of delusions (Spitzer argued for a computational account, discussed in section 3.8.2). Kapur argued that dopamine mediates the conversion of the neural representation of an external stimulus from neutral information to an attractive or aversive stimuli, and proposed that:

in psychosis there is a dysregulated dopamine transmission that leads to stimulus-independent release of dopamine. This neurochemical aberration usurps the normal process of contextually driven salience attribution and leads to *aberrant assignment of salience to external objects and internal representations*. Thus, dopamine, which under normal conditions is a mediator of contextually relevant saliences, in the psychotic state becomes a creator of saliences, albeit aberrant ones. [Italics the original].

Nevertheless, it is perhaps worth sounding a note of caution, in that Kriekhaus et al. and Kapur both present speculative theories that have not been directly tested, and still require



considerable conceptual leaps to link their proposed mechanisms with the presence of delusions.

A recent study by Myin-Germneys et al. (2005) suggests that these approaches may have some ecological validity, however. The researchers used an experience sampling method to randomly request of either controls, or first-degree relatives of patients with psychosis, to rate current stress and psychosis-like experience. Both groups of participants had previously been assessed for dopamine reactivity. Psychotic experiences in response to stress were much more prevalent in the first degree relatives who also had much higher dopamine reactivity, suggesting that this reactivity may mediate the commonly proposed stress-vulnerability interaction.

Other evidence is still thin on the ground, however. Initially promising results suggesting that prevalence of the DRD4 gene, which codes for dopamine D<sup>4</sup> receptor, was linked to delusional disorder (Serretti et al., 2001), was not replicated in later studies (Serretti et al., 2004). Recent studies of the gene that codes for catechol-O-methyltransferase (COMT; an enzyme involved in the catabolism of the catecholamine neurotransmitters, including dopamine) has provided some promising results. For example, inheriting two valine bearing COMT alleles has being shown to interact with cannabis use to greatly enhance the risk of developing psychosis (Caspi et al., 2005).

Studies using 'latent inhibition' or 'Kamin blocking' paradigms, have suggested that poor attentional filtering may be one plausible mechanism that links dopamine function to psychosis or schizotypy (Gray et al., 2002; Gray and Snowden, in press), although methodological issues have made past results difficult to interpret (Fuller et al., 2000; Lubow,

1997) and there is no specific link to delusion formation, rather than a general deficit linked to both a clinical and non-clinical tendency for unusual thoughts and experiences.

Despite their promising nature, many of the neurobiological findings have the disadvantage of being either speculative, unreplicated or without a well-tested mechanism which links them specifically to delusion formation.

### **3.7.3 Neuropathology and neurobiology section summary**

Although research in this area is still in development, some significant drawbacks are obvious. The first is that the majority of the work has been done on psychosis or schizophrenia, rather than on delusions specifically, or has been done with reference to dementia, particularly Alzheimer's disease. Very little work has focused on explaining delusions within the general framework of a cognitive neuroscience of belief, and it is unclear how confidently inferences can be drawn from the findings that have been reported, or how specific they are to the conditions (such as schizophrenia or Alzheimer's) in which they occur. The contradictory findings reported by some of the studies may well be explained by the heterogeneity of the paradigms and participants used in these studies, although this lack of consistency also makes it difficult to determine emergent themes from a background of sometimes confusing methodology.

A further caveat is the fact that the majority of the findings simply show correlations between symptoms and brain areas, making causal inferences difficult to tease out. Combined with inconsistent patient samples, these correlations also become difficult to decipher.

Some general themes do emerge, however. The relatively consistent involvement of frontal and temporal areas may suggest a disrupted interaction between the executive system and semantic memory or related conceptual knowledge. Perhaps suggesting the role of top-down modulation of attention and perception. Nevertheless, this hypothesis is speculative and a more detailed account will depend on knowledge of how functional disruptions contribute to relevant cognitive pathologies.

It is clear that additional work needs to be completed to determine causal, rather than correlational factors in delusion formation, on samples of participants well-controlled for potentially confounding factors (see Chapter 6).

### **3.8 Computational and connectionist accounts of delusion formation**

Apart from one early exception, computational models have attempted to model the presumed pathophysiology of neural structures, to see if these produce outputs that could then be interpreted as delusional beliefs. Needless to say, the models developed so far are metaphorical interpretations of the proposed pathologies, although as functional systems, they aim to provide a framework from which testable hypothesis can be drawn.

#### **3.8.1 Kenneth Colby's procedural model of paranoia**

In his book, 'Artificial Paranoia', Colby (1975) outlined a computational model of paranoia based on a flowchart understanding of the mind, charting mental function as a process of manipulating symbols, segments and sequences of natural language thinking. Procedures such as the 'self-scanner' would check self-generated 'speech' for topics related to currently held delusions and would increase the 'FEAR' variable if found, similar procedures would affect

the values of 'MISTRUST' and 'ANGER', supposedly simulating the levels of these emotions during social interaction.

The core of Colby's approach to delusion formation itself, like the 'early Bentall model' (section 3.3.2.1), is largely a restatement of Adler's (1914) theory of paranoia.<sup>2</sup>

The detection of potential humiliation in the simulation serves as an anticipatory warning not to actually execute the acknowledging procedure since it will result in the painful re-experiencing of self-condemnation and loss of self-esteem. To avoid this acceptance of the self as being wrong, the interpreter tries an alternative simulation of assigning wrongdoing to others. (Colby, 1975, p32)

Although capturing a popular mechanism for the explanation of persecutory delusions, one of the quirks of the model is that it was designed for natural language interaction, in a similar manner to the ELIZA programme (Weizenbaum, 1966). Colby suggests that this is intended to simulate the paranoid process in a psychiatric interview, and might be useful for giving trainee psychiatrists experience of simulated interaction with paranoid patients before they enter the clinic.

Although Heiser et al. (1979) reported that Colby's model passed the Turing Test (Turing, 1950) where a series of psychiatrists were unable to distinguish its responses from those of genuine paranoid patients, it is doubtful, particularly given the research discussed earlier in this chapter, that paranoia is best understood in purely linguistic or natural language terms.

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<sup>2</sup> As with Bentall, Colby makes no reference to Adler's work, and was presumably developed in a similarly independent manner.

Recent models have exclusively applied a connectionist or artificial neural network approach to understanding delusions, in an attempt to link an understanding of the proposed neural dysfunction to the impaired cognitive mechanisms and phenomenology.

### **3.8.2 Dopamine modulation models**

Cohen and Servan-Schreiber (1992) produced a feed-forward neural network aiming to simulate the neuromodulatory effects of dopamine in the prefrontal cortex on context processing in schizophrenia. Their model simulates the performance of a sample of patients on a the Stroop task, the continuous performance test and a lexical disambiguation task. Although not specifically relating their model to any particular symptom, Cohen and Servan-Schreiber did produce a principled computational model linking the neurobiology of dopamine to cognition.

Spitzer (1995) did not produce an explicit computational model himself, but his theoretical analysis went further in exploring this link. Although Spitzer's work is not mentioned by Kapur (2003; section 3.7.2), the main thrust of the argument is similar – that dopamine modulates the salience of stimuli on the basis of neurobiological studies that suggest it modulates signal-to-noise ratio. Of particular relevance to Spitzer, is that this effect may be particularly important for modulating other, fast-acting neurons, potentially fast enough to support moment-to-moment thought, whose firing is largely mediated by GABA receptors. Spitzer notes that a type of artificial neural network, called a self-organising feature map, has been shown to be sufficient to create an orderly representation of any coherent input (Kohonen, 1989) and, therefore, may be a candidate for modelling the high-level processes that produce delusional beliefs, while being also able to model the influence of dopamine at the neurobiological level.

Interestingly, Spitzer makes the distinction between acute delusions and chronic delusions, suggesting that acute delusions may be due to short-term dopamine dysfunction, whereas chronic delusions may be due to the persistence of this effect, leading to neuroplastic changes in the cortex. While Spitzer's model was highly speculative at the time, the main arguments have been very well supported by recent reviews of the area, albeit with reference to schizophrenia in general, rather than to delusions specifically (Winterer and Weinberger, 2004).

Although these models are admittedly light on cognitive details, the key role of dopamine neuromodulation and the idea that delusions could be supported by a transition from one cortical state to another, have been influential in later computational models (section 3.8.4).

### **3.8.3 Neural pruning models**

Hoffman and colleagues (Hoffman and Dobscha, 1989; Hoffman and McGlashan, 1993, 2001; Siekmeier and Hoffman, 2002) have proposed that the core deficit in schizophrenia which causes delusions and hallucinations is the excessive pruning of local synaptic connections in the prefrontal cortex (e.g. Glantz and Lewis, 2000). They have modelled this process using fully interconnected Hopfield networks (Hopfield, 1982), known for their uses as content-addressable memory systems. When the local connections of these models are over-pruned after training, the model tends to produce what Hoffman calls 'parasitic foci', or fixed persistent output patterns, not related to stored memories, that interfere with the normal retrieval of information.

Hoffman and McGlashan (1993) claim that the presence of such patterns in cortical association areas could interfere with declarative memory, leading to false memories and, therefore, delusions. A recent study suggested that the model could be applied to enhanced priming of semantic memory (Hoffman and McGlashan, 2001), although the authors were cautious not to relate the findings to any specific positive symptom. It is plausible, however, that this could also contribute to delusion formation in light of the representation of semantic memory in the temporal lobes, and the implication of these areas in pathologies of belief (section 3.7.1).

Also using a Hopfield network, Ruppin et al. (1996) explicitly included temporal lobe dysfunction in their model, by simulating the degeneration of temporal lobe projections to the prefrontal cortex. In this case, by reducing the strength of external inputs to the nodes designated as representing prefrontal neurons. Furthermore, they modelled 'reactive frontal sprouting', based on Stevens' (1992) model of the pathogenesis of schizophrenia, where locally connected neurons in the prefrontal cortex have their connections strengthened. As a result of the simulated pathology, the Ruppin et al. model spontaneously retrieved information stored in the network, independent of a 'retrieval cue', which, they suggest, may be an analogue for hallucinatory and delusional phenomena.

At first sight, the Hoffman and Ruppin models seems to be based on contradictory theories concerning synaptic pathology, in that one suggests excessive local pruning, the other excessive local connectivity. Recent neurodevelopmental evidence suggests that neuronal migration during the second trimester may be incomplete in people later diagnosed with schizophrenia, in that neurons do not fully connect to the outer layers of the brain, yet begin to make connections to their neighbours nonetheless (McGlashan and Hoffman, 2000; de Haan

and Bakker, 2004). Connections from these misplaced neurons are thought to be sub-optimal, evidence which seems to favour the Hoffman account. It must be noted, however, that pathological behaviour was only noted in the Ruppin model when both degeneration of temporal lobe projections and reactive frontal sprouting was simulated, making an exact comparison between the two models difficult.

Chen (1994; 1995) has also proposed a Hopfield network model, inspired by similar ideas to Hoffman, in that delusions are conceptualised as 'spurious attractors' (analogous to Hoffman's 'parasitic foci'). Chen includes several other factors into his model, however, including the neuromodulatory function of dopamine, simulated by reducing the network noise parameter, and hippocampal dysfunction, which he simulates by introducing highly overlapping inputs, on the basis that the function of the hippocampus is to 'orthogonalise' information during memory encoding. One novel aspect is that Chen also attempts to include the presumed social, as well biological, factors in delusion formation. This he implements by 'overloading' the network with information, in an attempt to simulate the high cognitive demands induced by stress, as per the stress-vulnerability framework.

#### **3.8.4 Hyperassociation models**

The model of Vinogradov et al. (1992), although not explicitly implemented, is based on an associative model of memory, where spreading activation supports memory access (essentially, a model of semantic priming, inspired by Collins and Loftus, 1975). It further proposes a continuum of delusional phenomena that can be viewed as three overlapping states: normal functioning, the initial paranoid state and formation of a crystallised delusional system.



One innovation in the model is that it proposes that a linear change in the model's parameters (presumed to reflect some underlying neural process) can result in non-linear changes in spread of activation, leading to the 'hyperassociation' of memories. Vinogradov et al. propose that the different stages of delusional phenomena represent phase transitions in the activation of semantic associations. One drawback in comparison to other computational models, however, is that it is particularly abstracted from biological function and makes little attempt to include pertinent neurobiological simulations.

### **3.8.5 Computational and connectionist accounts section summary**

Computational models of delusion formation have become increasingly sophisticated, with Chen's (1994; 1995) seeming to cover the most theoretical ground. Despite the fact that they hold a great deal promise in terms of fusing neurobiological and cognitive accounts, their major shortcoming is undoubtedly the highly metaphorical way in which they represent the phenomena of delusions.

None of the connectionist models seems to be able to make any principled distinction between the simulation of a general information processing dysfunction (more analogous to memory impairment in many cases) and the simulation of delusion formation. The proposed equivalence of these two concepts draws its validity from the supposedly accurate simulation of the neurobiological findings in psychosis, despite the fact that, as discussed in section 3.7.2, the evidence for how these specifically relate to delusions is still rather sparse. Notably, Hoffman and McGlashan (2001) and Ruppin et al. (1996) suggest that their models simulate both delusions and hallucinations with equal validity, suggesting that these models are far from being an adequate account of how beliefs become pathological.

They do include some important ideas, however. The concept that psychotic phenomena may occur in coherent clusters, perhaps mediated by ‘phase transitions’ (Vinogradov et al., 1992) or due to long-term changes in cortical plasticity (Spitzer, 1995) could be thought of as consistent with the continuum model of psychosis where clinically relevant ‘illness behaviour’ is thought more likely to occur above a certain threshold of psychosis-like experience (Johns and van Os, 2001). Similarly, recent factor analyses of hallucinatory phenomena (Serper et al., 2005; Singh et al., 2003) and a recent study of pre-pulse inhibition in people with high schizotypy scores (Abel et al., 2004), suggest a similar form of threshold or transition may take place and that the psychosis continuum may not represent the function of a single underlying factor.

### **3.9 Chapter conclusion and research proposal**

In light of the theories discussed in both this, and the previous chapters, several areas can be highlighted where further empirical work is needed.

The first is the general point that, in line with the criteria of Bentall et al. (2001; outlined in the Preface), the problem of delusions needs to be tackled on multiple levels. There is a significant lack of knowledge on the phenomenological, psychological and neuropsychological levels of explanation. This thesis, therefore, takes a comprehensive view of the problem, and has applied a multi-level approach. The following sections note the aims of each chapter in light of these considerations.

#### **3.9.1 Chapter 4: Diagnostic boundaries and phenomenology of delusions**

In light of problems with the conceptual definition of delusions outlined in section 2.4 and the various philosophical approaches to delusions outlined in section 2.5, Chapter 4 will examine

the stability of the traditional psychopathological boundaries of delusion under the influence of a recent sociocultural development. This will involve the examination of how the internet has influenced the form, aetiology, and prognosis of internet-themed delusions, and how the use of the internet by people likely to be delusional, challenges the current diagnostic criteria.

### **3.9.2 Chapter 5: The Cardiff Anomalous Perceptions Scale and the role of anomalous perceptual experience in delusion formation.**

As highlighted in section 3.2.4, a crucial factor in many of the delusion formation models, is the role of anomalous perceptual experience, despite the fact that this is often poorly specified, is largely based on an understanding from clinical psychiatry and has not been systematically related to delusions or non-clinical psychosis continuum phenomena. One major shortcoming in research this area, is the lack of a valid, reliable measure of anomalous perceptual experience that is not rooted in the practise of clinical psychiatry. Therefore, Chapter 5 will comprehensively examine the phenomenology of anomalous perceptual experience, describe the development of such a scale and its use in investigating how this relates to the clinical presentation of delusions and the non-clinical presentation of delusional ideation.

### **3.9.3 Chapter 6: The role of the temporal lobes in delusion formation.**

As emphasised in section 3.7.3, research into the cognitive neuroscience of delusions relies on heterogeneous participants, produces largely correlational results and has implicated relatively large brain areas. This has limited the inferences that can be made about causal neuropsychological factors. One method of overcoming this problem is to attempt to modulate processes thought to be involved in delusion formation using carefully controlled non-clinical participants with a focused and well-controlled method. The transcranial

magnetic stimulation study reported in Chapter 6, aims to elucidate such causal factors, as well as further investigating the role of the temporal lobes in perceptual distortion and magical thinking in an attempt at validating some of the conclusions from Chapter 5.

### **3.9.4 Chapter 7: Distress, anomalous perceptual experience and delusions.**

Section 2.4 noted the lack of criteria for a pragmatic or 'ecologically valid' approach to the diagnosis of delusions, in that a belief could fulfil the diagnostic criteria, without the believer being in need of any significant clinical or social assistance. Notably, several of the theories of delusion formation discussed in this chapter do not differentiate between 'magical' or anomalous beliefs, and those which are personally distressing or disabling. There seems to be little room for anomalous beliefs which could be considered benign, or even beneficial to the individual concerned, despite the fact that they may otherwise fulfil the current diagnostic criteria.

The attributes of such beliefs may prove to be a significant counter example to the assumptions on which many theories of delusions are based. In response to this, Chapter 7 will examine a sample of Pagan participants, who have high levels of anomalous experience in the context of anomalous and magical beliefs, and compare their experiences to those of people with mainstream religious beliefs, no religious beliefs and clinically diagnosable pathologies of belief (psychotic inpatients). This will allow an examination of the relationship between distress and anomalous experience, and conclusions to be drawn as to the nature of belief pathology.

### **3.9.5 Chapter 8: Conclusions and theoretical integration**

Chapter 8 will summarise the findings and conclusions from previous chapters before examining the implications for theories of delusions and normal belief. Shortcomings and limitations of the evidence in light of these theories will be discussed, as will future research avenues for the development of this area.

### 3.10 References

- Abel, K. M., Jolley, S., Hemsley, D. R., Geyer, M. A. (2004). The influence of schizotypy traits on prepulse inhibition in young healthy controls. *Journal of Psychopharmacology*, 18, 181-188.
- Abu-Akel, A. (1999) Impaired theory of mind in schizophrenia. *Pragmatics and Cognition*, 7, 247-282.
- Adler, A. (1914/1929) Melancholia and paranoia. In A. Adler (ed.) *The practice and theory of individual psychology*. London: Routledge ,Kegan Paul Ltd.
- Baumeister, R. F., Campbell, J. D., Krueger, J. I., Vohs, K. D. (2003). Does high self-esteem cause better performance, interpersonal success, happiness, or healthier lifestyles? *Psychological Science in the Public Interest*, 4, 1-44.
- Bayne, T., Pacherie, E. (2005). In Defence of the Doxastic Conception of Delusions. *Mind and Language*, 20, 163-188.
- Bell, D. (2003) *Ideas in Psychoanalysis: Paranoia*. Cambridge: Icon Books Ltd.
- Bell, V. (2002) Book review: Pathologies of Belief by M. Coltheart, M. Davies. *Cognitive Neuropsychiatry*, 7 (4), 329-333.
- Bentall, R. P. (2003) *Madness Explained: Psychosis and Human Nature*. London: Penguin Books Ltd.

Bentall, R. P., Corcoran, R., Howard, R., Blackwood, N., Kinderman, P. (2001) Persecutory delusions: a review and theoretical integration. *Clinical Psychology Review*, 21, 1143-1192.

Bentall, R. P., Kaney, S. (1989) Content specific information processing and persecutory delusions: an investigation using the emotional Stroop test. *British Journal of Medical Psychology*, 62, 355-364.

Bentall, R. P., Kaney, S., Dewey, M. E. (1991) Paranoia and social reasoning: an attribution theory analysis. *British Journal of Clinical Psychology*, 30, 13-23.

Bentall, R. P., Kinderman, P., Kaney, S. (1994) The self, attributional processes and abnormal beliefs: towards a model of persecutory delusions. *Behaviour Research and Therapy*, 32, 331-341.

Berlim MT, Mattevi BS, Belmonte-de-Abreu P, Crow TJ. (2003) The etiology of schizophrenia and the origin of language: overview of a theory. *Comprehensive Psychiatry*, 44, 7-14.

Blackwood NJ, Bentall RP, Ffytche DH, Simmons A, Murray RM, Howard RJ. (2004) Persecutory delusions and the determination of self-relevance: an fMRI investigation. *Psychological Medicine*, 34 (4), 591-6.

Blackwood NJ, Bentall RP, ffytche DH, Simmons A, Murray RM, Howard RJ. (2003) Self-responsibility and the self-serving bias: an fMRI investigation of causal attributions.

*Neuroimage*, 20 (2), 1076-85.

Blackwood NJ, Howard RJ, Bentall RP, Murray RM. (2001) Cognitive neuropsychiatric models of persecutory delusions. *American Journal of Psychiatry*, 158 (4), 527-39

Blackwood NJ, Howard RJ, ffytche DH, Simmons A, Bentall RP, Murray RM. (2000) Imaging attentional and attributional bias: an fMRI approach to the paranoid delusion.

*Psychological Medicine*, 30, 873-83.

Blakemore, S. J. (2003) Deluding the motor system. *Consciousness and Cognition*, 12, 647-655.

Blakemore, S. J., Frith, C. (2003a) Self-awareness and action. *Current Opinion in Neurobiology*, 13, 219-224.

Blakemore, S. J., Frith, C. (2003b) Disorders of self-monitoring and the symptoms of schizophrenia. In T. Kircher and A. David (eds) *The Self in Neuroscience and Psychiatry*. Cambridge: Cambridge University Press.

Blakemore, S. J., Sarfati, Y., Bazin, N., Decety, J. (2003) The detection of intentional contingencies in simple animations in patients with delusions of persecution. *Psychological Medicine*, 33, 1433-1441.



Blakemore, S. J., Smith, J., Steel, R., Johnstone, C. E., Frith, C. D. (2000) The perception of self-produced sensory stimuli in patients with auditory hallucinations and passivity experiences: evidence for a breakdown in self-monitoring. *Psychological Medicine*, 30, 1131-1139.

Blakemore, S. J., Wolpert, D. M., Frith, C. D. (2002) Abnormalities in the awareness of action. *Trends in Cognitive Sciences*, 6, 237-242.

Bly BM, Kosslyn SM. (1997) Functional anatomy of object recognition in humans: evidence from positron emission tomography and functional magnetic resonance imaging. *Current Opinion in Neurology*, 10, 5-9.

Bourget D, Whitehurst L (2004) Capgras syndrome: a review of the neurophysiological correlates and presenting clinical features in cases involving physical violence. *Canadian Journal of Psychiatry*, 49, 719-25.

Bowins B, Shugar G. (1998) Delusions and self-esteem. *Canadian Journal of Psychiatry*, 43, 154-8.

Brébion, G., Amador, X., Smith, M. J., Malaspina, D., Sharif, Z., Gorman, J. M. (1999) Opposite links of positive and negative symptomatology with memory errors in schizophrenia. *Psychiatry Research*, 88, 15-24.

Brébion, G., Amador, X., David, A., Malaspina, D., Sharif, Z., Gorman, J. M. (2000) Positive symptomatology and source-monitoring failure in schizophrenia--an analysis of symptom-specific effects. *Psychiatry Research*, 95, 119-131.

Brébion, G., Gorman, J. M., Amador, X., Malaspina, D., Sharif, Z. (2002) Source monitoring impairments in schizophrenia: characterisation and associations with positive and negative symptomatology. *Psychiatry Research*, 112, 27-39.

Breen N, Caine D, Coltheart M. (2001) Mirrored-self misidentification: two cases of focal onset dementia. *Neurocase*, 7, 239-54.

Bruce, V., Green, P.R, Georgeson, M. (2003) *Visual Perception Physiology, Psychology and Ecology (4<sup>th</sup> ed)*. Taylor and Francis Ltd.

Brune M (2004) Schizophrenia-an evolutionary enigma? *Neuroscience and Biobehavioral Reviews*, 28, 41-53.

Burgess, P.W., Shallice, T. (1996) Bizarre responses, rule detection and frontal lobe lesions. *Cortex*, 32, 241-259.

Cahill C, Silbersweig D, Frith CD (1996) Psychotic experiences induced in deluded patients using distorted auditory feedback, *Cognitive Neuropsychiatry*, 1, 201-211

Candido, C. L., Romney, D. M. (1990) Attributional style in paranoid vs. depressed patients. *British Journal of Medical Psychology*, 63, 355-363.

Caspi A, Moffitt TE, Cannon M, McClay J, Murray R, Harrington H, Taylor A, Arseneault L, Williams B, Braithwaite A, Poulton R, Craig IW. (2005) Moderation of the effect of adolescent-onset cannabis use on adult psychosis by a functional polymorphism in the catechol-O-methyltransferase gene: longitudinal evidence of a gene X environment interaction. *Biological Psychiatry*, 57, 1117-27.

Chadwick, P., Birchwood, M. (1994) The omnipotence of voices. A cognitive approach to auditory hallucinations. *British Journal of Psychiatry*, 164, 190-201.

Chadwick, P., Birchwood, M., Trower, P. (1996) *Cognitive therapy for delusions, voices and paranoia*. Chichester: Wiley.

Chen EY. (1994) A neural network model of cortical information processing in schizophrenia. I: Interaction between biological and social factors in symptom formation. *Canadian Journal of Psychiatry*, 39, 362-7.

Chen EY. (1995) A neural network model of cortical information processing in schizophrenia. II: Role of hippocampal-cortical interaction: a review and a model. *Canadian Journal of Psychiatry*, 40, 21-6.

Clarke, I. (2001) *Psychosis and Spirituality*. London: Whurr.

Clore, G.L., Gasper, K.(2000) Some affective influences on beliefs. In Frijda, N.H., Manstead, A.S.R., Bem, S.(eds) *Emotions and Beliefs: How Feelings Influence Thoughts*. Cambridge: Cambridge University Press.

Cohen JD, Servan-Schreiber D. (1992) Context, cortex, and dopamine: a connectionist approach to behavior and biology in schizophrenia. *Psychological Review*, 99, 45-77.

Colby, K.M. (1975) *Artificial Paranoia: A Computer Simulation of Paranoid Processes*. Oxford: Pergamon Press Ltd.

Collins, A.M., Loftus, E.F. (1975) A spreading activation theory of semantic information processing. *Psychological Review*, 82, 407-428.

Corcoran, R., Mercer, G., Frith, C. D. (1995) Schizophrenia, symptomatology and social inference: Investigating 'theory of mind' in people with schizophrenia. *Schizophrenia Research*, 17, 5-13.

Corcoran, R., Cahill, C., Frith, C. D. (1997) The appreciation of visual jokes in people with schizophrenia: a study of 'mentalizing' ability. *Schizophrenia Research*, 24, 319-327.

Craig JS, Hatton C, Craig FB, Bentall RP. (2004) Persecutory beliefs, attributions and theory of mind: comparison of patients with paranoid delusions, Asperger's syndrome and healthy controls. *Schizophrenia Research*, 69, 29-33.

Crow TJ. (1997) Schizophrenia as failure of hemispheric dominance for language. *Trends in Neurosciences*, 20, 339-43.

Currie, G. (2000) Imagination, delusion and hallucination. In M. Coltheart, M. Davis (eds) *Pathologies of Belief*. Oxford: Blackwell.

Currie, G., Jureidini, J. (2001) Delusion, Rationality, Empathy. *Philosophy, Psychiatry and Psychology*, 8, 159-162.

Currie, G., Ravenscroft, I. (2002) *Recreative Minds*. Oxford: Oxford University Press.

Damasio AR. (1996) The somatic marker hypothesis and the possible functions of the prefrontal cortex. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, 351 (1346), 1413-20.

Davies, M., Coltheart, M., Langdon, R., Breen, N. (2001) Monothematic delusions: Towards a two-factor account. *Philosophy, Psychiatry, and Psychology*, 8, 133-158.

de Clerambault, G. (1942) *Oeuvre Psychiatrique*. Paris: Presses Universitaires de France.

de Haan L, Bakker JM. (2004) Overview of neuropathological theories of schizophrenia: from degeneration to progressive developmental disorder. *Psychopathology*, 37, 1-7.

Dickey CC, McCarley RW, Voglmaier MM, Niznikiewicz MA, Seidman LJ, Hirayasu Y, Fischer I, Teh EK, Van Rhoads R, Jakab M, Kikinis R, Jolesz FA, Shenton ME. (1999)

Schizotypal personality disorder and MRI abnormalities of temporal lobe gray matter.

*Biological Psychiatry*, 45, 1393-402.

Dinn WM, Harris CL, Aycicegi A, Greene P, Andover MS. (2002) Positive and negative schizotypy in a student sample: neurocognitive and clinical correlates. *Schizophrenia Research*, 56, 171-85.

Doody, G. A., Gotz, M., Johnstone, E. C., Frith, C. D., Cunningham Owens, D. G. (1998) Theory of mind and psychoses. *Psychological Medicine*, 28, 397-405.

Drury, V. M., Robinson, E. J., Birchwood, M. (1998) 'Theory of mind' skills during an acute episode of psychosis and following recovery. *Psychological Medicine*, 28, 1101-1112.

Ebmeier KP, Blackwood DHR, Murray C, Souza V, Walker M, Dougall N, Moffoot APR, O'Carroll RE, Goodwin GM (1993) Single-photon emission computed tomography with 99mTc-exametazine in unmedicated schizophrenic patients. *Biological Psychiatry*, 33, 487-495.

Edelstyn NM, Oyebode F. (1999) A review of the phenomenology and cognitive neuropsychological origins of the Capgras syndrome. *International Journal of Geriatric Psychiatry*, 14, 48-59.

Ellett, L., Lopes, B., Chadwick, P. (2003) Paranoia in a nonclinical population of college students. *Journal of Nervous and Mental Disease*, 191, 425-430.

Ellis HD. (1994) The role of the right hemisphere in the Capgras delusion. *Psychopathology*, 27, 177-85.

Ellis, H.D. (1998) Cognitive neuropsychiatry and delusional misidentification syndromes: An exemplary vindication of the new discipline. *Cognitive Neuropsychiatry*, 3, 81-90.

Ellis, H.D., Young, A. W. (1996) Problems of person perception in schizophrenia. In C. Pantelis, H. Nelson, T. Barnes (eds) *Schizophrenia: A neurological perspective*. Chichester: Wiley .

Ellis, H.D., Young, A. W., Quayle, A.H., De Pauw, K. W. (1997) Reduced autonomic responses to faces in Capgras delusion. *Proceedings of the Royal Society of London B: Biological Sciences*, 264, 1085-1092.

Fear, C. F., Healy, D. (1997) Probabilistic reasoning in obsessive-compulsive and delusional disorders. *Psychological Medicine*, 27, 199-208.

Fear, C., Sharp, H., Healy, D. (1996) Cognitive processes in delusional disorders. *British Journal of Psychiatry*, 168, 1-8.

Feinberg I. (1979) Efference copy and corollary discharge: implications for thinking and its disorders. *Schizophrenia Bulletin*, 4, 636-40.

Fitzgerald PB, de Castella AR, Folia KM, Folia SL, Benitez J, Kulkarni J. (2005)

Victimization of patients with schizophrenia and related disorders. *Australia and New Zealand Journal of Psychiatry*, 39, 169-74.

Ford JM, Mathalon DH. (2004) Electrophysiological evidence of corollary discharge dysfunction in schizophrenia during talking and thinking. *Journal of Psychiatric Research*, 38, 37-46.

Forstl, H., Almeida, O.P., Owen, A.M., Burns, A., Howard, R. (1991) Psychiatric, neurological and medical aspects of misidentification syndromes: a review of 260 cases. *Psychological Medicine*, 21, 905-910.

Freeman, D., Garety, P. (2004) *Paranoia: The Psychology of Persecutory Delusions*. Hove: Psychology Press.

Freeman, D., Garety, P. A., Fowler, D., Kuipers, E., Bebbington, P. E., Dunn, G. (2004) Why do people with delusions fail to choose more realistic explanations for their experiences? An empirical investigation. *Journal of Consulting and Clinical Psychology*, 72, 671-680.

Freeman, D., Garety, P., Fowler, D., Kuipers, E., Dunn, G., Bebbington, P., Hadley, C. (1998) The London-East Anglia randomized controlled trial of cognitive-behaviour therapy for psychosis. IV: Self-esteem and persecutory delusions. *British Journal of Clinical Psychology*, 37, 415-430.



Freeman, D., Garety, P. A., Kuipers, E., Fowler, D., Bebbington, P. E. (2002) A cognitive model of persecutory delusions. *British Journal of Clinical Psychology*, 41, 331-347.

Freeman, D., Garety, P. A., Kuipers, E. (2001). Persecutory delusions: developing the understanding of belief maintenance and emotional distress. *Psychological Medicine*, 31, 1293-1306.

Freeman, D., Garety, P. A., McGuire, P., Kuipers, E. (2005) Developing a theoretical understanding of therapy techniques: an illustrative analogue study. *British Journal of Clinical Psychology*, 44, 241-254.

Freud, S. (1911 / 2003) *The Schreber Case*. London: Penguin Classics.

Frijda, N.H., Manstead, A.S.R., Bem, S. (2000) The influence of emotion on beliefs. In Frijda, N.H., Manstead, A.S.R., Bem, S.(eds) *Emotions and Beliefs: How Feelings Influence Thoughts*. Cambridge: Cambridge University Press.

Frith, C.D. (1992) *The Cognitive Neuropsychology of Schizophrenia*. Hove: Psychology Press.

Frith CD, Done DJ. (1989) Experiences of alien control in schizophrenia reflect a disorder in the central monitoring of action. *Psychological Medicine*, 19, 359-63.

Frith, C. D., Corcoran, R. (1996) Exploring 'theory of mind' in people with schizophrenia. *Psychological Medicine*, 26, 521-530.

Fujii DE, Ahmed I. (2002) Characteristics of psychosis due to traumatic brain injury: an analysis of case studies in the literature. *Journal of Neuropsychiatry and Clinical Neuroscience*, 14, 130-40.

Fukuhara R, Ikeda M, Nebu A, Kikuchi T, Maki N, Hokoishi K, Shigenobu K, Komori K, Tanabe H. (2001) Alteration of rCBF in Alzheimer's disease patients with delusions of theft. *Neuroreport*, 12, 2473-6.

Fuller, R., Frith, C.D., Jahanshahi, M. (2000) Reduced negative priming does indicate reduced cognitive inhibition in schizophrenia. *Cognitive Neuropsychiatry*, 5, 21-35

Gallagher, S. (2004) Neurocognitive models of schizophrenia: A neurophenomenological critique. *Psychopathology*, 37, 8-19.

Garety, P. A., Freeman, D. (1999) Cognitive approaches to delusions: a critical review of theories and evidence. *British Journal of Clinical Psychology*, 38, 113-154.

Garety PA, Freeman D, Jolley S, Dunn G, Bebbington PE, Fowler DG, Kuipers E, Dudley R. (2005) Reasoning, emotions, and delusional conviction in psychosis. *Journal of Abnormal Psychology*, 114, 373-84.

Garety, P.A., Hemsley, D.R. (1994) *Delusions: Investigations into the Psychology of Delusional Reasoning*. Oxford: Oxford University Press.

- Garety, P. A., Kuipers, E., Fowler, D., Freeman, D., Bebbington, P. E. (2001) A cognitive model of the positive symptoms of psychosis. *Psychological Medicine*, 31, 189-195.
- Garlipp, P., Godecke-Koch, T., Dietrich, D. E., Haltenhof, H. (2004) Lycanthropy - psychopathological and psychodynamical aspects. *Acta Psychiatrica Scandinavica*, 109, 19-22.
- Gauntlett-Gilbert, J. Kuipers, E. (2005) Visual hallucinations in psychiatric conditions: Appraisals and their relationship to distress. *British Journal of Clinical Psychology*, 44, 77-87.
- Geroldi, C., Bresciani, L., Zanetti, O., Frisoni, G. B. (2002) Regional brain atrophy in patients with mild Alzheimer's disease and delusions. *International Psychogeriatrics*, 14, 365-378.
- Gibbs, A. A., David, A. S. (2003) Delusion formation and insight in the context of affective disturbance. *Epidemiologia e Psichiatria Sociale*, 12, 167-174.
- Gilleen, J., David, A. S. (2005) The cognitive neuropsychiatry of delusions: from psychopathology to neuropsychology and back again. *Psychological Medicine*, 35, 5-12.
- Gilovich, T. (1993) *How we know what isn't so: The fallibility of human reason in everyday life*. New York: Free Press.
- Glantz, L. A., Lewis, D. A. (2000) Decreased dendritic spine density on prefrontal cortical pyramidal neurons in schizophrenia. *Archives of General Psychiatry*, 57, 65-73.

Gold, I., Hohwy, J. (2000) Rationality and schizophrenic delusion. In M. Coltheart, M. Davis (eds) *Pathologies of Belief*. Oxford: Blackwell.

Gonzalez-Pinto, A.; van Os, J.; Peralta, V.; Perez de Heredia, J.L.; Mosquera, F.; Aldama, A.; Gonzalez, C.; Gutierrez, M.; and Mico, J.A. (2004) The role of age in the development of Schneiderian symptoms in patients with a first psychotic episode. *Acta Psychiatrica Scandinavica*, 109, 264-8.

Gourzis P, Katrivanou A, Beratis S. (2002) Symptomatology of the initial prodromal phase in schizophrenia. *Schizophrenia Bulletin*, 28, 415-29.

Gray, J.A. (1982) *The Neuropsychology of Anxiety: An Enquiry into the Function of the Septohippocampal System*. Oxford: Oxford University Press.

Gray, N. S., Fernandez, M., Williams, J., Ruddle, R. A., Snowden, R. J. (2002) Which schizotypal dimensions abolish latent inhibition? *British Journal of Clinical Psychology*, 41, 271-284.

Gray, N. S., Snowden, R. J. (in press). The relevance of irrelevance to schizophrenia. *Neuroscience and Biobehavioural Reviews*

Green, M. J., Phillips, M. L. (2004) Social threat perception and the evolution of paranoia. *Neuroscience and Biobehavioral Reviews*, 28, 333-342.

Gur RE, Chin S. (1999) Laterality in functional brain imaging studies of schizophrenia. *Schizophrenia Bulletin*, 25, 41-56.

Healy, D (2002) *The Creation of Psychopharmacology*. Harvard: Harvard University Press.

Heiser JF, Colby KM, Faught WS, Parkison RC. (1979) Can psychiatrists distinguish a computer simulation of paranoia from the real thing? The limitations of Turing-like test as measures of the adequacy of simulations. *Journal of Psychiatric Research*, 15, 149-62.

Hemsley, D.R. (1994) Perceptual and cognitive abnormalities as the bases for schizophrenic symptoms. In A.S. David, J.C. Cutting (eds) *The Neuropsychology of Schizophrenia*. Hove: LEA.

Higgins ET. (1987) Self-discrepancy: A theory relating self and affect. *Psychological Review*, 94, 319-40.

Hirono N, Mori E, Ishii K, Kitagaki H, Sasaki M, Ikejiri Y, Imamura T, Shimomura T, Ikeda M, Yamashita H (1998) Alteration of regional cerebral glucose utilization with delusions in Alzheimer's disease. *The Journal of Neuropsychiatry and Clinical Neurosciences*, 10, 433-439.

Hirstein, W. (2005) *Brain fiction: Self-deception and the riddle of confabulation*. MIT: MIT Press.

Hirstein, W., Ramachandran, V.S. (1997) Capgras syndrome: a novel probe for understanding the neural representation of the identity and familiarity of persons. *Proceedings of the Royal Society of London B: Biological Sciences*, 264, 437-444.

Hoffman RE, Dobscha SK. (1989) Cortical pruning and the development of schizophrenia: a computer model. *Schizophrenia Bulletin*, 15, 477-90

Hoffman RE, McGlashan TH. (1993) Parallel distributed processing and the emergence of schizophrenic symptoms. *Schizophrenia Bulletin*, 19, 119-40.

Hoffman RE, McGlashan TH. (2001) Neural network models of schizophrenia. *Neuroscientist*, 7, 441-54.

Hohwy, J., Rosenberg, R. (2005) Unusual Experiences, Reality Testing and Delusions of Alien Control. *Mind and Language*, 20, 141-162.

Hopfield, J. J. (1982) Neural networks and physical systems with emergent collective computational abilities. *Proceedings of National Academy of Sciences*, 74, 2554- 2558.

Hurn, C., Gray, N. S., Hughes, I. (2002) Independence of 'reaction to hypothetical contradiction' from other measures of delusional ideation. *British Journal of Clinical Psychology*, 41, 349-360.

Huq SF, Garety PA, Hemsley DR. (1988) Probabilistic judgements in deluded and non-deluded subjects. *The Quarterly Journal of Experimental Psychology. A, Human Experimental Psychology*, 40, 801-12.

Johns, L. C., van Os, J. (2001) The continuity of psychotic experiences in the general population. *Clinical Psychology Review*, 21, 1125-1141.

Johns LC, Rossell S, Frith C, Ahmad F, Hemsley D, Kuipers E, McGuire PK. (2001) Verbal self-monitoring and auditory verbal hallucinations in patients with schizophrenia. *Psychological Medicine*, 31, 705-15.

Johnson, M.K. (1988) Discriminating the origin of information. In T.F. Oltmanns and B.A. Maher (eds) *Delusional beliefs*. Chichester: Wiley.

Johnson, M.K. (1991) Reality monitoring: Evidence from confabulation in organic brain disease patients. In G.P Prigatano, D.L. Schacter (eds) *Awareness of Deficit After Brain Injury: Clinical and Theoretical Issues*. New York: Oxford University Press.

Johnson, M.K. (1997) Source monitoring and memory distortion. *Philosophical Transactions of the Royal Society of London*, 352, 1733-45.

Johnson M.K., Raye C.L. (1998) False memories and confabulation. *Trends in Cognitive Sciences*, 2, 137-145.

Johnson, M.K., Raye, C.L. (2000) Cognitive and brain mechanisms of false memories and beliefs. In D.L. Schacter and E. Scarry (eds) *Memory, Brain and Belief*. Cambridge Massachusetts: MIT Press.

Jones, H. M., Pilowsky, L. S. (2002) Dopamine and antipsychotic drug action revisited. *British Journal of Psychiatry*, 181, 271-275

Kaney, S., Bentall, R. P. (1989) Persecutory delusions and attributional style. *British Journal of Medical Psychology*, 62, 191-198.

Kaney, S., Bentall, R. P. (1992) Persecutory delusions and the self-serving bias. Evidence from a contingency judgment task. *Journal of Nervous and Mental Disease*, 180, 773-780.

Kaplan RD, Szechtman H, Franco S, Szechtman B, Nahmias C, Garnett ES, List S, Cleghorn JM (1993) Three clinical syndromes of schizophrenia in untreated subjects: relation to brain glucose activity measured by positron emission tomography (PET). *Schizophrenia Research*, 11, 47-54.

Kapur, S. (2003). Psychosis as a state of aberrant salience: a framework linking biology, phenomenology, and pharmacology in schizophrenia. *American Journal of Psychiatry*, 160, 13-23.

Kay SR, Fiszbein A, Opler LA. (1987) The positive and negative syndrome scale (PANSS) for schizophrenia. *Schizophrenia Bulletin*, 13, 261-276.



Keefe, R. S., Arnold, M. C., Bayen, U. J., Harvey, P. D. (1999) Source monitoring deficits in patients with schizophrenia; a multinomial modelling analysis. *Psychological Medicine*, 29, 903-914.

Keefe, R. S., Arnold, M. C., Bayen, U. J., McEvoy, J. P., Wilson, W. H. (2002) Source-monitoring deficits for self-generated stimuli in schizophrenia: multinomial modelling of data from three sources. *Schizophrenia Research*, 57, 51-67.

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

Kinderman, P., Bentall, R. P. (1997) Causal attributions in paranoia and depression: Internal, personal and situational attributions for negative events. *Journal of Abnormal Psychology*, 106, 341-345.

Kinderman P, Bentall RP. (2000) Self-discrepancies and causal attributions: studies of hypothesized relationships. *British Journal of Clinical Psychology*, 39, 255-73.

Kinderman, P., Prince, S., Waller, G., Peters, E. (2003) Self-discrepancies, attentional bias and persecutory delusions. *British Journal of Clinical Psychology*, 42, 1-12.

Klee, R. (2004) Why Some Delusions Are Necessarily Inexplicable Beliefs. *Philosophy, Psychiatry and Psychology*, 11, 25-34.

Kohonen, T. (1989) *Self-Organization and Associative Memory* (3<sup>rd</sup> ed). Berlin: Springer.

Koriat A. (2000) The feeling of knowing: some metatheoretical implications for consciousness and control. *Consciousness and Cognition*, 9, 149-71.

Kravetz, S., Faust, M.,Edelman, A. (1998) Dimensions of schizotypy and lexical decision in the two hemispheres. *Personality and Individual Differences*, 25, 857-871.

Kriekhaus EE, Donahoe JW, Morgan MA. (1992) Paranoid schizophrenia may be caused by dopamine hyperactivity of CA1 hippocampus. *Biological Psychiatry*, 31, 560-70.

Krstev, H., Jackson, H.,Maude, D. (1999) An investigation of attributional style in first-episode psychosis. *British Journal of Clinical Psychology*, 88, 181–194.

Krystal JH, D'Souza DC, Karper LP, Bennett A, Abi-Dargham A, Abi-Saab D, Cassello K, Bowers MB Jr, Vegso S, Heninger GR, Charney DS. (1999) Interactive effects of subanesthetic ketamine and haloperidol in healthy humans. *Psychopharmacology*, 145, 193-204.

Kubicki M, McCarley R, Westin CF, Park HJ, Maier S, Kikinis R, Jolesz FA, Shenton ME. (in press) A review of diffusion tensor imaging studies in schizophrenia. *Journal of Psychiatry Research*

Langdon, R., Coltheart, M. (1999) Mentalising, schizotypy, and schizophrenia. *Cognition*, 71, 43-71.

Langdon, R., Coltheart, M. (2000) The cognitive neuropsychology of delusions. In M. Coltheart and M. Davies (eds) *Pathologies of Belief*. Oxford: Blackwell Publishing.

Lee, H. J. (2000) Attentional bias, memory bias and the self-concept in paranoia. *Psychological Science*, 9, 77–99.

Lee DA, Randall F, Beattie G, Bentall RP. (2004) Delusional discourse: an investigation comparing the spontaneous causal attributions of paranoid and non-paranoid individuals. *Psychology and Psychotherapy*, 77, 525-40.

Leonhard, D., Brugger, P. (1998). Creative, paranormal, and delusional thought: a consequence of right hemisphere semantic activation? *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, 11(4), 177-183.

Liddle PF, Friston KJ, Frith CD, Hirsch SR, Jones T, Frackowiak RSJ (1992) Patterns of cerebral blood flow in schizophrenia. *British Journal of Psychiatry*, 160, 179-186.

Linney, Y. M., Peters, E. R., Ayton, P. (1998). Reasoning biases in delusion-prone individuals. *British Journal of Clinical Psychology*, 37, 285-302.

Lisanby SH, Kohler C, Swanson CL, Gur RE. (1998) Psychosis secondary to brain tumor. *Seminars in Clinical Neuropsychiatry*, 3, 12-21.

Lubow RE. (1997) Latent inhibition as a measure of learned inattention: some problems and solutions. *Behavioural Brain Research*, 88, 75-83

Lyon, H. M., Kaney, S., Bentall, R. P. (1994) The defensive function of persecutory delusions. Evidence from attribution tasks. *British Journal of Psychiatry*, 164, 637-646.

Maher, B. (1974) Delusional thinking and perceptual disorder. *Journal of Individual Psychology*, 30, 98-113.

Maher, B. (1988) Anomalous experience and delusional thinking: The logic of explanations. In T.F. Oltmanns, B.A. Maher (eds) *Delusional beliefs*. Chichester: Wiley.

Maher, B. A. (1999) Anomalous experience in everyday life: Its significance for psychopathology. *The Monist*, 82, 547-570.

Makarec, K., Persinger, M. A. (1985). Temporal lobe signs: electroencephalographic validity and enhanced scores in special populations. *Perceptual and Motor Skills*, 60, 831-842.

Malenka RC, Angel RW, Hampton B, Berger PA. (1982) Impaired central error-correcting behavior in schizophrenia. *Archives of General Psychiatry*, 39, 101-7.

Malloy PF, Richardson ED. (1994) The frontal lobes and content-specific delusions. *The Journal of Neuropsychiatry and Clinical Neurosciences*, 6, 455-66.

Maruff, P., Wood, S. J., Velakoulis, D., Smith, D. J., Soulsby, B., Suckling, J., Bullmore, E. T., Pantelis, C. (2005) Reduced volume of parietal and frontal association areas in patients

with schizophrenia characterized by passivity delusions. *Psychological Medicine*, 35, 783-789.

Martin JA, Penn DL. (2002) Attributional style in schizophrenia: an investigation in outpatients with and without persecutory delusions. *Schizophrenia Bulletin*, 28, 131-41.

McCabe, R., Leudar, I., Antaki, C. (2004) Do people with schizophrenia display theory of mind deficits in clinical interactions? *Psychological Medicine*, 34, 401–412.

McCarley RW, Wible CG, Frumin M, Hirayasu Y, Levitt JJ, Fischer IA, Shenton ME. (1999) MRI anatomy of schizophrenia. *Biological Psychiatry*, 45, 1099-1119.

McDonald C, Bullmore ET, Sham PC, Chitnis X, Wickham H, Bramon E And Murray RM. (2004) Association of genetic risks for schizophrenia and bipolar disorder with specific and generic brain structural endophenotypes. *Archives of General Psychiatry*, 61, 974-84.

McGlashan TH, Hoffman RE. (2000) Schizophrenia as a disorder of developmentally reduced synaptic connectivity. *Archives of General Psychiatry*, 57, 637-48.

McKay, R., Langdon, R., Coltheart, M. (2005) “Sleights of mind”: Delusions, defences and self-deception. *Cognitive Neuropsychiatry*, 10, 205-326.

Menon, M., Pomarol-Clotet, E., McCarthy, R. A., McKenna, P. J. (2002). Probabilistic reasoning bias is a function of having schizophrenia, not of being deluded. *Schizophrenia Research Supplement*, 53, 133.

Mentis MJ, Weinstein EA, Horwitz B, McIntosh AR, Pietrini P, Alexander GE, Furey M, Murphy DG. (1995) Abnormal brain glucose metabolism in the delusional misidentification syndromes: a positron emission tomography study in Alzheimer disease. *Biological Psychiatry*, 38, 438-49.

Mezulis, A. H., Abramson, L. Y., Hyde, J. S., Hankin, B. L. (2004) Is there a universal positivity bias in attributions?: A meta-analytic review of individual, developmental, and cultural differences in the self-serving attributional bias. *Psychological Bulletin*, 130, 711–747.

Miall, R. C., Weir, D. J., Wolpert, D. M., Stein, J. F. (1993) Is the cerebellum a Smith predictor? *Journal of Motor Behaviour*, 25, 203-216.

Mohr, C., Rohrbach, C. M., Laska, M., Brugger, P. (2001). Unilateral olfactory perception and magical ideation. *Schizophrenia Research*, 47(2-3), 255-264.

Moritz S, Werner R, von Collani G (in press) The inferiority complex in paranoia re-addressed. A study with the Implicit Association Test. *Cognitive Neuropsychiatry*

Moritz, S., Woodward, T. S. (2005). Jumping to conclusions in delusional and non-delusional schizophrenic patients. *British Journal of Clinical Psychology*, 44, 193-207.

Moritz S, Woodward TS, Whitman JC, Cuttler C. (2005) Confidence in errors as a possible basis for delusions in schizophrenia. *Journal of Nervous and Mental Disease*, 193, 9-16.

- Mortimer, A. M., Bentham, P., McKay, A. P., Quemada, L., Clare, L., Eastwood, N., McKenna, P. J. (1996). Delusions in Schizophrenia: A Phenomenological and Psychological Exploration. *Cognitive Neuropsychiatry*, 1, 289-303.
- Myin-Germeys, I., Marcelis, M., Krabbendam, L., Delespaul, P., van Os, J. (2005) Subtle fluctuations in psychotic phenomena as functional states of abnormal dopamine reactivity in individuals at risk. *Biological Psychiatry*, 58, 105-110.
- Myin-Germeys, I., Nicolson, N.A., Delespaul, P.A.E.G. (2001) The context of delusional experiences in the daily life of patients with schizophrenia. *Psychological Medicine*, 31, 489–498.
- Ohayon MM, Schatzberg AF. (2002) Prevalence of depressive episodes with psychotic features in the general population. *American Journal of Psychiatry*, 159, 1855-61.
- Pantelis C, Velakoulis D, McGorry PD, Wood SJ, Suckling J, Phillips LJ, Yung AR, Bullmore ET, Brewer W, Soulsby B, Desmond P, McGuire PK. (2003) Neuroanatomical abnormalities before and after onset of psychosis: a cross-sectional and longitudinal MRI comparison. *Lancet*, 25, 361, 281-8.
- Persinger, M. A., Makarec, K. (1987) Temporal lobe epileptic signs and correlative behaviors displayed by normal populations. *Journal of General Psychology*, 114(2), 179-195.

Peters, E., Day, S., Garety, P. (1999b). A longitudinal study of cognitive abnormalities in delusions at different levels of information processing. *Schizophrenia Research*, 36, 180.

Peters, E. R., Garety, P.A. (in press) Cognitive functioning in delusions: A longitudinal analysis. *Behaviour Research and Therapy*

Peters, E. R., Joseph, S. A., Garety, P. A. (1999a) Measurement of delusional ideation in the normal population: introducing the PDI (Peters et al. Delusions Inventory). *Schizophrenia Bulletin*, 25, 553-576.

Peters, E., Joseph, S., Day, S., Garety, P. (2004) Measuring delusional ideation: the 21-item Peters et al. Delusions Inventory (PDI). *Schizophrenia Bulletin*, 30, 1005-1022.

Phillips, M. L., Senior, C., David, A. S. (2000) Perception of threat in schizophrenics with persecutory delusions: an investigation using visual scan paths. *Psychological Medicine*, 30, 157-167.

Pia L, Neppi-Modona M, Ricci R, Berti A. (2004) The anatomy of anosognosia for hemiplegia: a meta-analysis. *Cortex*, 40, 367-77.

Pizzagalli, D., Lehmann, D., Brugger, P. (2001) Lateralized direct and indirect semantic priming effects in subjects with paranormal experiences and beliefs. *Psychopathology*, 34(2), 75-80.



Pizzagalli, D., Lehmann, D., Gianotti, L., Koenig, T., Tanaka, H., Wackermann, J., Brugger P. (2000) Brain electric correlates of strong belief in paranormal phenomena: intracerebral EEG source and regional Omega complexity analyses. *Psychiatry Research*, 100, 139-154.

Ramachandran, V. S. (1994a) Phantom limbs, neglect syndromes, repressed memories, and Freudian psychology. *International Review of Neurobiology*, 37, 291-333.

Ramachandran, V. S. (1994b) Phantom limbs, somatoparaphrenic delusions, neglect syndromes, repressed memories and Freudian psychology. In O. Sporns, G. Tononi (Eds.), *Neuronal group selection*. San Diego: Academic Press.

Ramachandran, V. S. (1995) Anosognosia in parietal lobe syndrome. *Consciousness, Cognition: an International Journal*, 4(1), 22-51.

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

Richards, P. M., Persinger, M. A., Koren, S. A. (1993). Modification of activation and evaluation properties of narratives by weak complex magnetic field patterns that simulate limbic burst firing. *International Journal of Neuroscience*, 71(1-4), 71-85.

Roberts, G. (1991) Delusional belief systems and meaning in life: a preferred reality? *The British Journal of Psychiatry Supplement*, 14, 19-28.

- Ruppin, E., Reggia, J. A., Horn, D. (1996) Pathogenesis of schizophrenic delusions and hallucinations: a neural model. *Schizophrenia Bulletin*, 22, 105-123.
- Sabri O, Erkwoh R, Schreckenberger M, Owega A, Sass H, Buell U (1997) Correlation of positive symptoms exclusively to hyperperfusion or hypoperfusion of cerebral cortex in never-treated schizophrenics. *Lancet*, 349, 1735–1739
- Sarfati, Y., Hardy-Baylé, M. C., Besche, C., Widlocher, D. (1997) Attribution of intentions to others in people with schizophrenia: A non-verbal exploration with comic strips. *Schizophrenia Research*, 25, 199-209.
- Salkovskis, P.M. (1991) The importance of behaviour in maintenance of anxiety and panic: A cognitive account. *Behavioural Psychotherapy*, 19, 6-19.
- Sartorius N, Jablensky A, Korten A, Ernberg G, Anker M, Cooper JE, Day R. (1986) Early manifestations and first-contact incidence of schizophrenia in different cultures. A preliminary report on the initial evaluation phase of the WHO Collaborative Study on determinants of outcome of severe mental disorders. *Psychological Medicine*, 16, 909-28.
- Schreber, D.P. (2000) *Memoirs of My Nervous Illness*. New York: NYRB Classics.
- Schuldberg D, French C, Stone BL, Heberle J. (1988) Creativity and schizotypal traits. Creativity test scores and perceptual aberration, magical ideation, and impulsive nonconformity. *Journal of Nervous and Mental Disease*, 176, 648-57.

Serper, M., Dill, C. A., Chang, N., Kot, T., Elliot, J. (2005). Factorial structure of the hallucinatory experience: continuity of experience in psychotic and normal individuals. *Journal of Nervous and Mental Disease*, 193, 265-272.

Serretti A, Lilli R, Lorenzi C, Lattuada E, Smeraldi E. (2001) DRD4 exon 3 variants associated with delusional symptomatology in major psychoses: a study on 2,011 affected subjects. *American Journal of Medical Genetics*, 105, 283-90.

Serretti A, Lorenzi C, Mandelli L, Cichon S, Schumacher J, Nothen MM, Rietschel M, Tullius M, Ohlraun S. (2004) DRD4 exon 3 variants are not associated with symptomatology of major psychoses in a German population. *Neuroscience Letters*, 368, 269-73.

Shallice, T., Burgess, P.W. (1998) The domain of supervisory processes and temporal organization of behaviour. In A.C. Roberts, T.W. Robbins and L. Weiskrantz (eds) *The prefrontal cortex: Executive and cognitive functions*. Oxford: Oxford University Press

Shanks, M. F., Venneri, A. (2002) The emergence of delusional companions in Alzheimer's disease: An unusual misidentification syndrome. *Cognitive Neuropsychiatry*, 7, 317-328.

Shanks, M. F., Venneri, A. (2004) Thinking through delusions in Alzheimer's disease. *British Journal of Psychiatry*, 184, 193-194.

Sharp, H. M., Fear, C. F., Healy, D. (1997) Attributional style and delusions : An investigation based on delusional content. *European Psychiatry*, 12, 1-7.

- Shenton ME, Dickey CC, Frumin M, McCarley RW. (2001) A review of MRI findings in schizophrenia. *Schizophrenia Research*, 49, 1-52.
- Siekmeier PJ, Hoffman RE. (2002) Enhanced semantic priming in schizophrenia: a computer model based on excessive pruning of local connections in association cortex. *British Journal of Psychiatry*, 180, 345-50.
- Singh, G., Sharan, P., Kulhara, P. (2003) Phenomenology of hallucinations: a factor analytic approach. *Psychiatry and Clinical Neurosciences*, 57, 333-336.
- Smith N, Freeman D, Kuipers E. (2005) Grandiose delusions: an experimental investigation of the delusion as defense. *Journal of Nervous and Mental Disease*, 193, 480-7.
- Spence, S. A. (1996) Free will in the light of neuropsychiatry. *Philosophy, Psychiatry and Psychology*, 3, 75-90.
- Spence, S. A., Brooks, D. J., Hirsch, S. R., Liddle, P. F., Meehan, J., Grasby, P. M. (1997) A PET study of voluntary movement in schizophrenic patients experiencing passivity phenomena (delusions of alien control). *Brain*, 120, 1997-2011.
- Spitzer, M. (1995) A neurocomputational approach to delusions. *Comprehensive Psychiatry*, 36, 83-105.
- Staff RT, Shanks MF, Macintosh L, Pestell SJ, Gemmell HG, Venneri A. (1999) Delusions in Alzheimer's disease: PET evidence of right hemispheric dysfunction. *Cortex*, 35, 549-60.

Staff RT, Venneri A, Gemmell HG, Shanks MF, Pestell SJ, Murray AD. (2000) HMPAO SPECT imaging of Alzheimer's disease patients with similar content-specific autobiographic delusion: comparison using statistical parametric mapping. *Journal of Nuclear Medicine*, 41, 1451-5.

Starkstein SE, Robinson RG, Berthier ML. (1992) Post-stroke hallucinatory delusional syndromes. *Neuropsychiatry, Neuropsychology and Behavioral Neurology*, 5, 114-8.

Starkstein SE, Vazquez S, Petracca G, Sabe L, Migliorelli R, Teson A, Leiguarda R (1994) A SPECT study of delusions in Alzheimer's disease. *Neurology*, 44, 2055-2059.

Steel, C., Fowler, D., Holmes, E. (2005) Trauma-related intrusions and psychosis: An information processing account. *Behavioural and Cognitive Psychotherapy*, 33, 139-153.

Steer RA, Kumar G, Pinninti NR, Beck AT. (2003) Severity and internal consistency of self-reported anxiety in psychotic outpatients. *Psychological Reports*, 93, 1233-8.

Stephens, G.L., Graham, G. (2000) *When Self-Consciousness Breaks: Alien Voices and Inserted Thoughts*. MIT: MIT Press.

Stevens, J.R. (1992) Abnormal reinnervation as a basis for schizophrenia. *Archives of General Psychiatry*, 49, 238-243.

Stone, T., Young, A.W. (1997) Delusions and brain injury: The philosophy and psychology of belief. *Mind and Language*, 12, 327-364.

Sultzer, D. L., Brown, C. V., Mandelkern, M. A., Mahler, M. E., Mendez, M. F., Chen, S. T., Cummings, J. L. (2003) Delusional thoughts and regional frontal/temporal cortex metabolism in Alzheimer's disease. *American Journal of Psychiatry*, 160, 341-349.

Sumich, A., Chitnis, X. A., Fannon, D. G., O'Ceallaigh, S., Doku, V. C., Faldrowicz, A., Sharma, T. (2005) Unreality symptoms and volumetric measures of Heschl's gyrus and planum temporal in first-episode psychosis. *Biological Psychiatry*, 57, 947-950.

Taylor, K. I., Zach, P., Brugger, P. (2002). Why is magical ideation related to leftward deviation on an implicit line bisection task? *Cortex*, 38(2), 247-252.

Tenyi, T., Herold, R., Szili, I. M., Trixler, M. (2002). Schizophrenics show a failure in the decoding of violations of conversational implicatures. *Psychopathology*, 35, 25-27.

Thorton, T. (2002). Thought insertion, cognitivism, and inner space. *Cognitive Neuropsychiatry*, 7, 237-249.

Tranel D, Damasio H, Damasio A (1995) Double dissociation between overt and covert face recognition. *Journal of Cognitive Neuroscience*, 7, 425-432.

Trzesniewski, K.H., Donnellan, M.B, Robins, R.W. (2003) Stability of self-esteem across the life span. *Journal of Personality and Social Psychology*, 84, 205-220.

Turing, A. (1950) Computing machinery and intelligence. *Mind*, 59, 433-460.

van Os, J., Hanssen, M., Bijl, R.V., Ravelli, A. (2000) Strauss (1969) revisited: a psychosis continuum in the general population? *Schizophrenia Research*, 45, 11-20.

Velligan DI, Bow-Thomas CC. (1999) Executive function in schizophrenia. *Seminars in Clinical Neuropsychiatry*, 4, 24-33.

Venneri A, Shanks MF, Staff RT, Della Sala S. (2000) Nurturing syndrome: a form of pathological bereavement with delusions in Alzheimer's disease. *Neuropsychologia*, 38, 213-24.

Vinogradov S, King RJ, Huberman BA. (1992) An associationist model of the paranoid process: application of phase transitions in spreading activation networks. *Psychiatry*, 55, 79-94.

Ward, J., Parkin, A.J., Powell, G., Squires, E., Townshend, J., Bradley, V. (1999) False recognition of unfamiliar people: "Seeing film stars everywhere". *Cognitive Neuropsychology*, 16, 293-315.

Weinstein, S., Graves, R. E. (2002). Are creativity and schizotypy products of a right hemisphere bias? *Brain and Cognition*, 49(1), 138-151.

Weizenbaum, J. (1966) ELIZA - A computer program for the study of natural language communication between man and machine. *Communications of the Association for Computing Machinery*, 9, 36-45.

Winterer G, Weinberger DR. (2004) Genes, dopamine and cortical signal-to-noise ratio in schizophrenia. *Trends in Neurosciences*, 27, 683-90.

Wolkin A, Rusinek H. (2003) A neuropathology of psychosis? *Lancet*, 361, 270-1

Woo TU, Whitehead RE, Melchitzky DS, Lewis DA. (1998) A subclass of prefrontal gamma-aminobutyric acid axon terminals are selectively altered in schizophrenia. *Proceedings of the National Academy of Sciences of the United States of America*, 95, 5341-6.

Young, A.W. (1999) Delusions. *The Monist*, 82, 571-589.

Young, H. F., Bentall, R. P. (1997) Social reasoning in individuals with persecutory delusions: The effects of additional information on attributions for the observed behaviour of others. *British Journal of Clinical Psychology*, 36, 569-573.

Zigler E, Glick M. (1988) Is paranoid schizophrenia really camouflaged depression? *American Psychologist*, 43, 284-90.



## Chapter Four

### The Diagnostic and Phenomenological Boundaries of Delusions

Elements of this chapter are in publication as:

Bell, V., Maiden, C., Muñoz-Solomando, A. & Reddy, V. (2006) 'Mind control experiences' on the internet: Implications for the psychiatric diagnosis of delusions. *Psychopathology*, 39, 87-91.

Bell, V., Grech, E., Maiden, C., Halligan, P.W. & Ellis, H.D. (2005) 'Internet Delusions': A Case Series and Theoretical Integration. *Psychopathology*, 38, 144-150.

Bell, V. (2004) Mind control experiences on the internet and the psychiatric diagnosis of delusions. *Proceedings of the British Psychological Society*, 12 (2), 162.

The following presentations have also included material from this chapter:

Bell, V. Mind Control Experiences on the Internet and the Psychiatric Diagnosis of Delusions. *BPS Annual Conference*, April 2004

## **4.1 Chapter outline**

Sections:

- 4.2 What can the internet tell us about delusions ?
- 4.3 “Mind control” experiences on the internet: Implications for the psychiatric diagnosis of delusions.
- 4.4 “Internet delusions”: A case series and theoretical integration
- 4.5 References

This is the first of the empirical chapters and presents two studies which address issues highlighted in Chapters 2 and 3. A particular concern is the effect of cultural and social influences on delusions. This has been neglected (section 2.4.3), and as such has potentially lead to an incomplete understanding based on the Jaspersian idea that delusions represent an encapsulated phenomenon (Jaspers, 1923), caused by the same “underlying biological signal”, whereas cultural influences simply affect “surface content” (Spence, 2001; section 2.4.4).

The two studies presented here focus on the interaction between the internet (a recent social and cultural development) and delusional beliefs. The interaction between delusions and the internet as a cultural phenomenon, and the use of the internet by people who are likely to be delusional, is examined to test the stability of the traditional psychopathological boundaries of delusion.

## **4.2 What can the internet tell us about delusions ?**

One of the most revolutionary social and cultural changes in recent times has been the growth and public availability of the internet. The internet is a particularly pertinent topic to study for

its influence and interaction with delusions, because, as a cultural phenomenon, it has a number of interesting properties.

- i) It entered popular culture relatively recently (during the mid-1990s) with the growth of the World Wide Web and the explosion in commercially available internet services.
- ii) Despite the fact that much of the telecommunications infrastructure relies upon it, very few people are aware of how it works, making it seem both powerful, mysterious and potentially dangerous.
- iii) It has been a matter of ongoing public concern, both over the content available online, and the possibilities that this new technology brings. Concern have centred on issues of privacy, fraud and the use of the network by people considered subversive, dangerous or deviant.
- iv) It is now ubiquitous. Internet access is cheap and widely available for both home and business use (Eurostat estimate that by 2004, almost two thirds of UK individuals and 90% of UK businesses used the internet; Eurostat, 2004).
- v) It allows people with minority beliefs to find like-minded others. “On the internet, people who share your interest and lean in the same direction as you are just a few keystrokes away, regardless of the issue’s obscurity, social desirability, bizarreness” (Wallace, 1999, p79).

These factors suggest that the internet has a complex influence on individuals and society, as the internet is concurrently, a social concern, a participatory activity, a mysterious technology and a source of refuge for people with fringe beliefs. On purely *prima facie* evidence, the

internet, therefore, would seem to be a prime topic for investigating the influence of a recent sociocultural development on delusions.

If such an influence could be shown to violate the current conceptual boundaries of delusion, enshrined in the diagnostic criteria and Jaspersian orthodoxy, further evidence would be provided that delusions are multi-faceted and complex phenomena, more akin to a syndrome than the traditional symptom assumption (Gilleen and David, 2005).

### **4.3 “Mind control” experiences on the internet: Implications for the psychiatric diagnosis of delusions.**

#### **4.3.1 Abstract**

*Background:* The DSM criteria for a delusion indicate that it should not include any beliefs held by a person’s ‘culture or subculture’. The internet has many examples of people reporting ‘mind control experiences’ (MCEs) on self-published web pages, many of which suggest a community based around such beliefs and experiences. It was hypothesised that some of these reports will reflect delusional beliefs and the hyperlinks between web reports are likely to show evidence of social structure, demonstrating the ‘culture or subculture’ exemption to be increasingly redundant in light of new technology.

*Sampling and Methods:* Texts from web sites reporting MCEs ( $N = 10$ ), experience of cancer ( $N = 10$ ), depression ( $N = 10$ ) and being stalked ( $N = 10$ ) were identified, and were blind-rated by three independent psychiatrists for the presence of delusions. Hyperlinks from web sites reporting MCEs were used to create a network structure; this was compared with a size-matched, randomly generated network and known social networks from the literature using social network analysis.

*Conclusions:* The sampled web-published accounts of MCEs are highly likely to be influenced by delusional beliefs. Social network analysis suggests there is significant evidence of an online community based around these beliefs. The fact that individuals can form a community based on the content of a potentially delusional belief present a paradox for the DSM diagnostic criteria for a delusion, and suggests the need to revise and revisit the original operational definition in the light of these new technological developments.

### **4.3.2 Background**

Accounts of mind control experiences have been among the most influential in psychiatry. The first book-length study of a psychiatric patient was of James Tilly Matthews' fantastical account of being controlled, mind and body, by a sinister-sounding pneumatic device called the 'air loom', originally published by the Bethlem Hospital apothecary John Haslam in 1810 (Haslam and Porter, 1989). In a seminal paper Tausk (1933) called these delusions of mechanistic control the phenomenon of the 'influencing machine' and related them particularly to schizophrenia. Kurt Schneider (1959) thought these symptoms of passivity or external control so important as to list them among his 'first rank symptoms', which he believed were of particular diagnostic importance in schizophrenia. Although it is now questioned whether first rank symptoms are reliably diagnostic (Bertelsen, 2002), such experiences remain a common theme, with studies reporting their prevalence of anything between 27% (Salleh, 1992) and 73% (O'Grady, 1990) in schizophrenia.

It has been noted that Matthews' experience of early nineteenth century mind control bears more than a passing resemblance to modern day reports of mind control experiences (MCEs) (Jay, 2003). Many of these modern-day reports are available as self-published web pages on

the internet. Increasingly, the world-wide web (or simply ‘the web’), is used to publish online diaries, journals and accounts of personally significant events. This form of personal publishing is becoming popular as a method of disseminating personal views and opinions (Gillmor, 2004). The dynamic nature of the web means that other people’s writing and research can be incorporated or referenced, making for a fast-moving online community.

Unusual, anomalous or distressing experiences are particularly noteworthy and it is hardly surprising that personal accounts of illness, both physical and mental, are now a permanent feature of the internet landscape. This material would seem to be a fertile ground for research scientists, but one which seems to have been largely ignored in reviews of this area. One exception is a recent review by Hewson (2003), who mentioned the internet as a source of observations, and Davies and Lipsey (2003), who have highlighted the proliferation of web sites promoting anorexia. Notably, the method used here (using social network analysis and the rating of internet texts for psychopathological content) is a novel approach to clinical research on the internet.

Of particular interest for this study is the exemption clause in the diagnostic criteria for a delusion, as laid out in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR; APA, 2000). According to this criterion a belief is not considered delusional if it is “accepted by other members of the person’s culture or subculture”. This criterion has already been recognised to be problematic, because the definition of what should be considered when assessing the culture or subculture to which a person belongs is ambiguous and largely subjective (Leeser and O’Donohue, 1999). As has been noted, one characteristic feature of internet authorship is the way in which authors may include themselves in an online community and create or join their own niche subculture. This creates a potential diagnostic

paradox if it is found that authors of accounts describing potentially delusional experiences are part of an online community based around the content of such experiences.

This study aimed to examine whether the content of selected web-published accounts of MCEs expressed delusional beliefs, and whether a structural analysis of the links made between the various web sites show signs of social organisation. This was completed with a view to testing whether the “culture or subculture” exclusion criteria in the DSM diagnosis of delusions can be sustained in light of new technological developments that allow cultures or subcultures based around minority beliefs to be formed online.

### **4.3.3 Method**

#### *4.3.3.1 Analysis of web site content*

Ten web published accounts of MCEs and ten accounts each of experiences of cancer, depression and being stalked were selected from the world-wide web. Accounts were identified by using internet search engines, and by following subsequent links to related pages. The majority of MCE reports on the internet are held on one web site (Light, 2003); for this study, however, to avoid any bias due to editorial control only accounts published independently and by a single author were included.

Three independent psychiatrists, blind to the purpose of the study, were asked to rate each account for the presence of delusions, hallucinations and passivity symptoms. Accounts were presented to the raters as printed material, untitled, and showing only the text from the relevant web pages in a uniform font and page layout. The raters were further asked to make a best guess as to a likely diagnosis, based only on the experiences described in the text.

#### *4.3.3.2 Social network analysis of links between 'mind control' web sites*

Social network analysis (SNA) is a research tool for identifying structures in social networks based on the relations between the components in the system, where a social network is a conceptualised as a set of 'nodes', which represent people, organisations or other social actors, with links between nodes representing relationships such as affiliation or information exchange (Wasserman and Faust, 1994).

Jackson (1997) and Wellman (2001) have argued that SNA is particularly suited to analysing the hyperlinks between web-sites and that the structure of such links is likely to reflect the communicative choices, agendas and underlying social structure of the authors – a view which has been supported by reviews of the empirical hyperlink analysis literature by Park (2003) and Park and Thelwall (2003).

For the purposes of this study the sampled network was created using the 'snowball sampling' method (Goodman, 1961) whereby the initial nodes (the ten accounts of mind control experiences) are identified and hyperlinks from these are used to identify a further set of connected nodes. This was completed by downloading webpages containing the accounts of MCEs; and using custom software to extract and collate the hyperlinks made by each author. This list was then processed so that duplicate hyperlinks made by the same author were counted as one, as were multiple hyperlinks made to the same web-site.

Using the methodology of Lusseau (2003), the sampled network was then compared with a network, generated to contain the same number of nodes and links as the sampled network, but connected randomly. Additionally, the sampled network was compared with several known social networks from the social network analysis literature. Two were technology



mediated social networks, namely Freeman and Freeman's (1979) study of users of an early computer conferencing system and Killworth and Bernard's (1976) study of calls between ham radio users. The other was a network centred around a shared interest, namely Zachary's (1977) study on ties between members of a university karate club. All network analysis was conducted using UCINET 6 (Borgatti et al., 2002) a software package for social network analysis, which also contains the data sets from the literature used for comparison.

Comparisons with both a size-matched random network and known social networks were conducted for several reasons. Network measures can be sensitive to the network size, so an ideal comparison would involve finding a pre-identified social network with the same number of nodes and links as the sampled network. This becomes increasingly non-trivial for networks containing more than a few elements. In an attempt to avoid this problem, Lusseau (2003) has favoured comparison with a size-matched random network. It might be argued, however, that demonstrating a sampled network to be non-random, is not the same as showing that it has signs of social organisation. Therefore, both methods were used in this study in an attempt to control for the shortcomings of each.

Various measures of network properties have been found to be useful in the analysis of social networks and the following were selected for this study. The distance or  $d$  of a network is defined as the mean length of the shortest path between any two nodes. The smaller the value of  $d$ , the quicker information can be transferred between individuals, a property thought to be important in cohesive subgroups (Wasserman and Faust, 1994). The clustering co-efficient or  $C$ , is a measure of social relatedness between individuals. For each node, it provides the likelihood that two associates of that node are associates themselves (Watts and Strogatz, 1998). Group degree centralisation or  $C_D$  is a measure of how network connections focus on

specific node or nodes and is thought to be an important structural attribute of social networks (Freeman, 1979).

#### **4.3.4 Results**

##### *4.3.4.1 Web site content*

Inter-rater reliability was assessed by calculating Cohen's kappa for each permutation of paired raters with an overall kappa for each category calculated as a mean value, as recommended by Conger (1980). The mean kappas for ratings on all 40 accounts were as follows: delusions = .92 (SD = .07); hallucinations = .68 (SD = .13); passivity symptoms = .90 (SD = .04). These results suggest an excellent level of discrimination between delusional and non-delusional authors, and those with and those without-passivity symptoms.

As the focus of the study is on potential community involvement among people likely to be delusional, it is notable that when only the accounts selected for the social network analysis were included in the inter-rater reliability analysis there was full agreement (mean kappa = 1.0) among raters that the accounts expressed delusional beliefs. The attributions for the source of the MCEs by the authors of the accounts are given in table 4.1.

The overall agreement among raters when considering all account for diagnoses was .78, calculated using Fleiss' kappa (Fleiss, 1971) to allow for empty cells in the comparison table. Notably, all MCE accounts were classified as describing experiences of schizophrenia by the raters, except on one occasion, where a rater classified the text as describing experiences of delusional disorder. Furthermore, seven of the ten authors of MCE accounts mention contact with psychiatric services, suggesting other mental health professionals may have considered their experiences as signs of psychopathology.

These results suggest that signs of psychosis are strongly present in the selected web-published accounts of mind control experiences.

<i>Text</i>	<i>Attribution of 'mind control' experiences</i>
1.	“ex-military neighbours” and “husband’s cohorts” using “recently declassified technology”
2.	“rings of sex deviates” (sic) using “high energy radiation” technology.
3.	Royal Canadian Mounted Police using a “telepathic amplifier that works with microwaves”
4.	“Freemasonic intelligence agencies” using “frequency weapons”
5.	“police” using a “brain implant”
6.	“implantable controlling chip”
7.	“Dutch government” using a “network of transmitters”
8.	“politicians and journalists” using “satellite surveillance and harassment technologies”
9.	“Bad Guys” using “psychotronics” and “microwaves”
10.	“Warsaw Pact Military Research” using “hypnosis and electromagnetic waves”

*table 4.1. Details of mind control experience reports. NB: Following the recommendation of Davies and Lipsey (2003) individual web addresses have not been published.*

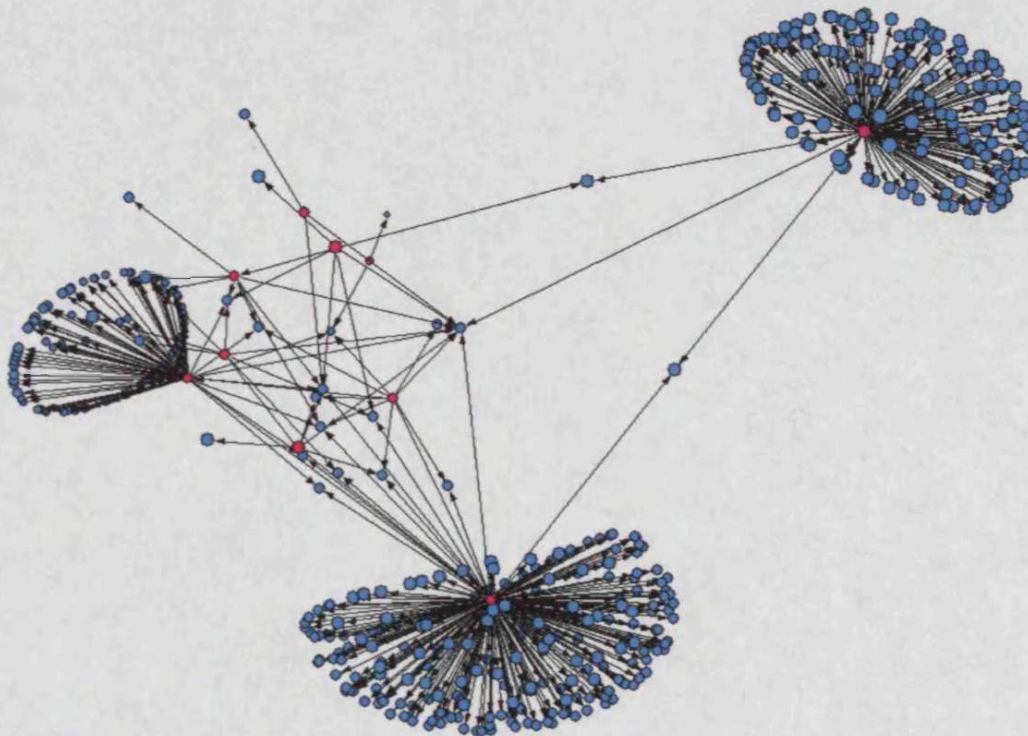
#### 4.3.4.2 Social network analysis

Table 4.2 outlines the network properties derived from the sampled network of accounts of MCEs, the known social networks from the literature and the size-matched randomly generated network. Figures 4.1 and 4.2 show the structure of the ‘mind control’ and size matched random networks.

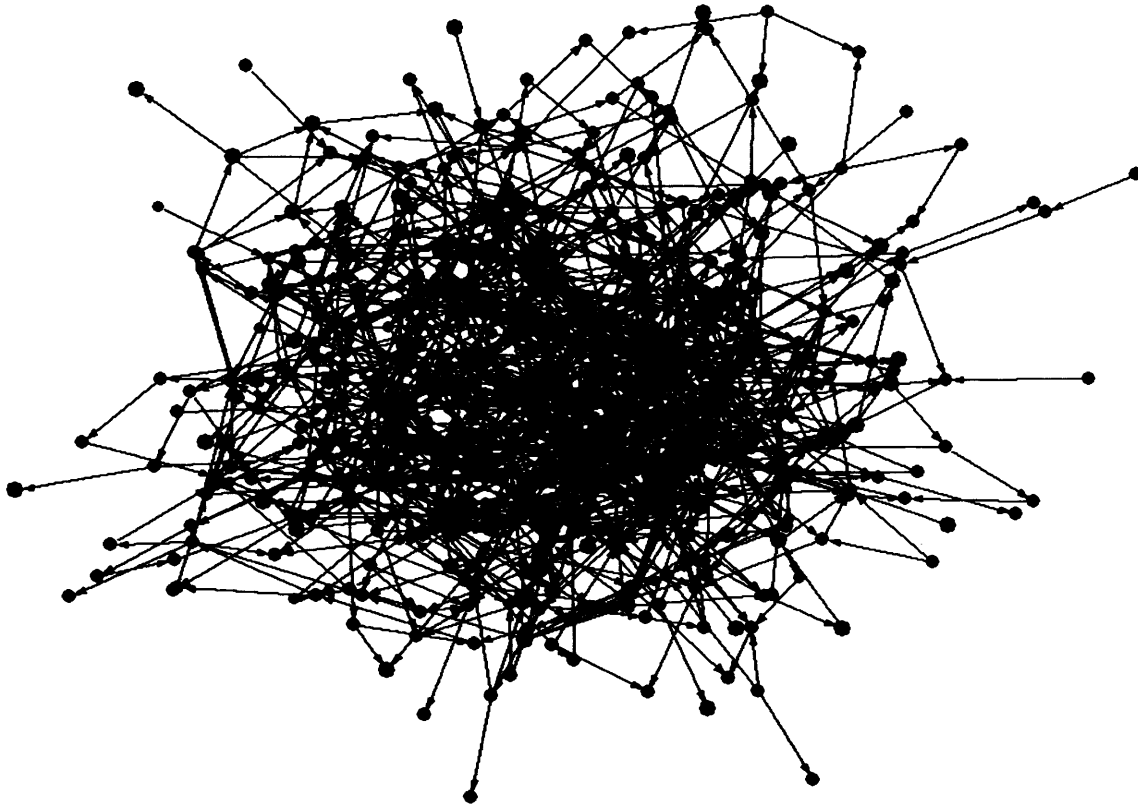
<i>Network</i>	<i>d</i>	<i>C</i>	<i>C<sub>D</sub></i>
Mind control experience	1.571	.158	49.25%
Computer conferencing	1.345	.750	49.26%
Ham radio	2.034	.687	48.84%
Karate Club	2.408	.588	39.96%
Size-matched random network	4.527	.010	1.29%

*d* = distance. *C* = clustering coefficient. *C<sub>D</sub>* = Group degree centralisation.

*table 4.2. Properties of network sampled from mind control experience reports compared to other social networks and size-matched random network.*



*figure 4.1 Structure of sample 'mind control' network using 3D Fruchterman-Reingold algorithm. Purple signifies the 10 initially identified websites.*



*figure 4.2 Structure of size matched randomly generated network using 3D Fruchterman-Reingold algorithm*

From inspecting figures 4.1 and 4.2 it is immediately apparent that, despite having exactly the same number of links and nodes, the sampled ‘mind control’ network has a more coherent structure.

In terms of network metrics, the sampled mind control network has a small  $d$  or distance, comparable with the other known social networks, and is almost three times smaller than the random network. This suggests that the sampled network shows properties similar to a real social network and indicates an efficient transfer of information through its members.

While the value of the clustering coefficient,  $C$ , is relatively small compared with the known social networks, it is many times greater than the size-matched random network. This

suggests that the organisation of the network is far from random, but that there is a lower probability than in the known social networks that individuals form social clusters within the greater social group.

The group degree centralisation measure ( $C_D$ ) is equivalent to or greater than for the known social networks, and many times above that of the random network. This too suggests evidence of social organisation and also that the network is centralised around some key members.

#### **4.3.5 Discussion**

This study selected ten web-published account of mind control experiences and analysed them for both the content and hyperlink structure. Analysis showed a significantly higher level of psychosis-like symptomology and greater concordance with the diagnostic criteria for schizophrenia than similar reports describing experiences of cancer, depression or being stalked. Social network analysis of the hyperlinks made from accounts of MCEs suggests evidence of social organisation and community, based around the content of these experiences and beliefs.

Common themes also occur in the accounts of MCEs. Scientific papers are sometimes cited, particularly one by Frey (1962) entitled “Human auditory system response to modulated electromagnetic energy”. There is frequent mention of the CIA’s MKULTRA project, a classified behavioural modification project from the 1960s which was heavily criticised for testing (largely drug based techniques) on non-consenting and unwitting members of the public (U. S. Senate Select Committee on Intelligence, 1977). These themes are usually cited as evidence for the reality of the authors’ experiences. Additionally, authors may identify

themselves with organised campaigns to lobby authorities to stop the unauthorised testing of ‘mind control technology’ on members of the public. The presence of common and specific themes across accounts, despite the variations of belief in the alleged perpetrators, suggests a level of information exchange between web-site authors to explain their experiences.

It must be noted that it is not being suggested that all members of a community interested in these issues show signs of psychopathology. Although this study’s social network analysis showed signs of community engagement between likely-psychotic people, it is noteworthy that the sampled authors are also likely to be an active part of a wider, non-psychotic community, who may have thematically similar, albeit differently motivated, concerns.

Both of these facts, however, pose particular challenges for the current diagnostic criteria for delusions, particularly the clause in the DSM-IV definition exempting beliefs that are “accepted by other members of the person’s culture or subculture”. Indeed, the results presented here demonstrate a paradox, in that the sampled authors’ online community is based upon the content of potentially delusional beliefs, which, technically, cannot be diagnosed as delusional if they are shared by a community to which a person belongs. It now seems clear that online communities have much in common with offline communities, with many of the same dynamics apparent in each, including frequent intra-group communication and strong feelings of cohesion (Wallace, 1999; Forster, 2004).

This suggests that internet communities such as this one, may be as much of a “culture or subculture” as communities based on geographical location, at least in terms of core psychological features. To extend the paradox, by DSM-IV criteria it would seem that a person diagnosed as delusional would only have to find him or herself the nearest internet

connected computer to 'cure' themselves of delusion by searching out other people with the same belief and joining or forming an online community.

It seems that the use of the internet by potentially psychotic persons may be adding to the criticism of the philosophical basis for defining delusions, which has already been attacked on its criteria of fixedness (Myin-Germeys et al., 2001; section 2.4.1), certainty and incorrigibility (Garety and Hemsley, 1994; section 2.4.1) and falsity (Spitzer, 1990; section 2.4.2).

Also of interest is the parallel between the internet mind control community and (presumably) non-pathological belief formation networks such as the scientific community. In both of these communities access to a large corpus of material allows new beliefs to be inferred and defended on the basis of that corpus. Furthermore, it is doubtful whether the previously identified common themes in the 'mind control' accounts could have been independently incorporated into individual community members' potentially delusional explanations without significant community influence. Indeed, for a group that is highly likely to hold pathological beliefs they make excellent use of social belief formation mechanisms.

Given the evidence presented here, it is particularly noteworthy that a potentially disabled and disenfranchised group have co-opted available technology to create a complex, dynamic and information-rich community that serves to support and inform similarly-affected people within the confines of a world view driven by potentially psychotic symptoms. This is a striking example of a support network completely removed from the traditional medico-legal support networks of the state and even the grassroots support networks run by mental health services user groups. In particular it demonstrates that the internet may enable complex



support mechanisms without reference to a view of reality held by the authorities, or even the mainstream of opinion. Whether this sort of support network works well for all, if any individuals, remains to be seen.

In conclusion, the presence of a complex and evolving online community based around the content of potentially psychotic experience challenges mainstream psychiatric understanding and diagnostic criteria for how a delusion is defined. This suggests that the current diagnostic criteria do not adequately capture delusional phenomena, and in particular, that the criteria may become redundant in light of new sociocultural developments.

In terms of the debate over whether delusions are a unitary phenomenon, qualitatively different from normal belief, the evidence provided here suggest that they show a great deal of similarities with normal belief. The websites sampled for this study suggest that the authors express and integrate their beliefs in a comparable way to non-delusional people. Indeed, the major difference between the texts rated as expressing delusional rather than non-delusional beliefs was content (the authors made impossible or impossible sounding claims), rather than form, as the authors of the cancer, stalking and depression accounts also reported seemingly fixed, incorrigible beliefs, and in some cases, accompanied by persecutory ideation and other signs of mental illness.

Although the rating of self-produced texts created outside of a clinical context has some obvious advantages, it also has the disadvantage of being an atypical assessment of the authors' beliefs. A complementary clinical approach would allow the use of interview and case notes to draw together the observations of clinically trained staff, to produce an analysis based on descriptive psychopathology, as is reported in section 4.4.

## **4.4 “Internet delusions”: A case series and theoretical integration.**

### **4.4.1 Abstract**

*Background:* Delusions involving the internet have been reported as examples of the influence of cultural innovations on delusion formation, although there has been some debate as to whether such innovations simply affect surface content, or have more substantial clinical or psychopathological implications.

*Sampling and Methods:* Four cases of patients with delusions involving the internet were identified following a general request to local consultant psychiatrists for referrals.

*Results:* The internet had a specific effect on aetiology in one case, and knowledge of the internet seemed to constrain the type of delusion formed in two others. The presence of an internet-related delusion in the final case was used to frame a successful clinical intervention based on the ‘collaborative empiricism’ method, using cognitive behavioural therapy and collaborative use of the internet to resolve the delusional belief.

*Conclusions:* Cultural technical innovations may have specific influences on the form, origin and content of delusional beliefs. For some patients the presence of internet-themed delusions may be a good prognostic indicator since, given the rich sources of information available, they may be well suited to treatment with cognitive behavioural therapy.

### **4.4.2 Background**

The widespread public availability of the internet has brought about significant changes to commerce and social communication. It is now a standard method of communication for many and is a regularly referenced in commercials, news stories and academic study. It is not

surprising, therefore, that delusions have been reported in the psychiatric literature in which the internet has featured as a central theme (see table 4.3).

<i>Reference</i>	<i>Age</i>	<i>Sex</i>	<i>Psychiatric history</i>	<i>Reported delusions</i>
Tan et al (1997)	27	M	Body dysmorphic disorder	Feeling that his life was being controlled by the internet, concern about "double-talk on the internet", neighbour putting information about his life online.
Catalano et al (1999)				
Case 1	40	M	None reported	Sexual photographs and video of him / girlfriend on internet, mind and body control by 'internet bugs' and 'links' from a web page.
Case 2	41	M	None reported	Believed he was a witch and ran an online service for witches and could 'surf the net using only his mind'. Received 'magnetism' from the internet each day.
Podoll et al (2000)				
Case 1	32	M	1 <sup>st</sup> presentation	Receiving messages over the internet threatening to expose his use of internet pornography.
Case 2	19	M	1 <sup>st</sup> presentation	Bill Gates was destroying his files, spying and following him. Personal files were duplicating themselves and being broadcast over the internet beyond his control.
Kobayashi et al (2001)	57	F	Schizoaffective disorder	Heard commanding voices from two men, sent via radio waves originally emanating from a satellite and transmitted through the internet.
Duggal et al (2002)	31	M	Paranoid schizophrenia	Sister-in-law was controlling his thoughts and actions, sending voices and reading his thoughts through the internet.
Margolese et al (2002)	26	M	Schizophrenia	People follow his activities via internet chat-rooms devoted to him and that several well known websites are dedicated to him because of similarities with his name and theirs.
Compton (2003)				
Case 1	53	F	'previous hospitalisation'	Internet controlling her (via implanted microchips) and home appliances for past 3 years.
Case 2	21	F	1 <sup>st</sup> presentation	Photographs and recordings of her were being broadcast on the internet.
Case 3	64	F	1 <sup>st</sup> presentation	Being followed by 'www' (World Wide Web) people.
Schmid-Siegel et al (2004)	36	F	Paranoid schizophrenia	Activities were being broadcast via web-cam to the internet. Chip implanted in her brain broadcast everything she saw on the internet.

*table 4.3. Previous reports of delusions about the internet from the psychiatric literature.*

As mentioned earlier, broad sociocultural influences on delusional beliefs are well known and previous research has shown an influence on the presentation of delusions depending on

country (Kala and Wig, 1982; Kim et al., 1993; Stompe et al., 1999; Tateyama et al., 1993), gender and social class (Suhail, 2003) and social environment (Suhail and Cochrane, 2002). Considering that the DSM-IV-TR diagnostic criteria for a belief to be considered delusional are culturally relative (one of the criteria being that the belief “is not one ordinarily accepted by other members of the person's culture or subculture”; APA, 2000) it is perhaps unsurprising that cultural factors might cause differences in the prevalence of certain themes between localised populations.

More intriguing have been reports of people whose delusions have incorporated recent cultural and / or technical innovations. Several reports of ‘rock and roll’ delusions have been reported, where patients have believed themselves to be owed money by, persecuted by, or in a romantic relationship with specific and recently popular musicians or singers (Glass and Campbell, 1984; Robinson, 1984; Ruedrich et al., 1983). More specific still, Forsyth et al. (2001) reported on a patient with the delusional belief that he was a character in a particular computer game. Notably, the game in question was released only a short time before the report was published.

There is no clear understanding of how long it takes for cultural innovations to influence delusional beliefs, although as can be seen from figure 4.3, delusions about the internet were not reported until the internet was discussed quite widely in national publications, suggesting, that a level of cultural salience (or perhaps social concern) has to be achieved before such concepts typically become incorporated into paranoid or psychotic experiences.

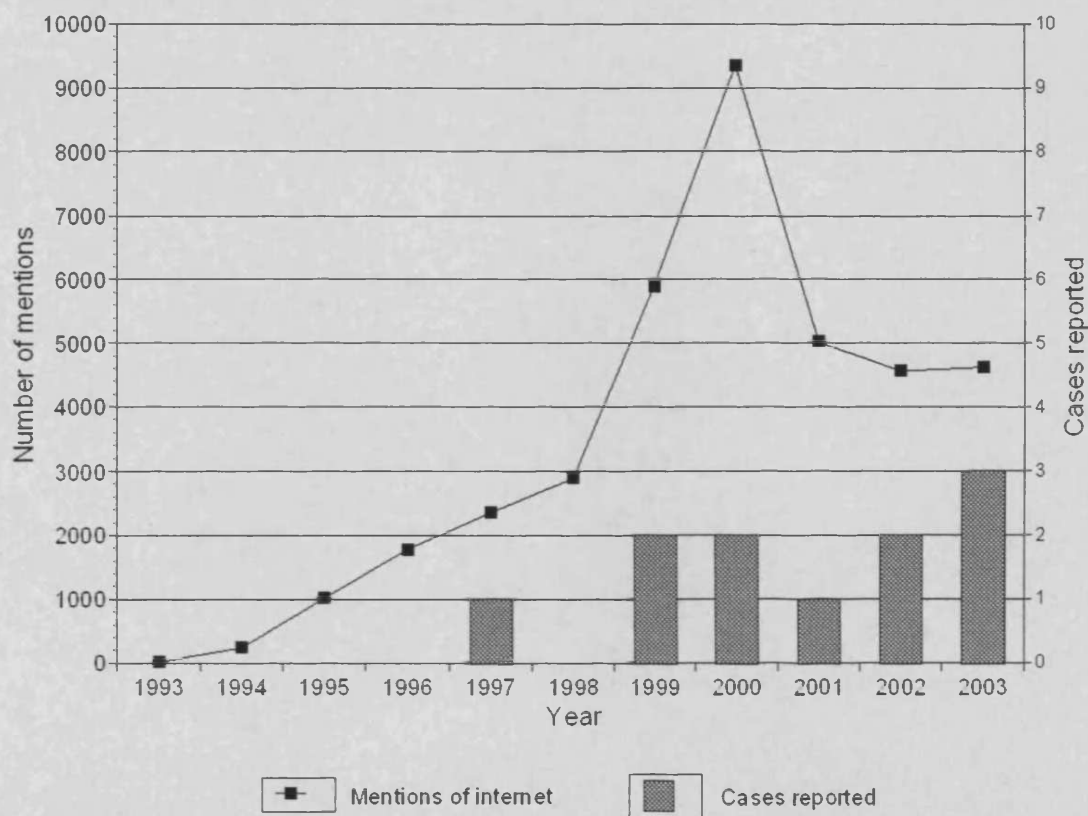


figure 4.3 Mentions of the internet in *The London Times* and *Sunday Times* from 1993 to 2003 and number of cases of reported “internet delusions” in the medical literature.

Commenting on the case of “computer game delusions”, Spence (2001), argued that such themes are simply a surface change to the same “underlying biological signal”, echoing Jaspers (1923) distinction between form and content in psychopathology (Spence, personal communication). In this formulation, an influence on the psychopathological form would entail a change to the phenomenological structure of an experienced symptom, including the type of psychological constructs involved (e.g. beliefs, perceptions, thoughts). Conversely, an influence on psychopathological content suggests a difference in the colouring of experience, for example, affecting exactly what is delusionally believed or hallucinated. The psychopathological form is the basis upon which psychiatric diagnosis is made, having

implications for prognosis and treatment, whereas the content is largely considered irrelevant in deciding upon such matters (Sims, 2003;section 2.4.4).

Reports of delusions about the internet, however, may suggest that cultural and environmental influences can represent more than superficial substitutions in content, with the internet in particular having an influence on psychopathological form and aetiology and being important for decisions of treatment methodology.

As can be seen from a review of published cases (table 4.3), delusions involving the internet can vary considerably in presentation. It is notable that the role of the internet in such delusional beliefs is largely restricted to two major themes. The first is the use of the internet as an explanatory tool to account for unusual experiences, such as experiences of control, voice hearing or having one's thoughts read. This phenomenon was first noted by Tausk (1933) in his seminal paper 'On the Origin of the Influencing Machine in Schizophrenia', where he noted that technology is often present as an explanatory device in psychosis and takes the form of a diabolical machine, just outside the technical understanding of the subject, usually claimed to be operated by enemies or persecutors of the affected person. Catalano et al. (1999) have argued that a lack of knowledge about the relevant technology may fuel internet-themed delusions, perhaps leading to the internet increasingly appearing as a modern-day incarnation of Tausk's "influencing machine".

A second theme is the supposed use of the internet by people who are thought to be conspiring against the affected person. Note here that the internet is not represented as having a direct malign influence, but typically is thought as a means of hosting chat rooms, photos or recordings about the person concerned. Although delusional 'bizarreness' is not a concept

without difficulties (Spitzer et al., 1993; sections 2.3.2; 2.4.3) the difference between these two themes may reflect the distinction between bizarre (impossible) and non-bizarre (possible but false) delusions, although perhaps further technological breakthrough might render delusions previously considered bizarre as simply false.

Whether such differences account for more than the trivial incorporation of themes into an otherwise well established psychopathological process remains a matter of debate. Stompe et al. (2004) argue that sociocultural influences on delusional presentation can be broken down into a number of levels each of which, in different ways, could be susceptible to the influences of cultural and social setting. They argue that technological innovations only affect the final stages ('concretization') of a multi-stage pathological process that culminates in the formation of a delusion; and, therefore, like Spence, they suggest that the influence of a technology such as the internet is relatively unimportant as an aetiological factor in psychosis.

In contrast, Catalano et al. (1999) suggest that 'internet delusions' may represent a "new subtype of a previously reported psychiatric illnesses", echoing similar ideas that internet addiction may be a novel form of psychopathology, owing to the internet having unique features which may lead people into heavy or even compulsive overuse (Griffiths, 1996; Shapira et al., 2003).

Other authors have taken the middle ground. Eytan et al. (2002) presented two cases of delusions where patients complained of being implanted with a microchip, and suggested that such technological delusions may be a form of Western culture-bound syndrome. The arguments put forward by Catalano et al. (1999) and Eytan et al. (2002) seem difficult to defend in light of the fact that they produce little persuasive evidence for a significant effect

of technological innovation on the form, aetiology or prognosis of the delusions they report. However, one recent case has suggested that technological themes may be involved in the development of novel forms of psychopathology, where previous symptom definitions and diagnostic criteria do not fully capture the obviously aberrant experience.

Schmid-Siegel et al. (2004) reported the case of a woman diagnosed with paranoid schizophrenia who believed that everything she saw was being broadcast over the internet. The authors labelled this as 'perception broadcast' and noted that, although seeming similar to thought broadcast, it involved visual perceptions and not thoughts, and did not involve the direct participation of others, as the internet was believed to be a mediating technology and, therefore, did not fulfil Schneider's (1959) thought broadcast criteria. In this case, a technological innovation seems to have influenced the psychopathological form of a psychotic symptom, rather than content alone.

Furthermore, a case reported by Duggal et al. (2002) suggests that the presence of internet-themed delusions may be a specific prognostic indicator. They noted particular success with using cognitive therapy to treat the delusional belief, largely because of the ease by which reality testing by 'collaborative empiricism' (Chadwick and Lowe, 1990) can be used in therapy.

The authors reported that they were able both to educate their client as to how the internet works (by using a widely available book) and collaboratively to draw up criteria that could potentially disprove the reality of the client's delusions. Crucially, they were then able directly to test these criteria by using the internet to gather evidence with the client, leading to the rejection of the delusional belief. The ubiquity of the internet makes it possible to easily



engage in these sorts of reality testing tasks, whereas doing the same for delusions concerning (for example) spirits, magical forces or even microchips or radio waves, may be difficult, if not impossible, to implement practically.

Such cases suggest that technological concepts may have specific influences on the aetiology, form and implications for the clinical management of people with such delusional beliefs.

Wishing to explore these issues further, I sought to examine locally-occurring cases of internet-themed delusions.

#### **4.4.3 Method**

A letter was written to consultant psychiatrists ( $N = 21$ ) in the South Wales area, requesting information on any cases of “delusions involving the internet” or other recent technological developments. Over a period of one year, four cases such cases were reported, and these are described below. One further case was reported but rejected for inclusion in this case series as the patient’s initial clinical presentation was put in doubt after further psychiatric assessment.

#### **4.4.4 Case Reports**

##### *4.4.4.1 Case 1*

WL, a 31 year old woman, with a previous diagnosis of bipolar affective disorder, was admitted to hospital after being found in the street in a distressed state. On admission, she recounted how she first felt unwell six weeks earlier and became suspicious when her credit card was refused in a shop, leading to a sense of unease and increasingly intrusive thoughts. Subsequently, while examining the packaging of a breath freshening product she noticed the ingredient ‘phenylalanine’, which she proceeded to use as a search term on an internet search engine. Her search resulted in finding a webpage, containing many numbers, which outlined

experimental studies on the chemical. Using the most personally-significant numbers from the initial page as search terms she further found a website explaining an Aramaic system for divining special meaning from numbers. She suspected this was significant and came to believe that she had found secret information about the 'Al-Queda' terrorist network. During the following days she believed that, because of her discovery of terrorist secrets, her computer and telephone had been tapped, in order to monitor her internet activity and phone calls, and she was being bugged by microphones and concealed cameras. WL was subsequently diagnosed as having a manic episode with psychotic symptoms. She has extensive experience with using the internet over the previous ten years, and when asked how the internet worked replied "by linking computers all over the world using energy and digital technology".

#### *4.4.4.2 Case 2*

KD, a 42 year old man had consulted his GP five weeks earlier for low mood and suspiciousness, although he had no previous contact with mental health services. At interview KD claimed that the websites of several international companies had a "darker side" and hidden sections that were being used by a secret organisation. KD believed the organisation had blackmailed his wife and possibly his daughter into involvement with pornography and indecent images of them were being distributed across the internet, partly as a "personal vendetta" against him waged by the two leaders of the organisation. He first suspected that this might be the case when he saw his wife turn their computer off in a hurry, but believed that his wife had left a trail of clues for him to find so he could protect her from danger. The clues were concealed in the names of the websites which he believed were involved in the conspiracy against him and his family. A diagnosis of schizophrenia was made during admission to hospital. KD is a competent internet user and had regularly used email and the

web at home over the previous two years for leisure and communicating with friends and family.

#### *4.4.4.3 Case 3*

AQ, a 36 year old woman with a previous diagnosis of bipolar affective disorder, was admitted to hospital concerned that she was being “tracked by cameras” that had been placed around her house, that were transmitting images of her across radio, television and the internet. She was particularly concerned that the internet was involved, as she claimed her daughter had mentioned that she had seen photographs and video of her online. AQ had also experienced “beams of light” coming into her house which she believed were being controlled via the internet and were involved in her surveillance. After admission to the hospital ward, she voiced concerns about being watched by beams of light and was suspicious of the electric lights in her room. AQ was subsequently diagnosed as having a manic episode with psychotic symptoms. She denied ever having used the internet before, and when questioned about how she believed the internet worked she said it operated using “beams of light”. When asked in reference to a specific example (sending an email), she said that cables might also be involved.

#### *4.4.4.4 Case 4*

DS, a 19 year old male with a previous diagnosis of schizophrenia and a history of alcohol and substance abuse, was seen while resident in a hostel shortly after discharge from hospital. DS reported that he believed that the internet was being used to tell others about a past “offence” (a practical joke he had carried out on a friend), and that he was followed by “thirty to forty” people who were disgusted at his past behaviour and wanted to see him back in hospital. DS was engaged in cognitive-behavioural therapy by his therapist to lessen his

distress and conviction in his delusional beliefs. DS rated his conviction that he was being followed at 90% and his conviction that the internet was involved was 70-80%. In collaboration with the client, the therapist used a popular book about the internet and world wide web to explore how they worked, including the use of search engines. It was established that the minimum information needed to find out about someone on the internet would be a name and geographical location. Subsequently, a reality testing exercise was undertaken where people of a similar name and location to DS were identified via search engines and the information about them was examined on-screen, all of which could be confirmed as being unrelated to DS in any way. His conviction about the internet being involved in his supposed persecution dropped to 0%, causing him to report significantly less anxiety and guilt, with the number of people he supposed were involved in his persecution reduced to “ten to twenty”.

#### **4.4.5 Discussion**

The four case studies reported here are notable as differing presentations of internet-themed delusions, or as with Case 4, an example of how a delusional belief involving the internet may be resolved in light of Chadwick and Lowe’s (1990) method of reality testing by collaborative empiricism.

None of the cases reported here suggests a novel form of psychotic symptom, as in the case of ‘perception broadcast’ reported by Schmid-Siegel et al. (2004). However, Case 1 is of interest because of the individual’s self reported use of an internet search engine during the initial stages of delusion formation. It is quite possible that the content of the delusion would have been different had another search engine or method been used, as different results might have been returned, leading to the establishment of different associations. In this way, the use of

the internet may affect the aetiology of delusion formation and may promote the loosening of associations when used during a psychotic episode.

A jumping-to-conclusions probabilistic reasoning style has been identified as a factor in delusion formation (Garety, 1991; Garety and Hemsley, 1994; section 3.4.4), suggesting that even relatively small amounts of tangential information, which would not otherwise be encountered, may form the basis, or provide a conducive framework for the formation of delusional beliefs. KD from Case 1, perhaps, is a striking example of this tangential process as she managed to get from a query about 'phenylalanine' to a belief concerning a terrorist network after only a short period of time. From this case alone it is not clear whether an effect on the loosening of associations caused by using the internet in this way would cause the resulting delusion to be any more or less aberrant, leading to more extreme, or, alternatively, more benign beliefs as a result.

However, it has been noted (Bell et al., 2006; section 4.3) that people who are likely to be psychotic may use the internet to form online communities based around their delusional beliefs and archive a large corpus of online information to support their conjectures. Research on the social psychology of the internet has suggested that people who strongly identify with a group identity or cause are more likely to have their attitudes influenced to polar extremes if they are not in the same physical location (Spears et al., 1990).

Combined with the increasing availability of domestic internet access and the fact that "on the internet, people who share your interest and lean in the same direction as you are just a few keystrokes away, regardless of the issue's obscurity, social desirability, or bizarreness" (Wallace, 1999, p79), people undergoing the initial stages of psychosis may have their

incipient delusional beliefs primed, strengthened or deepened by using the internet, where previously they might have encountered very few people (if any) who would agree with their interpretations.

One way of countering potentially disabling delusional beliefs may be through the use of cognitive behavioural therapy. Both Duggal et al. (2002) and the therapist involved in Case 4 used remarkably similar techniques (although the therapist reported here was unaware of the work of Duggal et al. at the time) to implement Chadwick and Lowe's (1990) collaborative empiricism, with remarkable success in both cases. The educational aspect of the therapy, where the technological limits of the internet are explored so the client becomes aware of what the internet can and cannot do, seems important in shaking the delusional conviction that the internet is involved in their experience.

For example, AQ reported both that the internet was responsible for "beams of light" entering her house, and that the internet relies on beams of light for its operation. Although this is not entirely inaccurate (fibre optic cables provide much of the infrastructure for large computer networks) it seems in these cases that an understanding of the technology (or rather, a lack of understanding) and the delusional explanation are linked. Author and scientist Arthur C. Clarke noted that for people who do not understand it, "any sufficiently advanced technology is indistinguishable from magic" (Clarke, 1962), implying that poor understanding of a technology provides greater degrees of freedom in how it can be used in an 'explanation' for an experience or event.

Dispelling 'magical' notions about internet technology would seem to be something which could be easily and powerfully demonstrated in a therapeutic situation by a combination of

both didactic and interactive exploration of the internet. This suggests that, in cases that involve the internet as part of a delusional explanation for unusual experiences, the prognosis may be more hopeful if such therapeutic techniques are used.

As reported in Case 2, however, not all delusional beliefs about the internet may be as easily swayed in this way if they are not based on a misunderstanding of the technology. KD is notable for having a delusion about the internet which was not practically falsifiable either by education about the internet's workings or by practical use of the internet. His assertion that there are hidden sections to major websites is almost certainly accurate, as system administrators need to be able to login to restricted areas to administrate the website. In cases such as these, therapy may need to focus on the unlikely nature of the conspiracy theory (the likelihood of a sinister organisation trying to persecute him), rather than the role of technology in the delusional explanation.

It was notable that WL (Case 1) and KD (Case 2) were already competent users and, therefore, perhaps less likely to incorporate 'magical' explanations of internet function into their concerns. This suggests that the assertion of Catalano et al. (1990) that ignorance about the internet may fuel "internet delusions" per se may be wrong, although an understanding of internet technology may affect the type of delusional presentation.

The cases presented here, combined with the previous reports in the literature, suggest that the internet may not simply affect a surface change to the same "underlying biological signal" as Spence (2001) claimed, but may influence aetiology and form, as well as having implications for the clinical management of the resulting symptomology. The extent of influence may not be equal for all aspects of society and culture; although the fact that there is an influence at

all, suggests that psychosis is only fully understandable in light of the wider social context. Clinicians should be aware of the influence of the internet on delusional beliefs, as it may occur as a factor in the aetiology of delusion formation, as a feature of delusion content, and have implications for the way in which therapy is framed.

#### **4.5 Chapter summary and conclusions**

Each of the studies presented here suggests that the current conceptual definition of delusions is in need of revision. The social network analysis study of ‘mind control experiences’ (section 4.3) implies that pathological beliefs are not necessarily those that are “not accepted by other members of the person’s culture or subculture” and, furthermore, the likely delusional members of the ‘mind control’ subculture seem to show evidence of a social network (and, perhaps, social belief formation) comparable to non-delusional people. The descriptive psychopathological approach used in the second study suggests that delusion content, or indeed general theme, can have an influence of the aetiology and prognosis, and, although not demonstrated in these particularly case studies, psychopathological form (as reported by Schmid-Siegel et al., 2004).

This provides further evidence that the original Jaspersian model of delusions is becoming increasingly untenable as a basis for a scientific psychopathology, and, therefore, is not a sound basis on which to base inferences on the structure or structures of normal belief.

In light of these findings, and previous work criticising the current definition of delusions (sections 2.4; 2.5), this thesis will reject the idea that delusions are unitary encapsulated phenomena wholly described by the diagnostic criteria, and will, therefore, look for



psychological and neuropsychological attributes that contribute towards the likelihood of a belief being considered clinically significant.

Furthermore, shortcomings in the definition of delusions suggests that, unlike in the approach of traditional cognitive neuropsychology, simply comparing clinical with non-clinical participants is unlikely to be a sufficient basis for understanding either delusions or belief. Therefore, this thesis will focus on converging evidence, and will include clinical cases, the general population, and those with high levels of anomalous experience and belief who are not considered clinically significant. This will allow a comprehensive approach to delusion and belief research that is not solely based on the assumptions of mainstream clinical psychiatry.

## 4.6 References

- American Psychiatric Association (2000) *Diagnostic and Statistical Manual of Mental Disorders, ed 4, Text Revision (DSM-IV-TR)*. Washington: American Psychiatric Association.
- Bell, V., Maiden, C., Muñoz-Solomando, A. & Reddy, V. (2006) 'Mind control experiences' on the internet: Implications for the psychiatric diagnosis of delusions. *Psychopathology*, 39, 87-91.
- Bertelsen A (2002) Schizophrenia and Related Disorders: Experience with Current Diagnostic Systems. *Psychopathology*, 35, 89-93.
- Borgatti SP, Everett MG, Freeman LC (2002) *Ucinet for Windows: Software for Social Network Analysis*. Harvard: Analytic Technologies.
- Catalano G, Catalano MC, Embi CS, Frankel RL (1999) Delusions about the Internet. *Southern Medical Journal*, 92, 609-10.
- Chadwick PDJ, Lowe CF (1990) Measurement and modification of delusional beliefs. *Journal of Consulting and Clinical Psychology*, 58, 225-232.
- Clarke AC (1962) *Profiles of the Future*. London: Pan.
- Compton MT (2003) Internet delusions. *Southern Medical Journal*, 96, 61-63.

Conger AJ (1980) Integration and generalization of kappas for multiple raters. *Psychological Bulletin*, 88, 322-328.

Davies P, Lipsey Z (2003) Ana's gone surfing. *The Psychologist*, 16, 424-425.

Duggal HS, Jagadheedshan K, Nizamie H (2002) 'Internet Delusion' responsive to cognitive therapy. *Indian Journal of Psychiatry*, 44, 293-296.

Eurostat (2004) *Internet usage by individuals and enterprises 2004*. Luxembourg: Eurostat.  
Retrieved from: [http://epp.eurostat.cec.eu.int/cache/ITY\\_OFFPUB/KS-NP-05-018/EN/KS-NP-05-018-EN.PDF](http://epp.eurostat.cec.eu.int/cache/ITY_OFFPUB/KS-NP-05-018/EN/KS-NP-05-018-EN.PDF)

Eytan A, Liberek C, Graf I, Golaz J (2002) Electronic chips implant: a new culture-bound syndrome? *Psychiatry*, 65, 72-74.

Fleiss JL (1971) Measuring nominal scale agreement among many raters. *Psychological Bulletin*, 76, 378-81.

Forster PM (2004) Psychological sense of community in groups on the internet. *Behaviour Change*, 21, 141-146.

Forsyth R, Harland R, Edwards T (2001) Computer game delusions. *Journal of the Royal Society of Medicine*, 94, 184-185.

Freeman LC (1979) Centrality in Social Networks: Conceptual clarification. *Social Networks*, 1, 215-239.

Freeman SC, Freeman LC (1979) The networkers network: A study of the impact of a new communications medium on sociometric structure. *Social Science Research Reports No 46*. Irvine CA: University of California.

Frey AH (1962) Human auditory system response to modulated electromagnetic energy. *Journal of Applied Physiology*, 17, 689-692.

Garety P (1991) Reasoning and delusions. *British Journal of Psychiatry Supplement*, 14, S14-18.

Garety PA, Hemsley DR (1994) *Delusions: Investigations in the Psychology of Delusional Reasoning*. Oxford, Oxford University Press.

Gilleen, J., David, A. S. (2005) The cognitive neuropsychiatry of delusions: from psychopathology to neuropsychology and back again. *Psychological Medicine*, 35, 5-12.

Gillmor D (2004) *We The Media*. Sebastopol CA: O'Reilly Books.

Glass J, Campbell TG (1984) Rock and roll delusions. *British Journal of Psychiatry*, 145, 95-96.

Goodman LA (1961) Snowball sampling. *Annals of Mathematical Statistics*, 32, 148-170.

Griffiths M (1996) Internet "addiction": an issue for clinical psychology? *Clinical Psychology Forum*, 97, 33-36.

Haslam J, Porter R: (1989) *Illustrations of Madness*. London: Routledge Kegan & Paul.

Hewson C (2003) Conducting research on the internet. *The Psychologist*, 16, 290-293.

Jackson MH (1997) Assessing the structure of communication on the world wide web. *Journal of Computer-Mediated Communication*, 3. Retrieved 8<sup>th</sup> August, 2005 from <http://jcmc.indiana.edu/vol3/issue1/jackson.html>

Jaspers, K. (1923) *General Psychopathology (7th edn)*. Translated by J. Hoenig & M. Hamilton. Manchester: Manchester University Press, 1969.

Jay M (2003) *The Air Loom Gang: The Strange and True Story of James Tilly Matthews and His Visionary Madness*. London: Random House.

Kala AK, Wig NN (1992) Delusions across cultures. *International Journal of Social Psychiatry*, 28, 185-193.

Killworth B, Bernard H (1976) Informant accuracy in social network data. *Human Organization*, 35, 269-286.

Kim K, Li D, Jiang Z, Cuix X, Lin L, Kang J (1993) Schizophrenic delusions among Koreans, Korean-Chinese and Chinese: A transcultural study. *International Journal of Social Psychiatry*, 39, 190-199.

Kobayashi T, Okada Y, Nisijima K, Kato S: "Internet delusion" in a patient with a schizoaffective disorder. *Canadian Journal of Psychiatry*, 46, 89-90.

Leeser J, O'Donohue W (1999) What is a delusion? Epistemological dimensions. *Journal of Abnormal Psychology*, 108, 687-694.

Light E (2003) Mind Control Forum Home Page. Retrieved June 4, 2003 from <http://www.mindcontrolforums.com/>

Lusseau D (2003) The emergent properties of a dolphin social network. *Proceedings of the Royal Society of London: Biology Letters*, Supplement 2, S186-S189.

Margolese HC, Chouinard G, Beauclair L, Miller R (2002) Using the rating scale for psychotic symptoms to characterize delusions expressed in a schizophrenia patient with "Internet psychosis". *Canadian Journal of Psychiatry*, 47, 485.

Myin-Germeys I, Nicolson NA, Delespaul PA (2001) The context of delusional experiences in the daily life of patients with schizophrenia. *Psychological Medicine*, 31, 489-498.

O'Grady JC (1990) The prevalence and diagnostic significance of Schneiderian first-rank symptoms in a random sample of acute psychiatric in-patients. *British Journal of Psychiatry*, 156, 496-500.

Park HW (2003) Hyperlink network analysis: A new method for the study of social structure on the web. *Connections*, 25, 49-61.

Park HW, Thelwall M (2003) Hyperlink Analyses of the World Wide Web: A Review, *Journal of Computer-Mediated Communication*, 8. Retrieved 8<sup>th</sup> August 2005 from <http://jcmc.indiana.edu/vol8/issue4/park.html>

Podoll K, Habermeyer E, Noller B, Ebel H, Sass H (2000) Internet als Wahnthema bei paranoider Schizophrenie [The internet as a delusional topic in paranoid schizophrenia]. *Nervenarzt*, 71, 912-914.

Robinson AJ (1984) Rock and roll delusions. *British Journal of Psychiatry*, 145, 672.

Ruedrich SL, Bishop RJ, Chu C (1983) Rock and roll music in delusion formation. *Journal of Operational Psychiatry*, 14, 115-117.

Salleh MR (1992) Specificity of Schneider's first rank symptoms for schizophrenia in Malay patients. *Psychopathology*, 25, 199-203.

Schmid-Siegel B, Stompe T, Ortwein-Swoboda G (2004) Being a webcam. *Psychopathology*, 37, 84-85.

Schneider K (1959) *Clinical Psychopathology*. New York: Grune & Stratton.

Sims A (2003) *Symptoms in the Mind: An Introduction to Descriptive Psychopathology* (3<sup>rd</sup> ed). London: Elsevier Science Ltd.

Shapira NA, Lessig MC, Goldsmith TD, Szabo ST, Lazoritz M, Gold MS, Stein DJ (2003) Problematic internet use: proposed classification and diagnostic criteria. *Depression and Anxiety*, 17, 207-216.

Spears R, Lea M, Lee S (1990) De-individuation and group polarization in computer-mediated communication. *British Journal of Social Psychology*, 29, 121-134.

Spence, SA (2001) Computer game delusions. *Journal of the Royal Society of Medicine*, 94, 369.

Spitzer M (1990) On defining delusions. *Comprehensive Psychiatry*, 31, 377-397.

Spitzer RL, First MB, Kendler KS, Stein DJ (1993) The reliability of three definitions of bizarre delusions. *American Journal of Psychiatry*, 150, 880-884.

Stompe T, Friedman A, Ortwein G, Strobl R, Chaudhry HR, Najam N, Chaudhry MR (1999) Comparison of delusions among schizophrenics in Austria and in Pakistan. *Psychopathology*, 32, 225-234.



- Stompe T, Ortwein-Swoboda G, Ritter K, Schanda H (2004) Old wine in new bottles? Stability and plasticity of the contents of schizophrenic delusions. *Psychopathology*, 36, 6-12.
- Suhail K (2003) Phenomenology of delusions in Pakistani patients: effect of gender and social class. *Psychopathology*, 36, 195-9.
- Suhail K, Cochrane R (2002) Effect of culture and environment on the phenomenology of delusions and hallucinations. *International Journal of Social Psychiatry*, 48, 126-38.
- Tan S, Shea C, Kopala L (1997) Paranoid schizophrenia with delusions regarding the Internet. *Journal of Psychiatry and Neuroscience*, 22, 143.
- Tateyama M, Asai M, Kamisada M, Hashimoto M, Bartels M, Heimann H. (1993) Comparison of schizophrenic delusions between Japan and Germany. *Psychopathology*, 26, 151-158.
- Tausk V (1933) On the origin of the 'Influencing Machine' in schizophrenia. *Psychoanalytical Quarterly*, 2, 519-556.
- U. S. Senate Select Committee on Intelligence (1977) *Project MKULTRA, the CIA's program of research in behavioral modification. Ninety-fifth Congress, first session, August 3, 1977.* Washington, U.S: GPO.
- Wallace, P. (1999) *The Psychology of the Internet*. Cambridge: Cambridge University Press.

Wasserman S, Faust K (1994) *Social network analysis: Methods and applications*.  
Cambridge, Cambridge University Press.

Watts DJ, Strogatz SH (1998) Collective dynamics of 'small-world' networks. *Nature*, 393,  
440-442.

Wellman B (2001) Computer networks as social networks. *Science*, 293, 2031-2034.

Zachary W (1977) An information flow model for conflict and fission in small groups.  
*Journal of Anthropological Research*, 33, 452-473.

## Chapter Five

### **The Cardiff Anomalous Perceptions Scale (CAPS) and the Role of Anomalous Perceptual Experience in Delusion Formation**

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The following presentations have also included material from this chapter:

Bell, V., Halligan, P.W., Dietrich, C., Ellis, H.D. Aspects of the psychosis continuum and the Cardiff Anomalous Perceptions Scale (CAPS): Are there multiple factors underlying anomalous experience? *British Association for Behavioural and Cognitive Psychotherapies Annual Conference*, Canterbury, July 2005.

## 5.1 Chapter outline

Sections:

- 5.2 Abstract
- 5.3 Background
- 5.4 Method
- 5.5 Results
- 5.6 Discussion
- 5.7 Chapter summary and conclusion
- 5.8 References

As highlighted in Chapter 3, anomalous perceptual experience plays a crucial role in many delusion formation theories. Indeed, for both ‘one-stage’ and ‘two-stage’ (sections 3.2.1; 3.2.2) theories, perceptual distortions are seen as a necessary condition.

Very little work, however, has focused on investigating this link in a principled and robust manner. This may, in part, be due to the fact that anomalous perceptual experience and the psychiatric concept of hallucination or illusion are often used synonymously, with little critical insight into the assumptions underlying these medical constructs.

This is reflected in the current psychometric scales, which, despite their roots in the dimensional tradition, are still wedded to many problematic assumptions and preconceptions about how perceptual distortions present.

The lack of a psychometric scale, able to assess anomalous perceptual experience in a ‘clinically neutral’ manner, makes it difficult to investigate adequately the role of anomalous

perceptual experience in delusion formation, without including concepts that might bias or presuppose the outcome.

To address this issue, this chapter describes the development of the Cardiff Anomalous Perceptions Scale (CAPS), and its deployment in an investigation into the role of anomalous perceptual experience in delusion formation and delusional ideation.

As well as investigating these *a priori* hypotheses, a principal components analysis was conducted to determine any emergent themes in the presentation of anomalous perceptual experience. This was to examine whether the contribution of anomalous perceptual experience to the proposed 'continuum model of psychosis' (2.4.4) was the likely result of a single underlying factor, or whether there may be multiple contributory aspects.

## **5.2 Abstract**

The study describes the Cardiff Anomalous Perceptions Scale (CAPS), a new validated measure of perceptual anomalies. The 32 item CAPS measure is a reliable, self-report scale which uses neutral language and demonstrates high content validity. The CAPS was completed by a general population sample of 336 participants and 68 psychotic inpatients. A principal components analysis of the general population data revealed three components: 'clinical psychosis' (largely Schneiderian first-rank symptoms), 'temporal lobe disturbance' (largely related to temporal lobe epilepsy and related seizure-like disturbances) and 'chemosensation' (largely olfactory and gustatory experiences), suggesting that there are multiple contributory factors underlying anomalous perceptual experience and the 'psychosis continuum'. Odds analyses on insight-related item categories suggested that being able to label an experience as a non-shared sensory experience most distinguished non-clinical from

clinical groups. No significant difference was found between general population participants and deluded inpatients without hallucinations, suggesting, contrary to ‘one-stage theories’, pathological levels of anomalous experience are neither necessary nor sufficient for delusion formation.

### **5.3 Background**

As a general label for a range of symptoms associated with severe mental illness, ‘psychosis’ is typically characterised as a “loss of contact with reality”. Although lacking a consistent operational definition, one of the most problematic aspects of the term, as traditionally employed, is its assumed categorical nature. In contrast to the traditional categorical approach to psychosis adopted by the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR; APA, 2000), there is growing interest in a more dimensional view, that argues that psychosis-like beliefs, perceptual distortions and idiosyncrasies of thought and communication, considered the hallmark diagnostic criteria for psychosis, are distributed (albeit to varying degrees) throughout the general population.

Such an approach considers florid psychosis as representing the most extreme pole of the population spectrum (Johns and van Os, 2001; Peters et al., 1999; Peters et al., 2004; van Os, 2003; Verdoux and van Os, 2002). The view that psychotic manifestations may exist on a continuum (section 2.5.3), rather than as a discrete entity, however, is not new. In contrast to the more popular Kraepelinian view, Bleuler, and later others, argued throughout the twentieth century against a clear separation between sanity and madness (Bleuler, 1911; Laing, 1960; Meehl, 1962; Rado, 1953; Strauss, 1969).

The development of psychometric measures that have attempted to capture the continuum of psychosis and psychosis-like experience has facilitated this non-categorical view. The focus for such scales, however, has varied, with some aiming to measure a general psychosis-proneness, while others have focused on particular aspects of the psychosis continuum (such as delusional ideation or hallucination proneness) influenced by the symptom boundaries of clinical psychiatry.

One of the earliest attempts to capture a general concept of psychosis-proneness was Eysenck's inclusion of the psychoticism dimension as an aspect of personality (Eysenck, 1952; Eysenck and Eysenck, 1976). Adopting a personality-theory standpoint, Eysenck aimed to capture psychosis-proneness on a dimensional construct varying from normality (necessarily defined in culturally relative terms) and psychosis. This was subsequently developed into a multidimensional concept of schizotypy (Claridge et al., 1996), based on a factor analysis of various psychosis-proneness scales (Bentall et al., 1989) which has been developed into the 'unusual experience', 'cognitive disorganisation', 'introverted anhedonia' and 'impulsive nonconformity' subscales of the Oxford and Liverpool Inventory of Feelings and Experiences (O-LIFE) schizotypy scale (Mason et al., 1995).

In contrast to this approach, most other measures of 'psychosis-proneness' are grounded in clinical psychiatry and aimed at measuring attenuated or 'soft' psychotic symptoms in the general population. Of particular relevance, understandably, are those measures that attempt to quantify aspects of the 'positive symptoms' of psychosis, such as delusions and hallucinations.

The Magical Ideation Scale by Eckblad and Chapman (1983) covers a range of beliefs and experiences from first rank symptoms of schizophrenia (Schneider, 1959) and ideas of reference, to popular paranormal and conspiracy theory themes (e.g. “The government refuses to tell us the truth about flying saucers”). The Peters et al. Delusions Inventory (PDI; Peters et al., 1999; 2004) is a measure of delusional ideation that enquires about beliefs, interpretations and experiences, which uses items derived from the Present State Examination (Wing et al., 1974), an internationally-recognised clinical measure, often used to detect and assess clinically-defined psychotic symptoms. The PDI, however, is unique, in that it not only measures the total number of beliefs or experiences endorsed, but also the concurrent perceptions of distress, preoccupation and conviction associated with the endorsed items.

Other measures have focused on perceptual and hallucinatory experiences associated with psychosis. The Perceptual Aberration Scale (Chapman et al., 1978) measures the level of body-image aberration, with items based on experiences of somatic distortions and hallucinations as reported in the clinical literature on schizophrenia and associated diagnoses. Morrison, Wells and Nothard created and revised the Launay-Slade Hallucinations Scale (RLSHS; Morrison et al., 2000; 2002) to measure predisposition to hallucinations, in an attempt to capture some clinically-recognisable hallucinatory phenomena (such as ‘hearing voices’ and having non-veridical visual experiences), as well as any tendency to have vivid imagery and daydreams.

The Structured Interview for Assessing Perceptual Anomalies (SIAPA; Bunney et al., 1999) is one alternative assessment method that does not rely on self-report. Although it aims to be comprehensive in its coverage of the ‘five senses’, it is designed as an interview-based assessment of the frequency of sensory anomalies and, therefore, has the disadvantage of



being time consuming and requiring one-to-one assessment. It also is restricted in that, unlike some of the psychosis-inspired scales mentioned above, it does not assess hallucinatory phenomena directly, but instead focuses on changes in sensory intensity, attention, and sensory flooding. It is clear that a measure is needed to assess the range of perceptual anomalies not covered by any single existing scale.

This issue is particularly pertinent when the ongoing debate over the role of anomalous perceptual experience in delusion formation is considered. Hallucinatory experiences have traditionally been associated with delusions and delusional ideation across the range of major psychotic disorders (Peralta et al., 1992). They have also been shown to be associated with paranormal beliefs (Thalbourne, 1994) and non-clinical measures of delusional ideation (Verdoux et al., 1998); although the exact causal relationship between anomalous experience and unusual belief remains unclear.

This is, perhaps, partly due to the confusion between hallucinations as diagnosed in clinical practice, and perceptual distortions in general, which may include experiences which do not fulfil the typical definition of an hallucination as a false sensory perception in the absence of an external stimulus (Sims, 2003). Hallucinations and delusions, although correlated, are known to occur separately, as enshrined in the diagnostic criteria for ‘delusional disorder’ (APA, 2000). Current theories of delusion, however, typically include the wider concept of perceptual anomaly, rather than frank hallucination, as a necessary condition in delusion formation.

As indicated in section 3.2.1, Maher (1988; 1999) has consistently argued for a one-stage account of delusion formation, where delusions are the product of normal reasoning processes

applied to anomalous perceptual experience, whereas other authors have stressed the need for a ‘two-stage’ account of delusion formation, where a bias or deficit in reasoning has to be also involved (Ellis and Young, 1990; Ellis, 1998; Stone and Young, 1997; section 3.2.2).

According to these accounts, anomalous perceptual experiences are not considered sufficient in themselves to account for the presence of delusions, since participants other than patients report such experiences without seeking psychiatric intervention. Many of the psychometric measures of anomalous perceptual experience, however, derive both their content and language from mainstream clinical psychiatry (which depends on frank and often chronic forms of mental illness), and it is apparent that they may lack face validity when trying accurately to assess the full range of perceptual anomalies in the general population.

These biases can make perceptual and cognitive distortions difficult to tease apart adequately. Several of the scales are not ‘pure’ measures of perceptual anomaly (although deliberately so in many cases). For example, the Launay-Slade Hallucinations Scale, despite its name, conflates items concerning both perceptual experience (e.g. “I hear the telephone ring and find that I am mistaken”) and delusional ideation (e.g. “I fantasise about being someone else”) into a single measure.

There is also an implicit assumption in some scales, that respondents are able to distinguish between experiences that stem from perceptions that exist out in the ‘real world’ and those that may arise from distortions with the respondent’s own cognitive processes – that is, those that are considered ‘not to be really there’, as illustrated by this item from the O-LIFE, “when in the dark, do you often see shapes and forms even though there’s nothing there?”.

Other measures rely on a related concept of strangeness or unusualness (for example, “When I look at things they appear strange to me”, from the RLSHS) that presupposes that a non-veridical perceptual experience will necessarily present as strange or anomalous.

Both assumptions are potentially problematic “since virtually all waking perceptual experiences are veridical, a long personal history of validated perception would dictate accepting hallucinations as veridical” (Sackeim, 1998, p9). Of course, it may be that perceptual anomalies are accompanied by insight into their unusual nature, but it is important that this is not the only criterion by which such anomalies are measured. In fact, there may be several indicators that a perceptual experience is not veridical for an individual, including those that may arise without a clear source, those that do not seem to be shared by other people in the vicinity and those that are accompanied by a sense of strangeness.

Another drawback of assessing perceptual anomalies by extrapolating exclusively from the context of clinical psychiatry is over reliance on hallucinatory phenomena that occur in the visual and auditory modalities. For example, surveys of hallucinatory phenomena in the general population indicate that olfactory and gustatory hallucinations are particularly common (Ohayon, 2000), yet these modalities are rarely explored in psychometric measures of hallucination or psychosis-proneness. Likewise, alterations in sensory intensity, rather than experiencing discrete perceptual phenomena, are not normally covered by existing scales.

Another legacy of clinical psychiatry is the lack of coverage of perceptual anomalies associated with temporal lobe disturbance, despite the fact that temporal lobe disturbance has been linked to almost every ‘stage’ on the psychosis continuum, from full blown psychosis (Maier et al., 2000; Toone et al., 1982), to paranormal beliefs and experiences (Persinger and

Makarec, 1987) and anomalous perceptual phenomena in non-clinical participants (Persinger and Healey, 2002). Thus, there is a need for a comprehensive scale capable of measuring a range of sensory experience, covering both clinical and non-clinical populations. As highlighted earlier, this is of particular relevance for the debate over the necessity of anomalous perceptual experience in delusion formation.

Consequently, the aim in creating the Cardiff Anomalous Perceptions Scale was to devise a valid and reliable psychometric measure of perceptual anomalies that is not dependent on the clinical psychiatric context and that considers subjective experiences from a range of different perspectives of insight awareness (including knowing that the percept is 'not really there', the percept seeming strange or unusual, or the percept being a non-shared sensory experience). Moreover, the CAPS includes items pertaining to distortions in perceptual intensity, items tapping experiences in all appropriate sensory modalities, and sensory experiences traditionally associated with temporal lobe disturbances. Following the usefulness of their inclusion in the PDI (Peters et al., 1999; 2004) dimensional ratings to measure associated distress, intrusiveness and frequency for each experience endorsed were also included.

## 5.4 Method

### 5.4.1 Construction of the CAPS

#### 5.4.1.1 Item selection.

Measures related to psychosis-proneness, hallucination-proneness, clinical assessment of psychosis, delusional and magical ideation and hallucinatory experience, including temporal lobe disturbance, were collected and reviewed (see table 5.1), and all items relating to sensory experience were considered.

<i>Scale or assessment</i>	<i>Authors</i>
Present State Examination	Wing et al. (1974)
Magical ideation scale	Eckblad and Chapman (1983)
Makarec and Persinger Temporal Lobe Scale	Makerac and Persinger (1985)
Scale for the Assessment of Positive Symptoms	Andreasen (1994)
Psychosis screening questionnaire	Bebbington and Nayami (1995)
O-LIFE schizotypy scale	Mason et al. (1995)
Structured Interview for Assessing Perceptual Anomalies	Bunney et al. (1999)
Peters et al. Delusions Inventory	Peters et al. (1999)
Revised Launay-Slade Hallucinations Scale	Morrison et al. (2002)

*table 5.1 Scales reviewed in construction of CAPS items.*

To focus particularly on anomalous perceptual experience, rather than other more general aspects of schizotypy or psychosis-like experience or proneness, experiences relating to experiences of thought broadcast, insertion, blocking, and interference were excluded unless they had been subsequently experienced via one of the senses (e.g. 'hearing thoughts out loud'). Similarly, any experiences relating to dissociation, depersonalisation or existential feelings of strangeness or unease (e.g. 'sometimes everything around me feels strange') were also excluded, as were those specifically related to hypnopompic, hypnagogic or other sleep-related states such as dreaming, to exclude any experiences that may not have occurred in clear consciousness.

<i>Selection category</i>	<i>CAPS Items</i>
Changes in levels of sensory intensity (Relevant domains: Sight, Sound, Taste, Touch, Smell)	1) Do you ever notice that sounds are much louder than they normally would be ? 18) Do you ever smell everyday odours and think that they are unusually strong ? 20) Do you ever find that your skin is more sensitive to touch, heat or cold than usual ? 21) Do you ever think that food or drink tastes much stronger than it normally would ? 23) Do you ever have days where lights or colours seem brighter or more intense than usual ?
Having a non-shared sensory experience (Relevant domains: Sight, Sound, Smell)	13) Do you ever hear voices saying words or sentences when there is no-one around that might account for it ? 29) Do you ever experience smells or odours that people next to you seem unaware of ? 31) Do you ever see things that other people cannot ? 32) Do you ever hear sounds or music that people near you don't hear ?
Inherently unusual or distorted (Relevant domains: Sight, Sound, Taste, Touch, Smell)	5) Do you ever experience unusual burning sensations or other strange feelings in or on your body ? 16) Do you ever find that sounds are distorted in strange or unusual ways ? 25) Do you ever find that common smells sometimes seem unusually different ? 26) Do you ever think that everyday things look abnormal to you ? 30) Do you ever notice that food or drink seems to have an unusual taste ?
Sensory experience from an unexplained source (Relevant domains: Sight, Sound, Taste, Touch, Smell)	4) Do you ever see shapes, lights or colours even though there is nothing really there ? 6) Do you ever hear noises or sounds when there is nothing about to explain them ? 8) Do you ever detect smells which don't seem to come from your surroundings ? 12) Do you ever feel that someone is touching you, but when you look nobody is there ? 14) Do you ever experience unexplained tastes in your mouth ? 28) Have you ever heard two or more unexplained voices talking with each other ?
Distortion of form (size, shape) of own body and of external world	9) Do you ever have the sensation that your body, or a part of it, is changing or has changed shape ? 10) Do you ever have the sensation that your limbs might not be your own or might not be properly connected to your body? 19) Do you ever find the appearance of things or people seems to change in a puzzling way, e.g. distorted shapes or sizes or colour ? 22) Do you ever look in the mirror and think that your face seems different from usual ?
Verbal hallucinations	11) Do you ever hear voices commenting on what you are thinking or doing ? 13) Do you ever hear voices saying words or sentences when there is no-one around that might account for it ? 28) Have you ever heard two or more unexplained voices talking with each other ?
Sensory flooding	15) Do you ever find that sensations happen all at once and flood you with information ? 17) Do you ever have difficulty distinguishing one sensation from another ?
Thought echo and hearing thoughts out loud	3) Do you ever hear your own thoughts repeated or echoed ? 7) Do you ever hear your own thoughts spoken aloud in your head, so that someone near might be able to hear them ?
Temporal Lobe	2) Do you ever sense the presence of another being, despite being unable to see any evidence ? 10) Do you ever have the sensation that your limbs might not be your own or might not be properly connected to your body? 24) Do you ever have the feeling of being uplifted, as if driving or rolling over a road while sitting quietly ? 27) Do you ever find that your experience of time changes dramatically ?

Note. Questions may appear in more than one category.

*table 5.2 CAPS items broken down by pre-selected category of anomalous experience*

Relevant items were generated from candidate experiences and items that repeated or substantially overlapped with other items were removed, with further items created to cover

additional sensory modalities where necessary. The final CAPS items, are listed by category in table 5.2. In the final scale, each item is presented as a question requiring an answer of 'yes' or 'no', with the participants required simply to rate the item for distress, intrusiveness and frequency of occurrence on a 5 point (1 - 5) Likert scale if they had responded with a 'yes' to the initial question. A copy of the full scale is included in Appendix I.

## **5.4.2 Participants and procedure**

### *5.4.2.1 Controls*

A total of 358 participants from a non-clinical population participated in the study. Excluded from the study were 22 participants who incorrectly completed or missed items on questionnaires, leaving a total of 336 participants included in the final analysis (mean age = 21.6, SD = 5.4, range 18 - 54). Participants were largely drawn from undergraduate students ( $N = 305$ ) including 111 males and 176 females (not disclosed = 18) with a mean age 19.9 (SD = 2.6; range 18 - 44; not disclosed = 13), who took part as part of their induction programme or who responded to requests for participants.

The remaining 32 participants were drawn from an anonymous postal survey. They responded to advertisements posted on general purpose internet discussion groups requesting participants for a study on 'beliefs and experiences', with no reference to the specific aims of the study in the original advertisement or any of the supporting material. This sample consisted of 17 females and 14 males (not disclosed = 1) with a mean age of 32.4 (SD = 10.2; range 18 - 54; not disclosed = 1).

All participants completed the CAPS and the 21-item Peters et al. Delusions Inventory (PDI-21; Peters et al., 2004; Peters and Garety, 1996). The Revised Launay-Slade Hallucinations

Scale (RLSHS; Morrison et al., 2000) was completed by a subset of 288 individuals and the O-LIFE schizotypy scale (Mason et al., 1995) by a subset of 184 participants. The smaller number of participants completing these latter questionnaires was due to their not being included in the early phases of the study.

Six months after initially completing the CAPS, undergraduate students were invited to complete the scale again, of whom 44 participated (males = 7; females = 37). This allowed a test-retest reliability assessment to be carried out.

#### *5.4.2.2 Clinical sample*

In addition to the non-clinical sample, a sample of 68 psychotic inpatients completed the CAPS, consisting of 33 females and 35 males (mean age 39.72, SD = 10.2, range 18 – 65), each of whom was categorised into one of three groups:

- i) inpatients with unselected psychotic symptoms ( $N = 20$ )  
diagnoses of: schizophrenia = 9; schizoaffective disorder = 1; bipolar disorder = 6;  
psychotic depression = 2; delusional disorder = 1; unspecified psychosis = 1;
- ii) deluded inpatients with hallucinations ( $N = 24$ )  
diagnoses of: schizophrenia = 19; schizoaffective disorder = 2; bipolar disorder = 2;  
unspecified psychosis = 1.
- iii) deluded patients without hallucinations ( $N = 24$ )  
diagnoses of: schizophrenia = 14; schizoaffective disorder = 5; bipolar disorder = 2;  
psychotic depression = 2; unspecified psychosis = 1.



The data from inpatients with unselected psychotic symptoms were collected from four acute psychiatric admission wards in the Cardiff area. Patients were selected on the basis of having been diagnosed with a current psychotic episode by the responsible clinician. Patients in this sample were screened with the Psychosis Screening Questionnaire (Bebbington and Nayani, 1995) to confirm the clinical classification. Out of an original sample of 22 inpatients, two were excluded due to screening negative on the PSQ, leaving 20 inpatients from this sample.

CAPS data from a further 48 deluded psychiatric inpatients were collected and provided by Nicola Smedley and Dr Emmanuelle Peters (Department of Psychology, Institute of Psychiatry, London) from inpatients in acute wards ( $N = 19$ ), inpatients in rehabilitation wards ( $N = 20$ ) and service users in residential rehabilitation housing in the South London and Maudsley NHS Trust area ( $N = 9$ ).

Of these 48 inpatients, 24 were diagnosed as being currently deluded with hallucinations and 24 were diagnosed as being currently deluded without hallucinations. Diagnoses was originally made by the responsible clinician, and confirmed by administration of the Scale for the Assessment of Positive Symptoms (SAPS; Andreasen, 1994). Participants in the group of deluded patients with hallucinations had a mean hallucinations subscale score of 1.98 ( $SD = .88$ , range 2 - 5), and a mean delusions subscale score of 1.82 ( $SD = .71$ , range 2 - 5), participants in the group of deluded patients without hallucinations had a mean hallucinations subscale score of .1 ( $SD = .19$ , range 0 - 1), and a mean delusions subscale score of 1.22 ( $SD = .66$ , range 2 - 5).

Participants in all clinical samples were referred by the responsible clinician as being without a history of brain injury, substance or alcohol abuse, and, except for one patient, they were all

on a medication regime at the time of testing. This was confirmed by review of the clinical notes. The CAPS was completed with the experimenter present to assist with any difficulties in reading or comprehension.

#### *5.4.2.3 Potential confounds*

As can be seen from the descriptions of the participant samples, there was a small imbalance in the numbers of male and female participants between the clinical and non-clinical samples, although the distribution was not significantly different from chance when tested with a chi-square test ( $\chi^2 = 3.415, p = .078$ ). There was, however, a significant difference in age between the samples when tested with an independent samples t-test ( $t = -20.89, p < .0005$ ) with the non-clinical sample comprising significantly younger participants than the clinical sample. Although the age differences in the samples is likely to have affected the results, it does not significantly affect the main conclusions drawn from this study, however, as age is inversely associated with psychotic symptoms in adults, in both clinical and non-clinical populations (Gonzalez-Pinto et al., 2004; van Os et al., 2000).

## **5.5 Results**

### **5.5.1 Psychometric properties**

Four separate scores were obtained from the CAPS: (i) total number of items endorsed; (ii) a distress score; (iii) an intrusiveness score; and (iv) frequency of occurrence. A total score was calculated by summing the number of items endorsed.

For each item endorsed, participants were required to rate the item on 1-5 scales for distress, intrusiveness and frequency. The total scores for these dimensions were calculated by summing the ratings for all endorsed items, with non-endorsed items considered to have a

score of 0 in each of these three categories. Therefore, the possible range for the CAPS total was 0 (low) to 32 (high) , and for each of the dimensions the possible range was 0 to 160.

Descriptive statistics for the CAPS, PDI-21, RLSHS and O-LIFE schizotypy scale are given in tables 5.3.1 and 5.3.2. The sum of male and female participants does not add up to the total sample, owing to participants occasionally not disclosing their gender. As can also be seen from these tables, there was a significant difference between males and females on all dimensions and total score on the CAPS and on the RLSHS, with males scoring significantly higher.

Scale (Total N)	CAPS (337)				PDI-21 (337)			
Male N / Female N	125 / 193				125 / 193			
Subscale	Total	Distress	Intr.	Freq.	Total	Distress	Preoc.	Conv.
Males, mean	9.3	18.3	21.9	18.2	6.2	15.0	15.2	18.2
(SD)	(6.3)	(15.3)	(17.6)	(15.3)	(4.0)	(12.6)	(12.3)	(13.9)
Females, mean	6.3	14.0	16.1	12.8	5.0	12.2	11.6	14.7
(SD)	(5.3)	(14.0)	(16.5)	(13.4)	(3.0)	(8.4)	(8.4)	(10.5)
Total, mean	7.3	15.5	18.0	14.6	5.4	13.2	12.9	15.9
(SD)	(5.8)	(14.5)	(17.0)	(14.2)	(3.4)	(10.2)	(10.1)	(11.9)
Range	0 - 26	0 - 81	0 - 81	0 - 77	0 - 19	0 - 59	0 - 51	0 - 69
Median	6	12	13	11	5	11	11	14
Mode	1	0	0	0	4	11	6	11
Gender, Z*	-4.31†	-2.73†	-3.31†	-3.68†	-2.41†	-1.08	-1.97†	-2.05‡

Intr. = Intrusiveness; Freq. = Frequency; Preoc. = Preoccupation; Conv. = Conviction.

\*Mann Whitney U test (two tailed); † Significant at  $p < .01$ ; ‡ Significant at  $p < .05$

table 5.3.1

*Descriptive statistics for CAPS, PDI-21, OLIFE and RLSHS in non-clinical sample part 1*

Scale (Total N)	OLIFE (184)				RLSHS (288)
Male N / Female N	130 / 24				114 / 157
Subscale	UE	CD	IA	IN	-
Males, mean	5.1	12.6	7.3	10.7	41.5
(SD)	(6.3)	(4.5)	(3.1)	(3.5)	(11.1)
Females, mean	5.9	11.8	7.2	9.3	36.7
(SD)	(5.3)	(4.2)	(3.0)	(2.7)	(7.5)
Total, mean	5.7	12.1	7.4	9.7	38.6
(SD)	(5.4)	(4.4)	(3.3)	(3.0)	(9.3)
Range	0 - 30	3 - 25	2 - 19	4 - 19	18 - 78
Median	4	12	7	10	37
Mode	1	14	6	10	33
Gender, Z*	-0.92	-0.70	-0.27	-1.91	-3.46†

UE = Unusual experiences, CD = Cognitive Disorganisation, IA = Introvertive anhedonia, IN = Impulsive non-conformity. \*Mann Whitney U test (two tailed); † Significant at  $p < .01$ ; ‡ Significant at  $p < .05$

*table 5.3.2*

*Descriptive statistics for CAPS, PDI-21, OLIFE and RLSHS in non-clinical sample part 2*

This contrasts with the results from the original study on the 40-item PDI (Peters et al., 1999) where no significant difference were reported between male and female scores. In this study, males scored significantly more on PDI-21 total score, preoccupation and conviction dimensions. The sex differences in psychosis-proneness reported here, although not consistent with the original PDI study, may be due to the preponderance of younger participants in the non-clinical sample. This finding is consistent with the results reported by Spauwen et al. (2003), who found that psychosis-like experiences were more prevalent in males under 21 years than females of the same age. This was thought to reflect the increased vulnerability to psychosis in younger males (Hafner et al., 1992).

CAPS total score showed a left-skewed frequency distribution in the non-clinical population (figure 5.1). Johns and van Os (2001) previously noted a difference between the normal and left-skewed 'half-normal' distribution of measures of psychosis-like experience, and argued

that a half-normal distribution could result from various causes contributing independently but interacting when expressed. They further suggest that this distribution is the most likely a reflection of the 'real' distribution of psychosis, suggesting that (on their criteria at least) the CAPS distribution is a statistically accurate reflection of the proposed continuum of psychosis distribution.

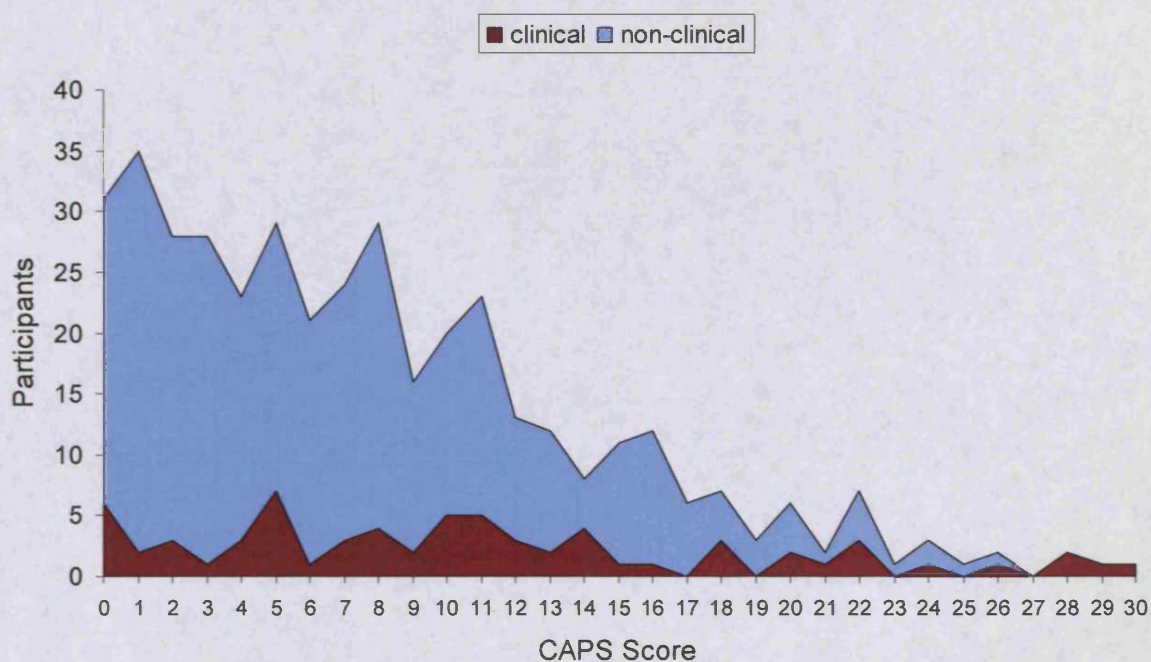


figure 5.1 frequency distribution of CAPS total score for clinical and non-clinical samples.

Figure 5.1 also illustrates that the distribution of CAPS total score from the pooled clinical sample cut across the entire range of CAPS scores and overlaps considerably with the distribution of scores from the non-clinical population. For CAPS total score, 11.3% of the non-clinical sample score above the unselected psychotic mean. Similar patterns have been found with other measures of psychosis-proneness and psychosis-like experience with either participants in the healthy control group endorsing items usually associated with clinical psychosis, or patients with psychosis scoring less than members of the non-clinical population (Bentall and Slade, 1985; Bunney et al., 1999; Peters et al., 1999; 2004).

### 5.5.2 Reliability

Internal reliability of the CAPS was good, with a Cronbach's Alpha coefficient of .87. Test-retest reliability was determined from the group of 44 participants who completed the CAPS a second time, after a six month gap, and was also found to be acceptable for all CAPS measures when tested with Pearson correlations: CAPS Total score = 0.77 ( $p < .0005$ ); CAPS distress score = 0.779 ( $p < .0005$ ); CAPS intrusiveness score = 0.783 ( $p < .0005$ ); CAPS frequency score = 0.778 ( $p < .0005$ ). The standard error of measurement for the CAPS total score, therefore, can be calculated as 1.34, showing a low margin of error when measuring the hypothetical true score (Clarke-Carter, 1997). The Cronbach's Alpha coefficient of the test-retest sample was .92, demonstrating that internal reliability remained stable over time.

### 5.5.3 Validity

Construct validity was assessed by correlating CAPS Total score with the RLSHS, OLIFE subscales and PDI-21. Pearson correlation co-efficients between each scale are given in table 5.4. The CAPS total score shows significant positive correlations with the PDI-21, RLSHS and OLIFE unusual experiences (UE) subscale, suggesting good convergent validity. In particular, the small or non-significant correlations with the other subscales of the OLIFE schizotypy scale demonstrates good discriminant validity, suggesting that the CAPS is largely selective in tapping perceptual anomalies, rather than measuring schizotypy in general.

Scale	PDI-21	OLIFE UE	OLIFE CD	OLIFE IA	OLIFE IN	RLSHS
CAPS Total	.60*	.57*	.36*	.03	.20†	.65*
(N)	(337)	(170)	(170)	(169)	(171)	(288)

\*Significant at  $p < .01$ ; † Significant at  $p < .05$

table 5.4 Correlations between CAPS total score and PDI-21 total score, OLIFE subscale and RLSHS in non-clinical sample.

Criterion validity was assessed by comparing the CAPS score of the non-clinical population with the sample of psychotic patients. The mean CAPS scores for all groups are provided in figure 5.2.

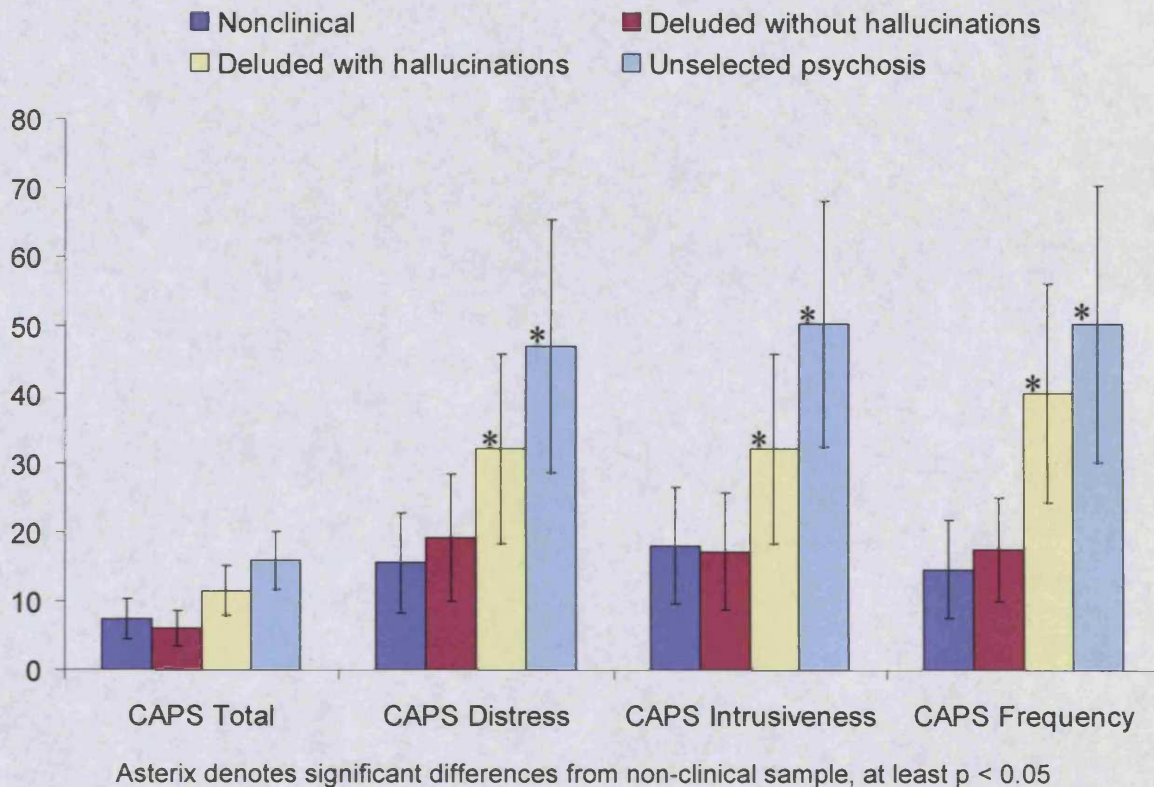


figure 5.2 CAPS scores for non-clinical, deluded without hallucinations, deluded with hallucinations and unselected psychotic groups.

The sample of unselected psychotic inpatients and the sample of deluded patients with hallucinations each had significantly higher mean scores on the CAPS total score and all CAPS dimension than the non-clinical population, with the unselected psychotic inpatient showing the highest mean CAPS scores of all. Interestingly, data from the sample of deluded patients without hallucinations were not significantly different from those of the non-clinical population when compared on CAPS total score or any of the CAPS dimensions, providing

evidence against theories that argue for anomalous perceptual experience as a necessary condition in delusion formation.

In keeping with the rationale for the new instrument, this further suggests that the CAPS is selective for perceptual anomalies, rather than psychosis per se. Although the sample size of the non-clinical sample is very large, the small size of the deluded patients without hallucinations sample means the null hypothesis cannot be accepted with sufficient power. Nevertheless, the fact that the unselected psychotic sample showed the highest mean CAPS scores can probably be explained by the fact that they were solely recruited from acute admission wards. Participants from other clinical samples were recruited from acute and rehabilitation inpatient wards and from clinically-supported accommodation. The latter two of these three groups comprise patients who may be past the most disabling stages of psychosis and may be experiencing less intense psychotic symptoms.

The CAPS is unique in terms of item selection – it tackles a comprehensive range of perceptual anomalies and does not assume that experiences present in a certain way (e.g. as ‘strange’ or ‘unusual’). In addition, it also includes dimensional scales to measure distress, intrusiveness and frequency, none of which is present in existing comprehensive measures of anomalous perceptual experience. As such the CAPS does not replicate existing measures and, therefore, has good incremental validity.

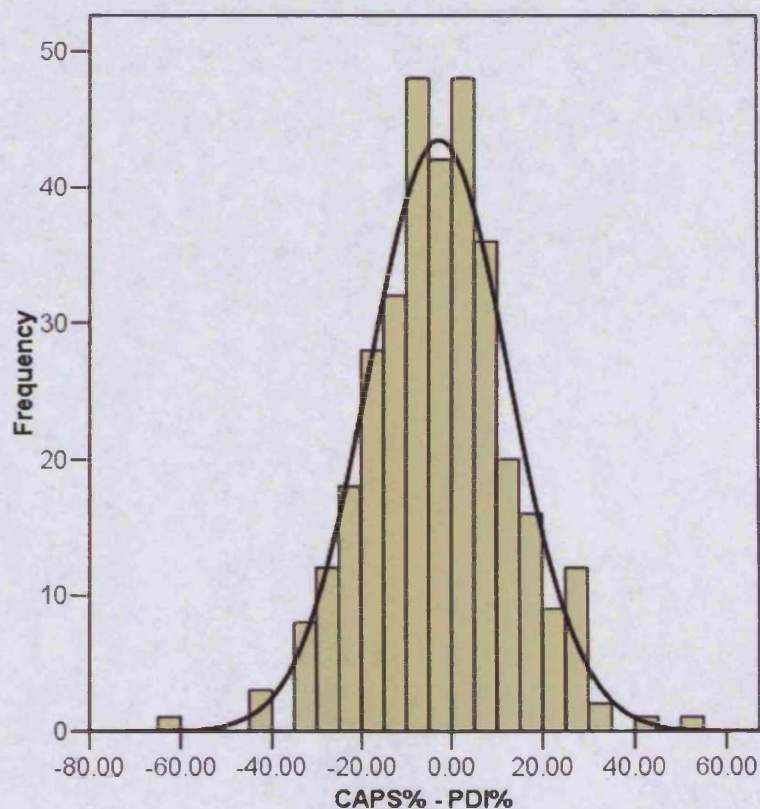
#### **5.5.4 Relationship between anomalous perceptual experience and delusional ideation**

Despite the importance placed on the second stage (section 3.2.2), second factor (section 3.2.3) or other presumed post-perceptual cognitive bias (usually described simply as a



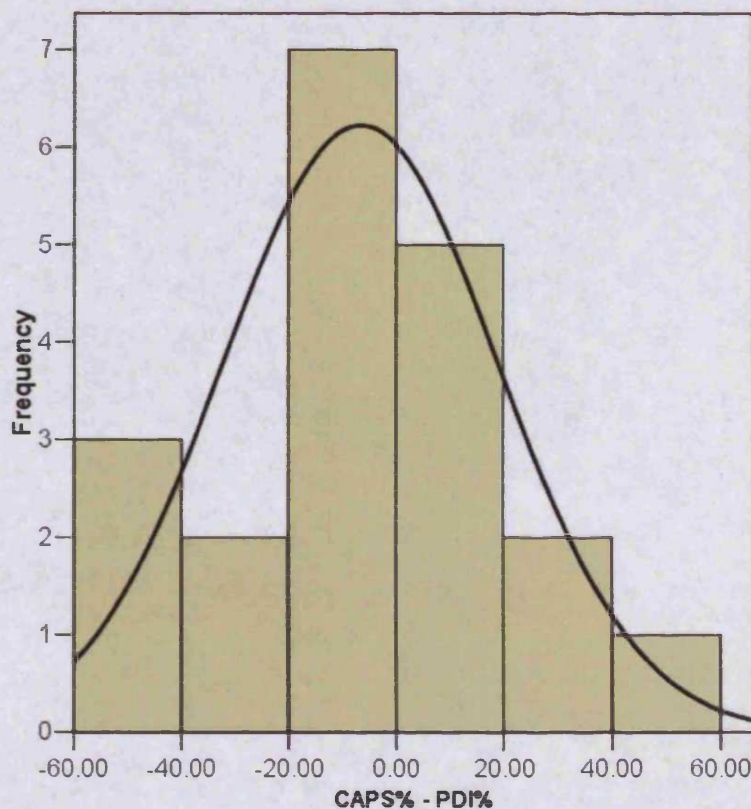
'cognitive bias', e.g. the Institute of Psychiatry model; section 3.41), this has not been quantified over a sample or population.

To achieve this, PDI total score was converted to a percentage and was subtracted from CAPS total score (also converted to a percentage) for each participant in the non-clinical sample. This produced a score which represented the percentage difference between each person's level of anomalous perceptual experience and delusional ideation. Because the range of possible scores on the CAPS is higher than the PDI (0-32, as opposed to 0-21), converting to percentages eliminates any skew due to range differences. The frequency distribution with overlaid normal curve is plotted in figure 5.3.



*figure 5.3 Frequency distribution of percentage difference between CAPS and PDI scores for non-clinical population (N = 337)*

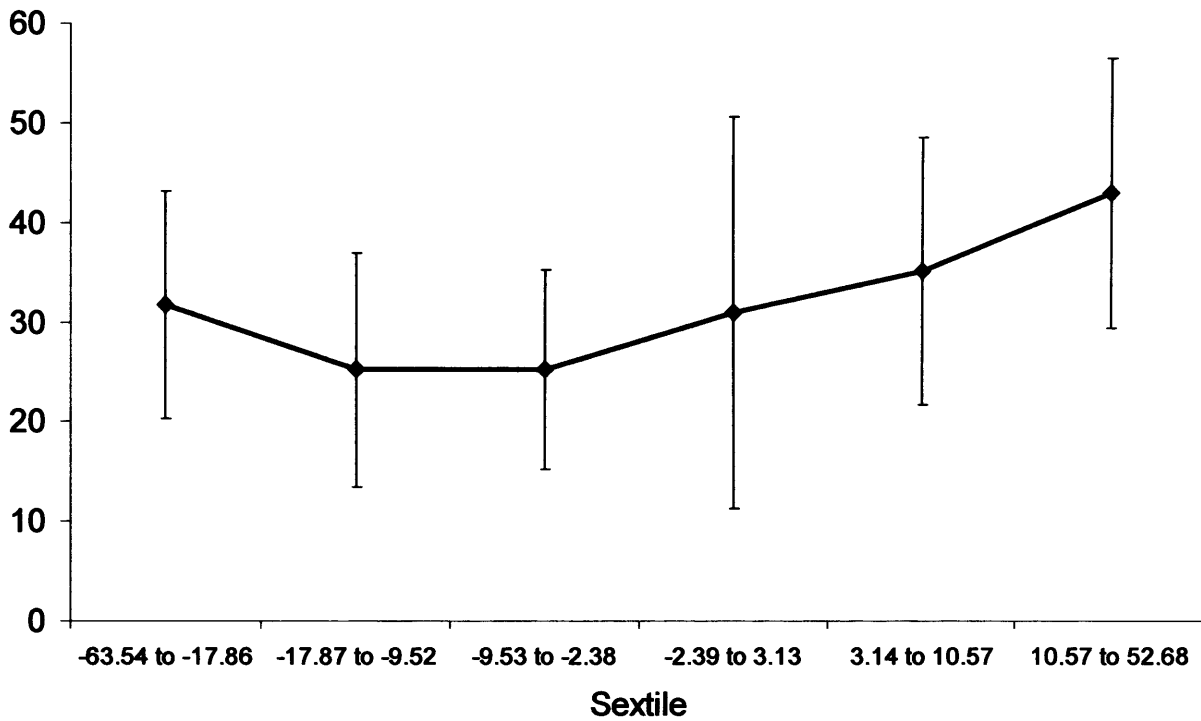
The distribution from the non-clinical sample, plotted in figure 5.3, has a mean of -2.86, a standard deviation of 15.48, a skewness of .043 and a kurtosis of .724. This suggests a highly symmetrical but 'peaked' normal distribution, with many of the data points clustering around the mean. As can be seen from the graph, the relationship between anomalous perceptual experience (as measured by the CAPS) and delusional ideation (as measured by the PDI) is almost perfectly normally distributed. This procedure was repeated for the sample of unselected psychotic patients (the only clinical sample who completed both the CAPS and PDI) and was similarly plotted (figure 5.4).



*figure 5.4 Frequency distribution of percentage difference between CAPS and PDI scores for unselected psychotic sample (N = 20).*

The distribution of the scores from the unselected psychotic sample, plotted in figure 5.4, has a mean of -6.66, a standard deviation of 25.67, a skewness of -.157 and a kurtosis of -.062. Although the small sample size means it is difficult to draw firm conclusions, there is, nevertheless, a suggestion of a normal distribution, albeit more closely resembling a standard normal distribution (i.e. a 'wider' spread of data points, as can be seen from the greater standard deviation and kurtosis closer to zero than in the non-clinical sample). Crucially, it is clear that in both the clinical and non-clinical samples, there are individuals for whom levels of anomalous perceptual experience and delusional ideation are closely linked (those close to the centre of the distribution), and those for whom these factors seem far more loosely related. Participants represented by the right hand tail of the graph have high levels of anomalous experience accompanying low levels of delusional ideation, and those represented by the left hand tail of the graph have low levels of anomalous experience in parallel with high levels of delusional ideation.

To explore the interaction between distress and differences in anomalous experience and delusional ideation, a combined PDI Distress and CAPS Distress subscale score was calculated and the non-clinical sample was divided by a sextile split on the percentage CAPS / PDI difference. As can be seen from figure 5.5, the higher levels of distress were seen in people with the greatest discrepancies between CAPS and PDI total scores, particularly where a positive difference indicated greater levels of anomalous experience than delusional ideation. This effect was significant when tested with a one-way between subjects ANOVA ( $F_{(5,351)} = 3.505, p < .005$ ).



*figure 5.5 Combined CAPS and PDI Distress scores by sextile split of percentage difference between CAPS and PDI Total scores.*

### 5.5.5 Factor structure

A principal components analysis (PCA) was conducted on data from the non-clinical sample to explore associations between items, without relying on any *a priori* hypotheses. Items on the CAPS that were endorsed by fewer than 10% of respondents were removed owing to lack of variance, leading to the removal of three items: item 19 (6.8%), item 13 (5.9%) and item 28 (1.2%). The remaining items on the CAPS were entered into the PCA using the oblique rotation (oblimin) procedure. An oblimin rotation was used since it assumes that the underlying factors are not necessarily independent from each other, which may often be the case in perceptual experience (for example, olfactory and gustatory experiences being strongly linked). The Kaiser-Meyer-Oklın value was .9, substantially exceeding the

recommended value of .6 and Bartlett's Test of Sphericity was significant ( $p < .0005$ ), supporting the suitability of PCA with this data set (Kaiser, 1974).

Principal components analysis revealed the presence of seven factors with eigenvalues exceeding 1, explaining a cumulative total of 50.26% of the variance. An inspection of the scree plot revealed a clear break after the third component, and a further PCA was run with three components retained for further investigation. Factor loadings from this analysis are outlined in table 5.5

Using a cut-off of .4 for factors loadings, three components can be identified: the first, (labelled as 'temporal lobe experience') consisting of items 26, 4, 32, 10, 12, 24, 2, 1, 16, 27 and 6; the second, (labelled as 'chemosensation') consisting of items 30, 18, 29, 21, 14, 25, 20, and 8; and the third (labelled as 'clinical psychosis') consisting of items 7, 11, 3 and 31. The three factor solution explained a cumulative total of 33.07% of the variance. The component totals correlated moderately (temporal lobe / chemosensation,  $r = .54$ ,  $p < .0005$ ; temporal lobe / clinical psychosis,  $r = .46$ ,  $p < .0005$ ; chemosensation / clinical psychosis,  $r = .32$ ;  $p < .0005$ ), supporting the appropriateness of the direct oblimin rotation for the PCA and suggesting that the components partially overlapped, but did not reflect identical sources of variance.

Item	Component		
	1	2	3
26. Do you ever think that everyday things look abnormal to you ?	.666		
4. Do you ever see shapes, lights or colours even though there is nothing really there ?	.648		
32. Do you ever hear sounds or music that people near you don't hear ?	.566		
10. Do you ever have the sensation that your limbs might not be your own or might not be properly connected to your body?	.546		-.311
12. Do you ever feel that someone is touching you, but when you look nobody is there ?	.534		
24. Do you ever have the feeling of being uplifted, as if driving or rolling over a road while sitting quietly ?	.502		
2. Do you ever sense the presence of another being, despite being unable to see any evidence ?	.453		
1. Do you ever notice that sounds are much louder than they normally would be ?	.441		
16. Do you ever find that sounds are distorted in strange or unusual ways ?	.441		
27. Do you ever find that your experience of time changes dramatically ?	.433		
6. Do you ever hear noises or sounds when there is nothing about to explain them ?	.400		
23. Do you ever have days where lights or colours seem brighter or more intense than usual ?	.389	-.306	
9. Do you ever have the sensation that your body, or a part of it, is changing or has changed shape ?			
30. Do you ever notice that food or drink seems to have an unusual taste ?		-.722	
18. Do you ever smell everyday odours and think that they are unusually strong ?		-.711	
29. Do you ever notice smells or odours that people next to you seem unaware of ?		-.641	
21. Do you ever think that food or drink tastes much stronger than it normally would ?		-.639	
14. Do you ever experience unexplained tastes in your mouth ?		-.614	
25. Do you ever find that common smells sometimes seem unusually different ?		-.493	
20. Do you ever find that your skin is more sensitive to touch, heat or cold than usual ?		-.486	
8. Do you ever detect smells which don't seem to come from your surroundings ?		-.458	
15. Do you ever find that sensations happen all at once and flood you with information ?		-.334	
22. Do you ever look in the mirror and think that your face seems different from usual ?		-.315	
17. Do you ever have difficulty distinguishing one sensation from another ?		-.308	
7. Do you ever hear your own thoughts spoken aloud in your head, so that someone near might be able to hear them ?			.674
11. Do you ever hear voices commenting on what you are thinking or doing ?			.641
3. Do you ever hear your own thoughts repeated or echoed ?			.607
31. Do you ever see things that other people cannot ?	.384		.443
5. Do you ever experience unusual burning sensations or other strange feelings in or on your body ?			.367

NB: Factor loadings below 0.3 not shown.

*table 5.5 CAPS Items and Factor Loading after Principal Components Analysis (Oblimin Rotation) of Response from Non-clinical Sample*

The first component (*temporal lobe experience*) encompasses items describing a number of different perceptual experiences. While many of these items could be associated with psychosis in general, the items present in this component seem a better match for perceptual

anomalies reported in temporal lobe disturbance than from frank psychosis. Gloor (1990) reviewed the temporal lobe epilepsy literature and outlined the phenomena associated with temporal lobe seizures as including visual illusions and hallucinations, auditory illusions and hallucinations of music or sounds (usually without clearly defined semantic content such as coherent verbal utterances as in the case of auditory hallucinations associated with psychosis), distortions in time perception, the relative lack of gustatory or olfactory experiences, and feelings of familiarity, recognition or emotion.

The perceptual component of Gloor's description seems to be well represented by CAPS items loading on this factor. Notably, this component consists of eleven items, whereas the items pre-chosen to reflect temporal lobe experiences not covered by the other items consists only of four. The total score of the pre-selected category, however, correlates highly with the total scores of temporal lobe component extracted from the PCA ( $r = .801, p < .0005$ ). Even when the shared items have been removed from the extracted component, the correlation remains strong and significant ( $r = .552, p < .0005$ ). Indeed, two items that load on this component (item 24 "Do you ever have the feeling that of being uplifted, as if driving or rolling over a road while sitting quietly ?", and item 2 "Do you ever sense the presence of another being, despite being unable to see any evidence ?") are taken directly from experiences present in the Temporal Lobe Scale of Makarec and Persinger, and are known to be present in populations that have temporal lobe disturbances (Makarec and Persinger, 1985; 1990). Such features are also known to be distributed throughout the general population in attenuated form (Persinger and Makarec, 1987). Similar experiences have been induced by the stimulation of the temporal lobes by magnetic fields (Cook and Persinger, 1997; Persinger and Healey, 2002) including anomalous proprioceptive experiences (Hill and Persinger, 2003), which may account for the loading of item 10 ("Do you ever have the sensation that

your limbs might not be your own or might not be properly connected to your body?") on this component.

The second component consists largely of items related to olfactory and gustatory experiences and has been tentatively labelled '*chemosensation*'. The presence of item 20 ("Do you ever find that your skin is more sensitive to touch, heat or cold than usual?") initially seems anomalous although is perhaps explained by the role of chemoreceptors in mediating perception of hot and cold, and the dual role of the trigeminal nerve in conducting olfactory and cutaneous sensitivity (touch, warmth / cold, pain) information (Cain, 1988).

Component three consists mainly of Schneiderian first-rank symptoms (Schneider, 1959), commonly used as clinically unambiguous indicators of schizophrenia, plus an additional item (item 31, "Do you ever see things that other people cannot?") concerning visual hallucinatory experiences and hence has been tentatively labelled '*clinical psychosis*'. Notably, item 5 ("Do you ever experience unusual burning sensations or other strange feelings in or on your body?") also loads on this factor, and is also a first-rank symptom, although it has a factor loading of .367 so is below the criterion for inclusion in this component (albeit only marginally so).

#### **5.5.6 CAPS item odds ratio analysis**

To identify those items that most distinguished the clinical from non-clinical groups, odds ratios were calculated for CAPS item frequencies (see Table 5.6). As the deluded-without-hallucinations group did not score significantly differently from the non-clinical group in the earlier analysis, they were excluded from the clinical group. Therefore, the clinical group consisted only of unselected psychotic and deluded-with-hallucinations patients.



Item	Item Text	Non-clinical		Clinical		Odds ratio	95% CI	
		Freq N	Freq %	Freq N	Freq %		Low	High
28	Have you ever heard two or more unexplained voices talking with each other ?	4	1.19	23	52.27	90.90	28.79	287.0 2
13	Do you ever hear voices saying words or sentences when there is no-one around that might account for it ?	20	5.95	26	59.09	22.82	10.76	48.41
11	Do you ever hear voices commenting on what you are thinking or doing ?	35	10.42	27	61.36	13.66	6.78	27.52
31	Do you ever see things that other people cannot ?	34	10.12	22	50.00	8.88	4.46	17.69
7	Do you ever hear your own thoughts spoken aloud in your head, so that someone near might be able to hear them ?	43	12.80	24	54.55	8.18	4.17	16.05
3	Do you ever hear your own thoughts repeated or echoed ?	73	21.73	27	61.36	5.72	2.96	11.07
19	Do you ever find the appearance of things or people seems to change in a puzzling way, e.g. distorted shapes or sizes or colour ?	23	6.85	13	29.55	5.71	2.63	12.37
9	Do you ever have the sensation that your body, or a part of it, is changing or has changed shape ?	43	12.80	16	36.36	3.89	1.95	7.78
32	Do you ever hear sounds or music that people near you don't hear ?	78	23.21	22	50.00	3.31	1.74	6.29
5	Do you ever experience unusual burning sensations or other strange feelings in or on your body ?	97	28.87	25	56.82	3.24	1.71	6.16
15	Do you ever find that sensations happen all at once and flood you with information ?	60	17.86	18	40.91	3.18	1.64	6.18
16	Do you ever find that sounds are distorted in strange or unusual ways ?	49	14.58	15	34.09	3.03	1.51	6.06
6	Do you ever hear noises or sounds when there is nothing about to explain them ?	135	40.18	29	65.91	2.88	1.49	5.57
12	Do you ever feel that someone is touching you, but when you look nobody is there ?	67	19.94	18	40.91	2.78	1.44	5.37
10	Do you ever have the sensation that your limbs might not be your own or might not be properly connected to your body?	36	10.71	11	25.00	2.78	1.29	5.97
25	Do you ever find that common smells sometimes seem unusually different ?	38	11.31	10	22.73	2.31	1.06	5.04
2	Do you ever sense the presence of another being, despite being unable to see any evidence ?	146	43.45	28	63.64	2.28	1.19	4.37
8	Do you ever detect smells which don't seem to come from your surroundings ?	82	24.40	18	40.91	2.14	1.12	4.11
30	Do you ever notice that food or drink seems to have an unusual taste ?	75	22.32	16	36.36	1.99	1.02	3.87
23	Do you ever have days where lights or colours seem brighter or more intense than usual ?	111	33.04	21	47.73	1.85	0.98	3.49
14	Do you ever experience unexplained tastes in your mouth ?	86	25.60	16	36.36	1.66	0.86	3.22
18	Do you ever smell everyday odours and think that they are unusually strong ?	86	25.60	16	36.36	1.66	0.86	3.22
1	Do you ever notice that sounds are much louder than they normally would be ?	99	29.46	17	38.64	1.51	0.79	2.89
29	Do you ever notice smells or odours that people next to you seem unaware of ?	103	30.65	17	38.64	1.42	0.74	2.73
26	Do you ever think that everyday things look abnormal to you ?	64	19.05	11	25.00	1.42	0.68	2.95
21	Do you ever think that food or drink tastes much stronger than it normally would ?	79	23.51	12	27.27	1.22	0.60	2.48

table 5.6 [continued on next page] Comparison between clinical (unselected psychotic and deluded-with-hallucinations patients only) and non-clinical group by odds ratio of item endorsement.

Item	Item Text	Non-clinical		Clinical		Odds ratio	95% CI	
		Freq N	Freq %	Freq N	Freq %		Low	High
22	Do you ever look in the mirror and think that your face seems different from usual ?	139	41.37	20	45.45	1.18	0.63	2.22
17	Do you ever have difficulty distinguishing one sensation from another ?	55	16.37	8	18.18	1.14	0.50	2.57
24	Do you ever have the feeling that of being uplifted, as if driving or rolling over a road while sitting quietly ?	71	21.13	10	22.73	1.10	0.52	2.33
27	Do you ever find that your experience of time changes dramatically ?	148	44.05	20	45.45	1.06	0.56	1.99
4	Do you ever see shapes, lights or colours even though there is nothing really there ?	132	39.29	17	38.64	0.97	0.51	1.85

table 5.6 [continued from previous page] Comparison between clinical (unselected psychotic and deluded-with-hallucinations patients only) and non-clinical group by odds ratio of item frequency.

Category	Non-clinical		Clinical		OR	95% CI	
	Freq N	Freq %	Freq N	Freq %		Low	High
By component from principal component analysis							
Component three 'clinical psychosis' (3,7,11,31)	46.25	13.76	25.00	56.82	8.24	4.21	16.15
Component one 'temporal lobe experience' (1,2,4,6,10,12,16,24,26,27,32)	93.18	27.73	18.00	40.91	1.80	0.94	3.44
Component two 'chemosensation' (8,14,18,20,21,25,29,30)	88.38	26.30	15.38	34.94	1.50	0.77	2.93
By sensory modality							
Audition (1,3,6,7,11,13,16,28,32)	59.56	17.72	23.33	53.03	5.24	2.72	10.09
Touch (5,9,10,12,20)	80.20	23.87	17.60	40.00	2.13	1.11	4.09
Vision (4,19,22,23,26,31)	83.83	24.95	17.33	39.39	1.96	1.02	3.76
Smell (8,18,25,29)	77.25	22.99	15.25	34.66	1.78	0.91	3.48
Taste (14,21,30)	80.00	23.81	14.67	33.33	1.60	0.81	3.14
By initial CAPS creation categories							
Verbal hallucinations (13,11,28)	19.67	5.85	25.33	57.58	21.83	10.29	46.29
Thought echo and hearing thoughts out loud (3,7)	58.00	17.26	25.50	57.95	22.17	10.45	47.04
Having a non-shared sensory experience (13,29,31,32)	58.75	17.49	21.75	49.43	4.61	2.40	8.88
Sensory experience from an unexplained source (4,6,8,12,14,28)	84.33	25.10	20.17	45.83	2.53	1.33	4.80
Distortion of form (size, shape) of own body and of external world (9,10,22,19)	60.25	17.93	15.00	34.09	2.37	1.20	4.69
Sensory flooding (15,17)	57.50	17.11	13.00	29.55	2.03	1.00	4.12
Inherently unusual or distorted (5,16,25,26,30)	64.60	19.23	15.40	35.00	2.26	1.15	4.45
Temporal Lobe (2,10,24,27)	100.25	29.84	17.25	39.20	1.52	0.79	2.90
Changes in levels of sensory intensity (1,18,20,21,23)	106.60	31.73	16.80	38.18	1.33	0.69	2.55

table 5.7 Odds ratios for mean frequency for CAPS items categories comparing between clinical and non-clinical populations. Contributory items listed in brackets.

Table 5.7 displays the odds ratios for various categories of CAPS items. These were derived by calculating the mean of the frequency of individual item endorsement in a particular category, and calculating the odds ratios based on these mean endorsement scores. The categories included the three components derived from the principal components analysis, five sensory modalities and the categories derived from the literature review that were used as an initial framework for the creation of CAPS items.

The odds ratio comparison between the clinical and non-clinical group shows that items resembling Schneiderian first-rank symptoms ('clinical psychosis' component, 'verbal hallucinations' and 'thought echo and hearing thoughts out loud' categories) most distinguished the clinical from the non clinical group. This is, perhaps, not surprising, since first-rank symptoms are commonly used in the UK for the diagnosis of schizophrenia (Sims, 2003) – a diagnosis that features highly in the clinical group. Notably, both the component from the PCA interpreted as 'temporal lobe experience' and the pre-selected 'temporal lobe' category do not significantly distinguish clinical from non-clinical groups (as the confidence intervals include even odds; i.e. 1), again suggesting that these experiences are not distinctive of clinical psychosis and may, therefore, exert an independent influence on the overall level of anomalous experience.

An odds ratio comparison of the insight-related categories 'having a non-shared sensory experience', 'sensory experience from an unexplained source' and 'inherently unusual or distorted' shows that they are all more frequent in the clinical groups, although only 'having a non-shared sensory experience' most significantly and substantially distinguishes between clinical and non-clinical groups. This may indicate that anomalous perceptual experiences stemming from clinical psychosis may be more likely to be self-recognised as one that is 'not

shared by others', rather than as one that seems 'inherently unusual or distorted' or stems from an 'unexplained source'.

When items are categorised by sensory modality, they reflect previous findings from large scale surveys of hallucinatory phenomena (Ohayon, 2000) in that gustatory and olfactory are equally as common in the non-clinical and clinical populations.

## **5.6 Discussion**

The aim behind developing the CAPS was to construct a scale that would be selective for perceptual anomalies, without being conceptually tied to the assumptions and language of previous clinical and psychometric scales. In particular, the object was to create a scale that would tap a range of experience within relevant sensory domains, without relying solely on judgements of 'strangeness' or 'unusualness' to establish the presence of perceptual anomalies. Dimensional ratings of distress, intrusiveness and frequency were also included for each item. The results of this study suggest that the Cardiff Anomalous Perceptions Scale is a reliable, valid measure of the presence of perceptual anomalies. No significant difference in CAPS score was found between general population participants and deluded inpatients without hallucinations, suggesting, contrary to 'one-stage theories', pathological levels of anomalous experience is neither necessary nor sufficient for delusion formation.

The correlation between the CAPS and RLSHS demonstrates a relationship between the presence of perceptual anomalies and predisposition to hallucinations. In particular, the correlation between the CAPS total score and the unusual experiences subscale of the OLIFE schizotypy scale, whilst showing weak or non-significant correlations with the other subscales, is good evidence for the selectivity of the CAPS in measuring perceptual

anomalies, indicating that the CAPS is not simply a measure of general psychosis-proneness or schizotypy. The results also show a relationship between CAPS total scores and PDI-21 total scores, indicating that the presence of perceptual anomalies and non-clinical delusional ideation may be linked.

Previous studies have tended to show that hallucinations and delusions commonly co-occur in clinical samples (Bilder et al., 1985; Peralta and Cuesta, 1999; Peralta et al., 1992). In contrast to this study, however, delusional patients have not previously been differentiated into hallucinating and non-hallucinating groups, and, therefore, such results may reflect a population average rather than a causal effect. This begs the question as to whether the presence either of hallucinations, or, of other anomalous perceptual experiences are either necessary or sufficient for delusion formation. Indeed, as mentioned earlier, both 'one-stage' Maher (1988; 1999; section 3.2.1) and 'two-stage' (Ellis and Young, 1990; section 3.2.2) theorists have argued that anomalous experiences are either necessary, or necessary and sufficient, to account for the presence of delusions. The fact that the non-hallucinating delusional sample from this study scored almost identically on the CAPS to the non-clinical sample suggests that accounts that argue for the necessity for pathological levels of anomalous perceptual experience in delusion formation need to be reconsidered.

Although the comparison did not have adequate power to accept the null hypothesis, the fact that these accounts argue for anomalous perceptual experience as a *necessary* condition, means that any failure to find elevated levels of anomalous experience in a group of deluded patients with a valid, reliable measure could be construed as evidence against these models. This suggests that, while the presence of anomalous experience may promote delusion formation, it cannot be considered a complete explanation, or even an essential factor.

Part of the confusion may lie in the way some clinically-relevant experiences have been classified. Gold and Hohwy have argued that many passivity symptoms (such as thought insertion), although traditionally classified as delusions, “are best explained as disorders of experience rather than disorders of belief desire or reasoning” (Gold and Hohwy, 2000, p160). More likely, it seems, is that processes involved in perception and experience explanation can be differentially affected, and that delusions could arise from pathology of either process. Two stage theorists have argued for the necessity of bias or deficit in reasoning processes to account for delusion formation (Ellis, 1998; Ellis and Young, 1997; Stone and Young, 1997; section 3.2.2). Stone and Young (1997) in particular, suspect that beliefs are the result of a process that weighs explanations that are observationally adequate against those that fit within a person’s current belief set.

A more detailed model by Langdon and Coltheart (2000), discussed earlier in section 1.6.1, specifies a multi-stage belief formation process and argues that delusions could arise from damage either to a hypothesis-generation stage or a rational evaluation stage. They do not exclude the influence of aberrant perceptions in their model and regard the generation of hypotheses as partly dependent on current perceptual input, potentially leading to a delusional belief if a hypothesis is generated to explain anomalous perceptual experience is crystallised into a belief without the benefit of the checks and balances of an optimally functioning cognitive system. The results of this study are consistent with such cognitive models, but further suggest that there may be a subgroup of people with delusions whose pathology is restricted to, or centred on, the evaluative processes (the ‘second factor’) involved in belief formation, rather than the integration of perceptual experience. Indeed, this is consistent with speculations in a recent model of persecutory delusions (Freeman et al., 2002).

An interesting finding was that the frequencies of the differences in percentage scores between the CAPS and the PDI suggested a normal distribution for both the non-clinical and the unselected psychotic sample (although there was a wider spread of data points in the unselected psychotic sample and the sample size was very small), suggesting that the *relationship* between anomalous perceptual experience and delusional ideation is normally distributed. This may reflect the operation of exactly the sort of appraisal or evaluative processes that are usually described as the ‘second factor’ (section 3.2.3).

If this is indeed the case, and by all accounts the link between anomalous perceptual experience and delusional ideation is exactly what the ‘second factor’ is supposed to capture (Blakemore et al., 2002; Ellis, 1998; Ellis and Young, 1997; Garety and Hemsley, 1994; Stone and Young, 1997; sections 3.2.2; 3.5.2), these findings have some important implications. The first is, perhaps, that the ‘second factor’ is distributed throughout the population like many other traits, potentially in both the delusional patients and the general population. If the ‘second factor’ in delusional patients follows a similar pattern of distribution to the general population, it suggests that this cannot be the site of an ‘all or nothing’ deficit. The second implication is that the subtraction method reported here is the first potential measure of the ‘second factor’ drawn from principled measures of the relevant phenomenology.

Although these results suggest the second factor is not necessarily pathological in itself, the fact that it can be measured allows a metric on which to base studies looking at interactions between the ‘second factor’ and other aspects, which, in combination, may eventually lead to a belief being considered pragmatically pathological or clinically significant. Emotion is an

increasingly likely candidate for a mediating factor in a pathway to pathology, considering its central role in defence and motivational accounts of delusions (section 3.3) and theories which put the direct role of emotion as central to delusion formation (section 3.4.3).

As suggested by the analysis of the link between the proposed measure of the ‘second factor’ and distress, it seems that those in the non-clinical population with the greatest discrepancy between anomalous perceptual experience and delusional ideation are more likely to experience distress. Particularly, it seems, those on the right of the graph, who experience high levels of anomalous experience, but low levels of delusional ideation, perhaps suggesting that delusional ideation might be protective against distress in those who experience high levels of anomalous experience. It is not clear, however, whether distress is the result of maladaptive appraisal of anomalous experience, or whether distress itself is feeding into the discrepancy. Relevant experimental evidence has recently been provided by a longitudinal study by Hanssen et al. (2005), who reported that participants experiencing distress in combination with an hallucinatory episode at the first assessment, had a fourfold chance of presenting with a delusion by the second. This would suggest that the emotional impact of an anomalous perceptual experience may be the critical factor in determining whether it leads to a delusion in the long-term.

The investigation reported in this chapter is unable to provide further evidence for the hypothesis implied by the Hanssen et al. results. These issues are addressed in Chapter 7, however, where the relationship between anomalous perceptual experience, distress and clinical diagnosis is examined in more detail.



Although there was a significant difference between groups on ratings of distress, intrusiveness and frequency (figure 5.2), there was little difference within groups when distress, intrusiveness and frequency ratings were compared, suggesting that for the populations sampled, these factors may be highly linked. Previous studies, however, have shown that meta-cognitive factors and affective reactions to anomalous experiences may be mediated by the framework and beliefs in which they are interpreted (Bhugra, 1996; Chadwick and Birchwood, 1994; Davies et al., 2001; Gauntlett-Gilbert and Kuipers, 2005; Morrison et al., 2002), a possibility which is also explored in Chapter 7.

Although it is not possible to distinguish which meta-cognitive factors may be important in this process from the data presented here, this may be an important avenue for future research to determine, and perhaps therapeutically target those aspects of anomalous experience that are particularly associated with distress, preoccupation or other disabling aspects. To do this effectively, however, it may be important to distinguish some of the factors influencing the presence of anomalous experience.

The results of the principal components analysis of CAPS results from the non-clinical sample suggest that there may be multiple factors contributing to the overall level of perceptual distortion. Three main components were revealed and were interpreted as experiences associated with clinical psychosis, experiences associated with chemosensation (largely olfactory and gustatory experiences) and experiences associated with temporal lobe disturbance.

The existence of a coherent multi-factor structure suggests that anomalous perceptual experience cannot simply be treated as a unitary dimension, as is often the case in theories of psychosis and psychosis-proneness.

In fact, the strong grouping of CAPS items that reflect the presence of Schneiderian first rank symptoms, suggests that experiences associated with psychotic mental illness (and particularly schizophrenia) may make a relatively independent contribution to the overall level of anomalous experience. A recent factor analytic study of the RLSHS (Serper et al., 2005) produced a two-factor solution (interpreted as 'clinical' and 'non-clinical' components) that looked remarkably similar in both patient and non-patient samples, which led the authors to argue that anomalous experience may become pathological above a certain threshold. The 'clinical psychosis' component of the CAPS may also reflect a similar process, although this leaves unexplained why this threshold exists and what might cause the transition (one possible theory is the role of emotion, discussed previously).

The 'chemosensation' component largely reflects anomalous experience in the olfactory and gustatory modalities. This is in accordance with phenomena that have been reported as the most prevalent hallucinatory experiences in a study of over 13,000 members of the general population (Ohayon, 2000), despite the fact that they are relatively uncommon in psychotic mental illness when compared with hallucinations in other modalities.

Perhaps more equivocal is the interpretation of the first component as being associated with temporal lobe disturbance. It is important to make the distinction here between perceptual anomalies associated with temporal lobe disturbances and clinical psychosis associated with temporal lobe epilepsy. Although seizures associated with temporal lobe epilepsy may

produce a number of perceptual anomalies (Gloor, 1990), this does not in itself constitute psychosis, although a minority of people with temporal lobe epilepsy may go on to develop psychosis.

Indeed, reviews of the literature on psychosis in temporal lobe epilepsy show a mean prevalence of 15.7% with a typical onset of 11-15 years after the onset of epilepsy (Trimble, 1991; Trimble et al., 1996). This suggests that, although there may be some commonalities between temporal lobe seizures and psychosis, the core phenomenology is largely distinct and distinguishable.

The work of Makarec and Persinger has suggested that levels of transient temporal lobe disturbance (so-called 'microseizures') may be distributed throughout the population and contribute to a continuum of unusual experiences and beliefs, although not necessarily of the same character to those associated with psychosis (Makarec and Persinger, 1985; Makarec and Persinger, 1990; Persinger and Makarec, 1987).

Notably, however, the connection between such findings and work on the continuum model of psychosis has rarely been made, despite obvious parallels. It is possible that perceptual distortions associated with psychosis and those associated with temporal lobe disturbance may share an overlapping neurological basis; for example, each is known to involve the tempero-limbic areas to varying degrees (Trimble, 1991; Bear, 1979). This may account for the fact that a number of items in the temporal lobe component, although characteristic of temporal lobe disturbance, could also be present in psychosis. The question of whether this component is best characterised as 'temporal lobe' experience, or to what extent verifiable

temporal lobe disturbance contributes to the variance of this component remains an empirical question.

Further work has provided some additional evidence for the validity of this factor, however. Temporal lobe disturbance induced by transcranial magnetic stimulation can reliably affect the perception of meaning in visual noise (Bell et al., in press) suggesting a causative role for the temporal lobes in anomalous experience (to be explained in Chapter 6). Similarly, a small validation study, conducted in collaboration with Caroline Dietrich (see Appendix II), has suggested, albeit from a small sample size, that the temporal lobe factor on the CAPS is the only factor which significantly predicts variance on Makarec and Persinger's (1985) temporal lobe scale.

In terms of the odds ratio comparisons of CAPS items (tables 5.6 and 5.7), however, both the pre-selected temporal lobe category and the category from the temporal lobe component from the PCA have similar odds ratios (1.52 and 1.80 respectively; with the confidence intervals of each encompassing even odds; i.e. 1), despite the latter consisting of eleven items when compared with the four items of the pre-selected temporal lobe category. The fact that these categories have a similar odds ratio between clinical and non-clinical groups, despite sharing only four items, and despite that fact that one was selected a priori whereas the other emerged post hoc, provides additional evidence for the validity of these categories.

The insight-related categories displayed a notable difference in prevalence, suggesting that insight-related attributes can differentiate similar experiences between clinical and non-clinical groups. The odds ratios differences between groups in these categories suggests, however, that insight is not a unitary process. Particularly, as the odds ratio for the category

‘having a non-shared sensory experience’ was almost twice that of the other two categories, and the confidence intervals of the other categories only marginally avoided including an even odds ratio. This implies that, although phenomena experienced by the clinical group may only seem marginally more ‘unusual’ or from an ‘unexplained source’ than those experienced by the general population, they are likely to be more aware of the fact that these are not experiences that are shared by others around them.

Traditionally insight has been seen as a unidimensional process, with lack of insight being the defining feature of psychotic experience (Berrios and Markova, 2004); although more recently insight has begun to be viewed as a multidimensional construct (Amador and David, 2004). The findings presented here suggest that psychosis may involve the selective impairment in the results of certain judgements or metacognitions associated with anomalous perceptual experiences, namely the realisation that they are indeed anomalous. The realisation that these experiences may not have an obvious environmental source seems not to substantially differentiate the clinical from the non-clinical group.

In contrast, the realisation that these experiences are not shared by others does, in that the clinical group tends to realise the non-shared nature of the experience, while the non-clinical group tends not to. This perhaps reflects the experience of personal significance and self-reference in psychosis, and further suggests that there are various ways of understanding and measuring insight. Notably, the presence of this awareness may have important implications for the framing of cognitive therapy, particularly the use of the ‘collaborative empiricism’ method in psychosis, where existing insight is used as a starting point to test and resolve aspects of the psychotic experience (Chadwick and Lowe, 1990).

Furthermore, one item, indicative of a frank hallucinatory experiences (item 4 “Do you ever see shapes, lights or colours even though there is nothing really there ?”) was actually marginally more common in the non-clinical group, suggesting that a visual hallucination, in itself, is not necessarily of clinical significance. Two other items, indicative of temporal lobe disturbance: item 27 “Do you ever find that your experience of time changes dramatically ?”; and item 24 “Do you ever have the feeling that of being uplifted, as if driving or rolling over a road while sitting quietly ?”, were also almost identically prevalent in the two groups. These, perhaps, provide further support for the notion that anomalous experience from temporal lobe disturbance may be non-clinical in nature, or at least are not sufficient to account for anomalous experience associated with psychosis.

The current study does have several limitations, however, most notably that the non-clinical sample was largely drawn from undergraduate students and may not be truly representative of the wider population. As mentioned earlier, the effect of the younger age of the non-clinical sample is unlikely to have confounded the conclusions, as younger adults tend to show higher levels of anomalous experience than older adults and suggests that the reported effects might be stronger in an age-matched sample.

Socioeconomic status was not recorded, although it is possible that the non-clinical sample were typically from a background of a higher mean socioeconomic status than the clinical sample, who were treated in services dealing with a wide range of clients from various areas of large city centres. Socioeconomic status is known to be inversely related to psychosis-continuum experiences (van Os et al., 2000) and, therefore, it is not possible to rule out an effect of this on the results reported here, although it is unlikely that this effect would have invalidated the main findings.

In summary, the CAPS is a useful and comprehensive measure of anomalous perceptual experience independent of psychiatric diagnosis that has obvious clinical applicability. Comparisons between CAPS scores of deluded hallucinating patients, deluded non-hallucinating patients and a non-clinical sample suggest that the presence of delusions is not necessarily associated with a level of perceptual anomaly above that present in a non-clinical population, in contrast to the predictions of one-stage theories of delusion formation. A principal components analysis of the CAPS data from the non-clinical sample shows three components that can be interpreted as 'clinical psychosis', 'chemosensation' and 'temporal lobe disturbance'. This suggests that multiple mechanisms underlie anomalous perceptual experience and the 'continuum of psychosis' is influenced by several sources of perceptual distortions.

## **5.7 Chapter summary and conclusion**

Perhaps the first notable finding to arise from this analysis is that overall level of anomalous perceptual experience involves several contributory factors. The component labelled ‘clinical psychosis’ largely consists of experiences classified as ‘first rank symptoms’, that are often designated as experiences pathognomic of schizophrenia (Sims, 2003). Notably, these experiences seem to cluster together even in the non-clinical sample, suggesting that, although there may be a psychosis continuum (section 2.5.3), there may also be a threshold above which experiences are more likely to become pathological. This concept has been suggested in recent empirical studies (Abel et al., 2005; Serper et al., 2005) and also used in some computational models of delusion formation (section 3.8.5).

The role of emotion has been highlighted as a potential mediating factor in the potential transition to pathology, and this issue is further explored in Chapter 7, in an attempt to get a handle on what might be the actual pathological component in delusions. This is particularly important, in light of the fact that the validity of the currently used definition has been put in doubt (section 2.4; Chapter 4); and that this study provided evidence that high levels of anomalous experience do not seem to be a necessary condition for delusion formation.

The potential contribution of temporal lobe disturbance to perceptual distortion is another point of interest underscored by this study, particularly given the fact that research into this area has produced remarkably similar findings to the psychosis continuum literature, although has been developed almost entirely independently with virtually no cross-referencing between the traditions.



The importance of validating and better specifying the potential ‘temporal lobe’ contribution is also amplified by the number of studies that have implicated the temporal cortices in psychosis and delusion formation (section 3.7.1) and the urgent need for these studies to move beyond a purely correlation account and look for causal factors (section 3.7.3). This issue is tackled in Appendix II, which investigates whether the temporal lobe component of the CAPS predicts the score on a well-validated psychometric measure of temporal lobe disturbance, and in Chapter 6, which uses transcranial magnetic stimulation to control for many of the confounds in previous clinical work and produce evidence for a causal role of the temporal lobes in anomalous experience and delusion formation.

## 5.8 References

- Abel, K. M., Jolley, S., Hemsley, D. R., & Geyer, M. A. (2004) The influence of schizotypy traits on prepulse inhibition in young healthy controls. *Journal of Psychopharmacology*, 18, 181-188.
- Amador X, David, A. (2004) *Insight and psychosis (2nd ed)*. Oxford: Oxford University Press.
- American Psychiatric Association (2000) *Diagnostic and Statistical Manual of Mental Disorders, ed 4, Text Revision (DSM-IV-TR)*. Washington: American Psychiatric Association.
- Andreasen, NC. (1994) *Scale for the assessment of positive symptoms (SAPS)*. Iowa City, University of Iowa.
- Bear, DM. (1979) Temporal lobe epilepsy - a syndrome of sensory-limbic hyperconnection. *Cortex*, 15, 357-384.
- Bebbington PE, Nayani T. (1995) The psychosis screening questionnaire. *International Journal of Methods in Psychiatric Research*, 5, 11-19.
- Bell V, Reddy V, Halligan PW, Kirov G, Ellis HD. (in press) Relative suppression of magical thinking: A transcranial magnetic stimulation study. *Cortex*

Bentall RP, Claridge GS, Slade PD. (1989) The multidimensional nature of schizotypal traits: a factor analytic study with normal subjects. *British Journal of Clinical Psychology*, 28, 363-75.

Bentall RP, Slade PD. (1985) Reality testing and auditory hallucinations: a signal detection analysis. *British Journal of Clinical Psychology*, 24, 159-69.

Berrios GE, Markova IS. (2004) Insight in the psychoses: A conceptual history. In: Amador X, David A, eds. *Insight and psychosis (2nd ed.)*. Oxford: Oxford University Press.

Bhugra D. (1996) *Psychiatry and religion*. London: Routledge.

Bilder RM, Mukherjee S, Rieder RO, Pandurangi AK. (1985) Symptomatic and neuropsychological components of defect states. *Schizophrenia Bulletin*, 11, 409-419.

Blakemore, S. J., Wolpert, D. M., Frith, C. D. (2002) Abnormalities in the awareness of action. *Trends in Cognitive Sciences*, 6, 237-242.

Bleuler E. (1911) *Dementia praecox or the group of schizophrenias*. Translated by J. Zinkin. New York: International Universities Press.

Brédart S, Young AW. (2004) Self-recognition in everyday life. *Cognitive Neuropsychiatry*, 9, 183-197.

Bunney WE, Hetrick WP, Bunney BG, Patterson JV, Jin Y, Potkin SG, Sandman CA. (1999) Structured Interview for Assessing Perceptual Anomalies (SIAPA). *Schizophrenia Bulletin*, 25, 577-92.

Cain WS. (1988) Olfaction. In: Atkinson RC, Herrnstein RJ, Lindzey G, Luce RD, eds. *Steven's handbook of experimental psychology (2nd ed)*. New York: John Wiley and Sons.

Chadwick P, Birchwood M. (1994) The omnipotence of voices. A cognitive approach to auditory hallucinations. *British Journal of Psychiatry*, 164, 190-201.

Chadwick PDJ, Lowe CF. (1990) Measurement and modification of delusional beliefs. *Journal of Consulting and Clinical Psychology*, 58, 225-232.

Chapman LJ, Chapman JP, Raulin ML. (1978) Body-image aberration in Schizophrenia. *Journal of Abnormal Psychology*, 87, 399-407.

Claridge G, McCreery C, Mason O, Bentall R, Boyle G, Slade P, Popplewell D. (1996) The factor structure of 'schizotypal' traits: a large replication study. *British Journal of Clinical Psychology*, 35, 103-15.

Clark-Carter D. (1997) *Doing quantitative psychological research: From design to report*. Hove: Psychology Press.

Cook CM, Persinger MA. (1997) Experimental induction of the 'sensed presence' in normal subjects and an exceptional subject. *Perceptual and Motor Skills*, 85, 683-693.

Davies MF, Griffin M, Vice S. (2001) Affective reactions to auditory hallucinations in psychotic, evangelical and control groups. *British Journal of Clinical Psychology*, 40, 361-70.

Eckblad M, Chapman LJ. (1983) Magical ideation as an indicator of schizotypy. *Journal of Consulting and Clinical Psychology*, 51, 215-225.

Ellis HD. (1998) Cognitive neuropsychiatry and delusional misidentification syndromes: An exemplary vindication of the new discipline. *Cognitive Neuropsychiatry*, 3, 81-90.

Ellis HD, Young AW. (1990) Accounting for delusional misidentifications. *British Journal of Psychiatry*, 157, 239-248.

Eysenck, HJ. (1952) Schizothymia-cyclothymia as a dimension of personality. *Experimental Journal of Personality*, 20, 345-384.

Eysenck, HJ, Eysenck SBG. (1976) *Psychoticism as a Dimension of Personality*. London: Hodder and Stoughton.

Freeman D, Garety PA (2003) Connecting neurosis and psychosis: the direct influence of emotion on delusions and hallucinations. *Behaviour Research and Therapy*, 41, 923-947.

Freeman D, Garety PA, Kuipers E, Fowler D, Bebbington PE. (2002) A cognitive model of persecutory delusions. *British Journal of Clinical Psychology*, 41, 331-47

Garety P, Hemsley DR. (1994) *Delusions: Investigations into the psychology of delusional reasoning*. Hove: Psychology Press.

Gauntlett-Gilbert J, Kuipers E. (2005) Visual hallucinations in psychiatric conditions: appraisals and their relationship to distress. *British Journal of Clinical Psychology*, 44, 77-87.

Gloor, P. (1990) Experiential phenomena of temporal lobe epilepsy. Facts and hypotheses. *Brain*, 113, 1673-1694.

Gold I, Hohwy J. (2000) Rationality and schizophrenic delusion. In: Coltheart M, Davies M, eds. *Pathologies of belief*. Oxford: Blackwell Publishers Ltd

Gonzalez-Pinto, A.; van Os, J.; Peralta, V.; Perez de Heredia, J.L.; Mosquera, F.; Aldama, A.; Gonzalez, C.; Gutierrez, M.; and Mico, J.A. (2004) The role of age in the development of Schneiderian symptoms in patients with a first psychotic episode. *Acta Psychiatrica Scandinavica*, 109, 264-8.

Hafner H, Riecher-Rossler A, Maurer K, Fatkenheuer B, Loffler W. (1992) First onset and early symptomatology of schizophrenia. A chapter of epidemiological and neurobiological research into age and sex differences. *European Archives of Psychiatry and Clinical Neuroscience*, 242, 109-18.

Hanssen M, Krabbendam L, de Graaf R, Vollebergh W, van Os J. (2005) Role of distress in delusion formation. *British Journal of Psychiatry Supplement*, 48, s55-s58.

Hill DR, Persinger MA. (2003) Application of transcerebral, weak (1 microT) complex magnetic fields and mystical experiences: are they generated by field-induced dimethyltryptamine release from the pineal organ? *Perceptual and Motor Skills*, 97, 1049-50.

Johns LC, van Os J. (2001) The continuity of psychotic experiences in the general population. *Clinical Psychology Review*, 21, 1125-1141.

Kaiser H. (1974) An index of factorial simplicity. *Psychometrika*, 39, 31-36.

Kapur S. (2003) Psychosis as a state of aberrant salience: a framework linking biology, phenomenology, and pharmacology in schizophrenia. *American Journal of Psychiatry*, 160, 13-23.

Laing RD. (1960) *The divided self: An existential study in sanity and madness*. London: Penguin Books.

Langdon R, Coltheart M. (2000) The cognitive neuropsychology of delusions. In: Coltheart M, Davies M, eds. *Pathologies of belief*. Oxford: Blackwell Publishers Ltd.

Maher B. (1988) Anomalous experience and delusional thinking: The logic of explanations. In: Oltmanns TF, Maher BA, eds. *Delusional beliefs*. Chichester: Wiley.

Maher BA. (1999) Anomalous experience in everyday life: Its significance for psychopathology. *Monist*, 82, 547-570.

Maier M, Mellers J, Toone B, Trimble M Ron MA. (2000) Schizophrenia, temporal lobe epilepsy and psychosis: an in vivo magnetic resonance spectroscopy and imaging study of the hippocampus / amygdala complex. *Psychological Medicine*, 30, 571-581.

Makarec K, Persinger MA. (1985) Temporal lobe signs: electroencephalographic validity and enhanced scores in special populations. *Perceptual and Motor Skills*, 60, 831-842.

Makarec K, Persinger MA. (1990) Electroencephalographic validation of a temporal lobe signs inventory in a normal population. *Journal of Research in Personality*, 24, 323-337.

Mason O, Claridge G, Jackson M. (1995) New scales for the assessment of schizotypy. *Personality and Individual Differences*, 18, 7-13.

Meehl PE. (1962) Schizotaxia, schizotypy, schizophrenia. *American Psychologist*, 17, 827-838.

Morrison AP, Wells A, Nothard S. (2000) Cognitive factors in predisposition to auditory and visual hallucinations. *British Journal of Clinical Psychology*, 39, 67-78.

Morrison AP, Wells A, Nothard S. (2002) Cognitive and emotional predictors of predisposition to hallucinations in non-patients. *British Journal of Clinical Psychology*, 41, 259-270.

Ohayon MM. (2000) Prevalence of hallucinations and their pathological associations in the general population. *Psychiatry Research*, 97, 153-64.



Peralta V, Cuesta MJ. (1999) Dimensional structure of psychotic symptoms: an item-level analysis of SAPS and SANS symptoms in psychotic disorders. *Schizophrenia Research*, 38, 13-26.

Peralta V, de Leon J, Cuesta MJ. (1992) Are there more than two syndromes in schizophrenia? A critique of the positive-negative dichotomy. *British Journal of Psychiatry*, 161, 335-43.

Persinger MA, Healey F. (2002) Experimental facilitation of the sensed presence: possible intercalation between the hemispheres induced by complex magnetic fields. *Journal of Nervous and Mental Disease*, 190, 533-541.

Persinger MA, Makarec K. (1987) Temporal lobe epileptic signs and correlative behaviors displayed by normal populations. *Journal of General Psychology*, 114, 179-195.

Peters ER, Garety PA. (1996) The Peters et al. Delusions Inventory (PDI): new forms for the 21-item version. *Schizophrenia Research*, 18, 119.

Peters ER, Joseph S, Day S, Garety P. (2004) Measuring delusional ideation: The 21-item Peters et al. delusions inventory (PDI). *Schizophrenia Bulletin*, 30, 1005-1016.

Peters ER, Joseph SA, Garety PA. (1999) Measurement of delusional ideation in the normal population: introducing the PDI (Peters et al. Delusions Inventory). *Schizophrenia Bulletin*, 25, 553-76.

Rado S. (1953) Dynamics and classification of disordered behaviour. *American Journal of Psychiatry*, 110, 406-416

Sackeim HA. (1998) The meaning of insight. In: Amador XF, David, AS, eds. *Insight and Psychosis (1st ed.)*. Oxford: Oxford University Press.

Schneider K. (1959) *Clinical Psychopathology*. Trans. Hamilton MW. New York: Grune and Stratton.

Serper, M., Dill, C. A., Chang, N., Kot, T., & Elliot, J. (2005) Factorial structure of the hallucinatory experience: continuity of experience in psychotic and normal individuals. *Journal of Nervous and Mental Disease*, 193, 265-272.

Sims A. (2003) *Symptoms in the mind: An introduction to descriptive psychopathology (3rd edition)*. Edinburgh: Elsevier Science Ltd.

Spauwen J, Krabbendam L, Lieb R, Wittchen HU, van Os J. (2003) Sex differences in psychosis: normal or pathological? *Schizophrenia Research*, 62, 45-9.

Spitzer M. (1995) A neurocomputational approach to delusions. *Comprehensive Psychiatry*, 36, 83-105.

Stone T, Young AW. (1997) Delusions and brain injury: The philosophy and psychology of belief. *Mind and Language*, 12, 327-364.

Strauss JS. (1969) Hallucinations and delusions as points on continua function. Rating scale evidence. *Archives of General Psychiatry*, 21, 581-586.

Thalbourne MA. (1994) Belief in the paranormal and its relationship to schizophrenia-relevant measures: a confirmatory study. *British Journal of Clinical Psychology*, 33, 78-80.

Toone BK, Garralda ME, Ron MA. (1982) The psychoses of epilepsy and the functional psychoses: a clinical and phenomenological comparison. *British Journal of Psychiatry*, 141, 256-61.

Trimble MR, Ring HA, Schmitz B. (1996) Neuropsychiatric aspects of epilepsy. In: Fogel BS, Schiffer RB, Rao SM, eds. *Neuropsychiatry*. Baltimore: Lippincott Williams & Wilkins..

Trimble MR. (1991) *The psychoses of epilepsy*. New York: Raven Press.

van Os J. (2003) Is there a continuum of psychotic experiences in the general population? *Epidemiologia e Psichiatria Sociale*, 12, 242-252.

van Os, J.; Hanssen, M.; Bijl, R.V.; and Ravelli, A. (2000) Strauss (1969) revisited: a psychosis continuum in the general population? *Schizophrenia Research*, 45, 11-20.

Verdoux H, van Os J, Maurice-Tison S, Gay B, Salamon R, Bourgeois, M. (1998) Is early adulthood a critical developmental stage for psychosis proneness? A survey of delusional ideation in normal subjects. *Schizophrenia Research*, 29, 247-54.

Verdoux H, van Os J. (2002) Psychotic symptoms in non-clinical populations and the continuum of psychosis. *Schizophrenia Research*, 54, 59-65.

Wing JK, Cooper JE, Sartorius N. (1974) *Measurement and classification of psychiatric symptoms*. Cambridge: Cambridge University Press.

## **Chapter Six**

### **The Role of the Lateral Temporal Lobes in Anomalous Perceptual Experience and Magical Thinking: A Transcranial Magnetic Stimulation Study**

Elements of this chapter are in publication as:

Bell, V., Reddy, V., Halligan, P.W., Kirov, G. & Ellis, H.D. (in press) Relative suppression of magical thinking: A transcranial magnetic stimulation study. *Cortex*

## **6.1 Chapter outline**

Sections:

- 6.2 Abstract
- 6.3 Background
- 6.4 Method
- 6.5 Results
- 6.6 Discussion
- 6.7 Chapter summary and conclusion
- 6.8 References

This chapter aims to contribute towards understanding the role of the temporal cortices in the neuropsychology of anomalous perceptual experience and delusion formation. Transcranial magnetic stimulation is a comparatively new tool in this area, but has the advantage of targeting relatively discrete areas of cortex, being non-invasive, having a temporal resolution for the onset of its effect in the millisecond range and providing evidence to support causal rather than purely correlational inferences.

This makes it an appropriate technique both to refine the understanding of the role of the temporal cortices in delusion formation (as highlighted in section 3.7.1) and to provide evidence to support the conclusion, drawn from the principal components analysis of the Cardiff Anomalous Perceptions Scale (CAPS; section 5.5.5), that temporal lobe disturbance may contribute to the overall level of anomalous perceptual experience.

## **6.2 Abstract**

The tendency to perceive meaning in noise (apophenia) has been linked to ‘magical thinking’ (MT), a distinctive form of thinking associated with a range of normal cognitive styles, anomalous perceptual experiences, delusions and frank psychosis. Important aspects of MT include the propensity to experience perceptual distortions and to imbue meaning or causality to events that might otherwise be considered coincidental. Structures in the lateral temporal lobes have been hypothesised to be involved in both the clinical and non-clinical aspects of MT. Accordingly, in this study we used single-pulse TMS to stimulate either the left or right lateral temporal areas, or the vertex, of 12 healthy participants (balanced for similar levels of magical thinking, delusional ideation and temporal lobe disturbance) while they were required to indicate if they had ‘detected’ pictures, claimed to be present by the experimenters, in visual noise. Relative to the vertex, TMS inhibition of the left lateral temporal area produced a significantly reduced tendency to report meaningful information, suggesting that left lateral temporal activation may be more important in ‘magical thinking’ and, therefore, in producing and supporting anomalous beliefs and experiences. The effect cannot simply be explained by TMS induced cognitive slowing as reaction times were not affected.

## **6.3 Background**

Discerning meaning in apparently random or non-intentional sources remains a common human experience, as anyone who has ‘seen’ pictures in clouds or reported figures in ambiguous projective psychological tests can testify. The experience of perceiving meaning in apparent noise was termed ‘apophenia’, originally to describe a critical aspect of ‘magical thinking’ commonly seen in psychotic patients who were considered to have a “specific experience of an abnormal meaningfulness” or the “unmotivated seeing of connections” (Conrad, 1958). Operationally, magical thinking describes a process by which meaningful

connections are made between semantically-distant or unrelated percepts (Brugger et al., 1993a, 1993b; Gianotti et al., 2001; Krummenacher et al., 2002; Leonhard and Brugger, 1998) and is considered by some researchers to exist on a continuum ranging from creative and unusual thought (Weinstein and Graves, 2002), to anomalous perceptual experience and frank psychosis (Johns and van Os; 2001; Peters et al., 1999; Strauss, 1969).

One of the first studies to have demonstrated the induction of apophenia in a non-clinical group of participants was conducted by Barber and Calverly (1964). Using simple suggestion, they produced an experience of 'hearing' the Bing Crosby song "White Christmas" in 54% of people played nothing but white noise. The use of stronger suggestion techniques increased this rate to 73% for a hypnosis condition and 80% for a condition using "task motivating instructions", compared with a rate of 40% in the control group.

A later study by Mintz and Alpert (1972) also found that 40% of non-clinical participants reported hearing 'White Christmas' when played nothing but white noise; but that hallucinating patients were much more likely to 'hear' the song than non-hallucinating patients. This study (and a subsequent replication by Young et al., 1987) suggested that the tendency to perceive a non-existent meaningful song from white noise is not necessarily a perception peculiar to pathological disorders, although the propensity is expected to be more consistent or stronger in people diagnosed with a psychotic illness. More recently, Merckelbach and van de Ven (2001) sought to find some of the psychological correlates of this tendency in a non-clinical population and found an association between 'detecting' the non-existent song, and measures of both fantasy- and hallucination-proneness.



Other studies have suggested that there may be general tendency to perceive “patterns in noise” all of which may account for different forms of magical thinking (Brugger et al. 1993a, Brugger and Graves, 1997; Pizzagalli et al., 2001; Gianotti et al., 2001) and that such tendencies may be explained in terms of a right hemisphere processing bias. As previously discussed in section 3.6, this link with hemisphere processing has subsequently been confirmed in a number of studies using methods that included EEG (Pizzagalli et al., 2000), lateralised word presentation (Brugger et al., 1993a; Kravetz et al., 1998; Leonhard and Brugger, 1998; Pizzagalli et al., 2001), lateralised visual noise presentation (Brugger et al., 1993b), olfactory discrimination (Mohr et al., 2001) and implicit line bisection measures (Taylor et al., 2002).

Pizzagalli et al. (2000) argue that the right hemisphere favours ‘coarse’ rather than ‘focused’ semantic processing and that a bias for right hemisphere processing may facilitate the emergence of loose and uncommon associations in both healthy participants and patients. This conclusion, however, could be seen to be at odds with some of the clinical literature which has suggested a tendency for left hemisphere hyperactivity in clinically-diagnosable psychotic disorders such as schizophrenia (review in Gur and Chin, 1999; sections 3.6; 3.7.1).

Although interesting, hemispheric accounts lack precision as to the neural systems involved in magical thinking, apophenia and associated anomalous experience (section 3.7.1).

Suggestions as to relevant brain areas have been provided by studies that have linked the temporal lobes with paranormal beliefs and anomalous perceptual experiences. Work by Persinger and colleagues (Makarec and Persinger, 1985; Persinger and Makarec, 1987; Persinger and Fisher, 1990; Skirda and Persinger, 1993) has shown that anomalous beliefs and experiences can be reliably linked to signs of temporal-lobe disturbance in both clinical

and non-clinical populations, and that anomalous experiences (including visual images, smells, emotions and the experience of a ‘sensed presence’) can be induced in some people by either applying weak complex magnetic fields (from outside the skull) over the right temporoparietal area, or to the same area bilaterally (Persinger and Healey, 2002; although see Granqvist et al., 2005; Persinger and Koren, 2005).

One relevant technique yet to be applied to the study of this phenomenon is single-pulse transcranial magnetic stimulation (TMS). TMS has a number of advantages for investigating putative neural networks, not least of which is a good spatial and temporal resolution, and the ability to safely and selectively disrupt or inhibit areas of the cortex of healthy individuals whilst they are undertaking a controlled task (Mills, 1999; Walsh and Pascual-Leone, 2003). This latter aspect allows experimental studies to move beyond the correlative designs of much of the previous work to look at the effects of neural intervention on a particular cognitive process.

This makes TMS particularly suitable for investigating the causal roles of relatively refined areas in the cortex, using well-controlled samples, not confounded by variables such as medication, diagnostic uncertainty or differing presentation. These are all issues which have been highlighted as problematic in past neuropsychological studies of delusion and anomalous perceptual experience (section 3.9).

In experimental studies, the tendency to detect non-existent meaningful information in visual noise patterns has been previously linked to magical thinking and anomalous experience (Brugger et al., 1993b) and measures of psychoticism and hallucination-proneness (Jakes and Hemsley, 1986). To further understand the hypothesised role of the lateral temporal areas in

magical thinking and the psychosis-continuum, this study recruited healthy participants within the normal range for measures of 'magical thinking', delusional ideation and unusual experiences and attempted to use TMS to try and affect perceptions of meaningful information in visual noise patterns.

## **6.4 Method**

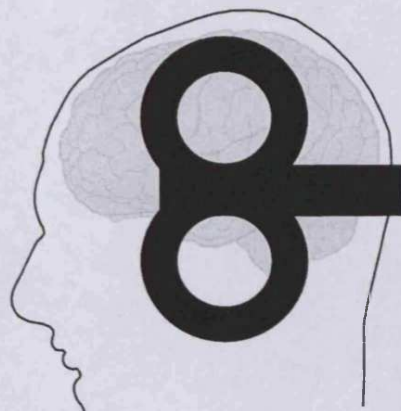
### **6.4.1 Participants**

The study involved 12 healthy participants (6 male, 6 female; mean age = 24.08, SD = 4.08). All participants were right handed. Laterality quotient for 11 of the participants was measured by the Edinburgh Handedness Inventory (Oldfield, 1971), giving a mean of +8.44 (range 5.0 - 10.0, SD = 1.82), where -10 is completely left handed and +10 is completely right handed. Participants were unaware of the exact hypothesis being investigated and were recruited on the basis that the study was investigating 'brain areas involved in perceiving and understanding ambiguous pictures'. The study was fully reviewed and approved by the appropriate local research ethics committee.

### **6.4.2 Stimulation protocol**

All participants were screened using the Transcranial magnetic stimulation Adult Safety Screen (Keel, Smith and Wasserman, 2001) and were subsequently interviewed by a medically qualified doctor to exclude anyone with a history of neurological or mental illness, current use of central nervous system medication, or any other factors contraindicated for TMS.

After screening, participants' were measured using the 10-20 system (American Electroencephalographic Society, 1991) and areas T7 (left temporal), T8 (right temporal), Cz (vertex) and C3 (left motor cortex) were marked on the scalp.

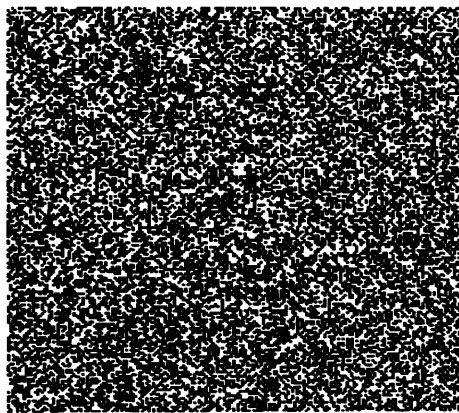


*figure 6.1 Approximate coil placement for left and right lateral temporal lobe stimulation.*

A Magstim Super Rapid system (Magstim Company Ltd, Whitland, Wales) with a 70mm figure-of-eight coil was used for TMS stimulation. The motor threshold for each participant was determined by finding the lowest stimulation strength needed to elicit visible movements of the digits of the right hand in 3 out of 6 pulses when areas around the left motor cortex were stimulated. Subsequent experimental stimulation was conducted at 110% of each participant's motor threshold, with the coil positioned flat against the skull and the handle pointing towards the posterior of the skull. During the experimental phase of the study, the coil was aligned with the appropriate 10-20 location and locked in place by use of a specifically-designed coil stand (Magstim, *ibid*) to ensure a consistent site of stimulation. Walsh and Pacual-Leone (2003) estimate that the type of figure-of-eight coil used in this study stimulates an area of 4cm x 3cm on the cortical surface. Figure 6.1 shows coil positioning for the temporal lobe stimulation conditions.

### 6.4.3 Procedure

Following the methodology of Brugger et al. (1993b), participants were told that they were about to be shown a series of dot patterns (example of pattern shown in figure 6.2) that would be briefly presented centrally on-screen, and that ‘about half’ had pictures hidden within, that might range from simple forms to more complex images. Crucially, it was stressed that participants were not to try and guess which dot patterns had pictures hidden in them, but were only to respond if they detected one of the ‘hidden images’. In reality, all of the patterns were randomly generated and none contained ‘hidden images’. Participants were asked to respond as quickly and accurately as possible by pressing one of two buttons with the right hand.



*figure 6.2 Example of random dot pattern*

Trials were presented in three blocks of 40, each consisting of 20 trials of two types (single-pulse TMS stimulation given either 100ms or 200ms before visual noise presentation) randomly ordered throughout the block. Stimulation was presented before stimulus onset to avoid potentially confounding factors such as non-specific reaction time facilitation (Terao et al., 1998) and post-pulse hesitation by participants (Walsh and Pacual-Leone, 2003, p84). Two latencies were used, both to increase the chances of finding an effect and to elucidate any

differences in online processing. Each of these latencies would allow the depolarising effect of TMS (approximately 500msec to return to baseline tissue current; Walsh and Cowey, 2000) to encompass stimulus onset, and post-stimulus latencies found effective in previous non-motor TMS studies that found inhibitory effects (see table 6.1). Each block involved stimulation to one site only; either the left temporal, right temporal or vertex area. Blocks were rotated in a Latin-squares arrangement between participants to control for order effects.

<i>Reference</i>	<i>Site</i>	<i>Task</i>	<i>Effective Latency</i>
Amassian et al (1989)	Occipital	Letter recognition	60-140msec
Hotson et al (1994)	Temporo-parieto-occipital junction		100-150msec
Düzel et al (1996)	Temporal	Serial order recall	0 and 200msec
Ashbridge et al (1997)	Parietal visual cortex	Conjunction search	100 and 160msec
Kamitani and Shimojo (1999)	Occipital	Visual observation	60-180msec
Pourtois and de Gelder (2002)	Left posterior parietal cortex	Audio visual pairing	200msec

*table 6.1 Effective pulse latencies in non-motor TMS studies.*

For each trial a central fixation point was presented for 500msec, followed by the presentation of the random dot pattern 500msec afterwards. Single-pulse TMS stimulation was given either 100msec or 200msec before random dot pattern presentation. The random dot patterns were presented for 140msec and consisted of 800 x 600 pixel (onscreen dimensions 238mm x 178mm) black and white pixels, presented on a white background in the centre of the screen (with a total screen area of 1024 x 768 pixels). The subsequent trial was presented 500msec after each response by the participant.

Participants were then asked to complete a number of scales designed to assess anomalous experiences and beliefs, namely the 21-item Peters et al. Delusions Inventory (PDI-21; Peters

and Garety, 1996; Peters et al., 2004), a measure of delusional ideation, the Magical Ideation Scale (MIS; Eckblad and Chapman, 1983) and Makarec and Persinger's Temporal Lobe Signs Inventory (TLSI; Makarec and Persinger, 1985; 1990), a measure of anomalous experience related to temporal lobe disturbance. Owing to time constraints, one participant did not complete these scales, and so data for only 11 participants are reported here.

## 6.5 Results

Statistical analysis was carried out using non-parametric tests, as they do not require assumptions about normal distribution of the data that would be difficult to justify in light of TMS intervention being used in all conditions (it is worth noting, however, there were no substantial differences in outcome when analyses were completed using parametric equivalents). Effect size (ES) for each comparison were calculated according to Cohen (1988).

When median reaction times were compared using a Wilcoxon signed rank test, there were no significant differences between median reaction times on the 100msec and 200msec conditions for any of the stimulation sites (left - 100msec vs 200msec:  $Z = -1.833$ ,  $ES = 0.53$ ,  $p = 0.60$ ; vertex - 100msec vs 200msec:  $Z = -1.647$ ,  $ES = 0.48$ ,  $p = 0.099$ ; right - 100msec vs 200msec:  $Z = -0.628$ ,  $ES = 0.18$ ,  $p = 0.530$ ). Therefore, these trials were collapsed for the purposes of further analyses (see table 6.2). A Friedman test did not reveal any effect of location site on median reaction time ( $\chi^2 = 0.167$ ,  $df = 2$ ,  $ES = 0.12$ ,  $p = 0.920$ ). Similarly, pair-wise comparisons of individual location sites using Wilcoxon signed rank tests revealed no further differences (left vs right:  $Z = -0.157$ ,  $ES = 0.05$ ,  $p = 0.875$ ; vertex vs right:  $Z = -0.196$ ,  $ES = 0.06$ ,  $p = 0.844$ ; vertex vs left:  $Z = -0.157$ ,  $ES = 0.05$ ,  $p = 0.875$ ) suggesting TMS did not induce any general cognitive slowing or response inhibition during the task.

<i>Location site</i>	<i>Mean of Median RTs (SD)</i>	<i>Mean 'detect' responses (SD)</i>
Left	1190.21msec (662.93)	2.42 (4.12)
Right	1212.75msec (708.2)	4.92 (8.6)
Vertex	1202.42msec (747.17)	5.92 (8.4)

*table 6.2. Mean median reaction times and 'detect' responses by site of TMS stimulation.*

However, when the number of 'detect' responses were compared using a Friedman test, a significant effect of location site was found ( $\chi^2 = 8.176$ ,  $df = 2$ ,  $ES = 0.83$ ,  $p < 0.05$ ). Post-hoc tests were conducted using two-tailed Wilcoxon sign rank tests to compare individual stimulation sites. In a direct comparison, there was no significant difference between 'detect' responses after right and left temporal areas had been stimulated ( $Z = -1.843$ ,  $ES = 0.53$ ,  $p = 0.065$ ). However, a differential effect was found in detection whereby there was no significant difference in responding when right stimulation was compared to vertex stimulation ( $Z = -1.279$ ,  $ES = 0.37$ ,  $p = 0.201$ ), but a significant reduction in 'detect' responses when left stimulation was compared to the vertex ( $Z = -2.433$ ,  $ES = 0.70$ ,  $p < 0.05$ ).

The standard deviations for group 'detect' responses were notably high. This was caused by one participant responding with 14, 29 and 30 'detect' responses for the left, right and vertex stimulation conditions respectively. On questioning, the participant claimed to be responding as requested during the task (indicating 'detection' of meaningful pictures rather than 'guesses'). As this participant was unremarkable in psychometric scale scores, it cannot be discounted that the participant misunderstood the instructions (although interestingly, the pattern of responses still suggest that left stimulation reduced positive responding). When this participant's data are removed from the analyses the mean 'detect' responses by site of stimulation are as follows: left (1.36;  $SD = 2.01$ ), right (2.74;  $SD = 4.24$ ), vertex (3.73;  $SD = 3.80$ ). The pattern of significance remained unaffected, however. A subsequent Friedman test



on the revised data set remained significant ( $\chi^2 = 6.467$ ,  $df = 2$ ,  $ES = 0.77$ ,  $p < 0.05$ ). The direct left - right comparison was non-significant ( $Z = -1.549$ ,  $ES = 0.47$ ,  $p = 0.121$ ), although there was still a differential effect whereby the right - vertex comparison was not significant ( $Z = -1.166$ ,  $ES = 0.35$ ,  $p = 0.244$ ); and there was still a significant 'detection' reducing effect for left stimulation when compared to the vertex ( $Z = -2.246$ ,  $ES = 0.68$ ,  $p < 0.05$ ).

The mean number of items endorsed on the PDI-21 was 4.18 (range 0 - 8,  $SD = 2.99$ ), mean MIS score was 5.08 (range 0 - 12,  $SD = 3.96$ ) and mean TLSI score was 7.0 (range 1 - 15;  $SD = 4.36$ ), suggesting the group was well within the normal range for these measures. There were no significant correlations between any of the psychometric scale scores and either the number of site-specific or total number of 'detect' responses, suggesting pre-existing levels of anomalous or paranormal beliefs or experiences did not significantly affect the number of 'detect' responses.

Barnett and Corballis (2002) have reported that consistent right-handers show lower levels of magical ideation than those with mixed-handedness, leading to a potential confound. As I wished to establish that participants were effectively homogeneous for levels of magical thinking, a two-tailed Mann-Whitney U test was used to compare scores on the PDI-21, MIS and TLSI, between consistent right-handers (those who scored 9 or 10 on the EHI;  $N = 5$ ) and inconsistent right-handers (those who scored 8 or below on the EHI;  $N = 6$ ). No significant differences were found between these groups on any of the psychometric scale scores (PDI-21 consistent vs non-consistent:  $Z = -1.588$ ,  $ES = 0.48$ ,  $p = 0.112$ ; MIS consistent vs non-consistent:  $Z = -0.188$ ,  $ES = 0.06$ ,  $p = 0.851$ ; TLS consistent vs non-consistent:  $Z = -0.092$ ,  $ES = 0.03$ ,  $p = 0.927$ ), suggesting that the findings were not likely to have been significantly influenced by this effect.

## 6.6 Discussion

The findings reported here indicate that single pulse TMS can significantly reduce the tendency to 'detect' non-existent meaningful images in visual noise by inhibiting localised areas of the left lateral temporal cortex. The tendency to perceive meaning in noise (apophenia) constitutes a distinctive component of magical thinking, a process thought by some to be important in the purported psychosis continuum. In particular the results are consistent with the idea that the temporal lobes may be important in supporting this process. The finding that reaction times were not significantly affected while 'detection' rates were, suggests that the effect was not due to any general effects of cognitive slowing or response inhibition.

Methodologically, the observed effect is interesting in itself, as the current literature considers single-pulse TMS as generally having an effect on reaction time but not other response measures such as accuracy (Walsh and Pascual-Leone, 2003). Although this study did not use a task that could be scored in terms of accuracy, the pattern of results suggests that single-pulse TMS can influence behaviour without significant cognitive slowing.

The exact mechanisms of TMS are still poorly understood and it is not impossible that the stimulation used in this study was producing a facilitatory rather than inhibitory effect in the targeted areas. There are several reasons to think this is unlikely in this case however.

Previous studies that have found facilitatory effects using single-pulse TMS (Ellison et al., 2003; Topper et al., 1998) have typically reported facilitation in terms of decreases in reaction time, which were not observed in our study. Furthermore, in the case of one report (Topper et

al., 1998), such facilitatory effects were only present with low intensity stimulation and disappeared at levels comparable to those used here.

In terms of the specific hypothesis being tested, it is notable that the differential effect of stimulation was only found relative to the vertex, rather than from the more direct comparison between left and right temporal stimulation alone. This is perhaps due to the fact that stimulation of a region in one cortical hemisphere may cause activation in the homologous area of the opposite hemisphere, albeit more weakly and after the time taken for the signal to travel across the brain (Ilmoniemi et al., 1997). This later and lesser response may still be significant, however, perhaps making it difficult to distinguish from the effect of the original site of stimulation with this paradigm, meaning only a comparison to vertex stimulation would make a differential effect apparent. It might also suggest that both lateral temporal areas may be involved in magical thinking, although one may be preferentially involved. Nevertheless, the direct left - right comparison was only marginally non-significant, and the possibility remains that this effect could become significant with a larger sample size.

An examination of the effect sizes reported in this study also suggests that this phenomenon may become more distinct with additional investigation. Cohen (1988) approximately defines small effects as 0.2, medium as 0.5 and large as 0.8, with some of the non-significant comparisons displaying small to medium effect sizes. As the small sample size used in this study does not provide adequate power to confidently accept the null hypothesis in these cases, it may be that such comparisons will become significant when further participants are tested. With this in mind, this study should be considered as an initial investigation in this area.

Nevertheless, the significant results, that typically show large or near large effect sizes, suggest that global hemisphere accounts (e.g. that right hemisphere processing bias leads to increased magical thinking; section 3.6) need to be revised. In our study, inhibiting the left lateral temporal cortex led to a reduction in the perception of meaningful information in visual noise. If it was simply the case that whole hemisphere activation was the major influence on this process, inhibiting the left temporal area should reduce the overall level of left hemisphere activation, bias processing so the right hemisphere was preferentially involved, and increase the chances of participants 'detecting' meaningful information. As this was not the case (in fact, almost the reverse was found), an alternative explanation needs to be developed to take into account neural networks on the sub-hemisphere scale.

Relevant clinical studies have typically found specific reductions in left lateral temporal lobe grey matter volume in the schizophrenia spectrum disorders (Dickey et al., 1999; McDonald et al., 2004, Pearlson et al., 1997); whereas functional neuroimaging studies have revealed increased regional cerebral blood flow in the left temporal areas, particularly the superior temporal gyrus, in patients with delusions and other 'reality distortion' symptoms (see Blackwood et al., 2001; section 3.7.1 for a review).

It is not entirely clear how these structural and functional findings relate, however, and it may be that increased blood flow may be due to areas with reduced grey matter having to do additional 'work' to maintain an adequate level of functioning. The association between these areas and 'reality distortion' experiences has typically been found with correlational studies, and it may be the case that up- or down-stream cognitive effects, or any number of other physiological factors associated with pathology could result in the findings of such studies. As mentioned earlier, one advantage of TMS studies is that they are not purely correlational. Any

effects found in such studies suggest that the targeted cortical areas are necessary for the process under investigation. In this case, the findings provide convergent evidence to accompany the imaging studies discussed section 3.7.1, which further link the temporal areas to delusion formation and 'reality distortion' experiences.

Nevertheless, the relative specificity of TMS stimulation for these temporal areas suggest that a simple correlation between relative global hemispheric activation and magical thinking is unlikely to account for all cases where magical thinking might occur. As it currently stands, the literature seems to suggest that sub-clinical measures of magical thinking are more likely to show this simple correlation, compared with formally diagnosable disorders (section 3.6). This might further suggest that there are multiple neuropsychological factors underling the psychosis-continuum rather than a single modulatory factor, as has also been suggested on the basis of the principal components analysis of the CAPS reported in section 5.5.5.

The fact that the stimulation of the temporal cortices alters the perception of meaningful information in visual noise, also provides evidence to validate the conclusion in section 5.6, that temporal lobe disturbance may contribute to the overall level of anomalous perceptual experience.

This study only measured quantitative variables, however, and there was no indication of exactly what the participants thought they saw when they indicated that they detected a 'hidden image'. An extension of this research might include measures of the qualitative aspects of falsely detected images (as has been done in the TMS visual-phosphene literature; Stewart et al. 1999), which might answer the question as to whether inhibiting the left lateral

temporal areas simply shifts response bias, or causes the perception of more complex or unusual images, or perhaps, false images of a certain type.

Certainly, temporal areas are known to be involved in semantic memory and object recognition, and activity or partial activation in these areas might induce a number of experiences of meaningfulness, of which TMS (or any other technique) may produce only a certain type. This suggests that there may be a commonality between processes that allow percepts to be understood as meaningful, and those that are thought to be up-regulated or damaged on the higher ends of the psychosis continuum.

This potential overlap and the neural basis of the 'experience of meaningfulness' may be a useful focus for future work in this area. One application of TMS that is currently being researched is its use as a treatment for various psychiatric disorders, with current paradigms focusing on longer-term suppression of cortical areas thought to be involved in producing unwanted symptoms and experiences (Fitzgerald et al., 2002). If the results in this study bear out, it may be possible to reduce levels of magical thinking by targeting the areas identified in this study. Considering the poorly-defined boundaries of delusion and insight, however, as well as the potential links between magical thinking, creativity and anomalous belief, this is an area which should be approached with caution, and certainly not without serious consideration as to ethical issues involved.

## **6.7 Chapter summary and conclusion**

The study presented here suggests that, in accordance with the results from the principal components analysis reported in Chapter 5 (section 5.5.5), temporal lobe disturbance may be a significant contributory factor to the overall level of anomalous perceptual experience and the psychosis continuum. Unlike many of the imaging studies discussed in section 3.7.1, this provides evidence that the temporal lobes (particularly the left lateral temporal cortex) may play a causal role in this process.

In light of evidence presented in Chapter 5, concerning the distribution of the link between anomalous perceptual experience and delusional ideation, it is interesting to speculate that this may be one of the factors that mediate the likely cognitive connection between perceptual distortion and magical or delusional interpretation.

What this study does not provide, however, is strong clues as to the pathological elements of a delusional or magical belief. In fact, evidence also presented in Chapter 5 (from the item frequency rank analysis; section 5.5.6), suggested that temporal lobe phenomena were more likely to be associated with the non-clinical rather than clinical group.

The interaction between distress and anomalous perceptual experience in producing clinically delusional, rather than simply 'unusual' or 'anomalous' beliefs is, therefore, tackled in Chapter 7 in an attempt to answer this question.

## 6.8 References

- American Electroencephalographic Society. (1991) American Electroencephalographic Society guidelines for standard electrode position nomenclature. *Journal of Clinical Neurophysiology*, 8, 200-2.
- Amassian VE, Cracco RQ, Maccabee PJ, Cracco JB, Rudell A and Eberle L. (1989) Suppression of visual perception by magnetic coil stimulation of human occipital cortex. *Electroencephalography and Clinical Neurophysiology*. 74, 458-62.
- Ashbridge E, Walsh V and Cowey A. Temporal aspects of visual search studied by transcranial magnetic stimulation. *Neuropsychologia*, 35: 1121-31, 1997.
- Barber TX and Calverley DS. (1964) An experimental study of “hypnotic” (auditory and visual) hallucinations. *Journal of Abnormal Psychology*, 68, 13-20.
- Barnett KJ and Corballis MC. (2002) Ambidexterity and magical ideation. *Laterality*, 7, 75-84.
- Blackwood NJ, Howard RJ, Bentall RP and Murray RM (2001) Cognitive neuropsychiatric models of persecutory delusions. *American Journal of Psychiatry*, 158, 527-39.
- Brugger P, Gamma A, Muri R, Schafer M and Taylor KI. (1993a) Functional hemispheric asymmetry and belief in ESP: towards a “neuropsychology of belief”. *Perceptual and Motor Skills*, 77, 1299-1308.



Brugger P and Graves RE. Testing vs. believing hypotheses: Magical ideation in the judgement of contingencies. *Cognitive Neuropsychiatry*, 2, 251-272, 1997.

Brugger P, Regard M, Landis T, Cook N, Krebs D And Niederberger J. (1993b) 'Meaningful' patterns in visual noise: effects of lateral stimulation and the observer's belief in ESP. *Psychopathology*, 26, 261-265, 1993b.

Cohen, J. (1988) *Statistical Power Analysis for the Behavioural Sciences (2nd ed)*. New Jersey: Lawrence Erlbaum Associates.

Conrad K (1958) *Die beginnende Schizophrenie. Versuch einer Gestaltanalyse des Wahns*. Stuttgart: Thieme.

Dickey CC, Mccarley RW, Voglmaier MM, Niznikiewicz MA, Seidman LJ, Hirayasu Y, Fischer I, Teh EK, Van Rhoads R, Jakab M, Kikinis R, Jolesz FA, Shenton ME. (1999) Schizotypal personality disorder and MRI abnormalities of temporal lobe gray matter. *Biological Psychiatry*, 45, 1393-402.

Duzel E, Hufnagel A, Helmstaedter C and Elger C. (1996) Verbal working memory components can be selectively influenced by transcranial magnetic stimulation in patients with left temporal lobe epilepsy. *Neuropsychologia*, 34, 775-83.

Eckblad M and Chapman LJ. (1983) Magical ideation as an indicator of schizotypy. *Journal of Consulting and Clinical Psychology*, 51, 215-225.

Ellison A, Battelli L, Cowey A and Walsh V (2003) The effect of expectation on facilitation of colour/form conjunction tasks by TMS over area V5. *Neuropsychologia*, 41, 1794-1801.

Fitzgerald PB, Brown TL and Daskalakis ZJ. (2002) The application of transcranial magnetic stimulation in psychiatry and neurosciences research. *Acta Psychiatrica Scandinavica*, 105: 324-340, 2002.

Gianotti LR, Mohr C, Pizzagalli D, Lehmann D, Brugger P. (2001) Associative processing and paranormal belief. *Psychiatry and Clinical Neurosciences*, 55, 595-603.

Granqvist P, Fredrikson P, Unge P, Hagenfeldt A, Valind S, Larhammar D, Larsson, M. (2005) Sensed presence and mystical experiences are predicted by suggestibility, not by the application of transcranial weak complex magnetic fields. *Neuroscience Letters*, 379, 1-6.

Gur RE and Chin S. (1999) Laterality in functional brain imaging studies of schizophrenia. *Schizophrenia Bulletin*, 25, 141-156.

Hotson J, Braun D, Herzberg W and Boman D. (1994) Transcranial magnetic stimulation of extrastriate cortex degrades human motion direction discrimination. *Vision Research*, 34, 2115-23.

Ilmoniemi RJ, Virtanen J, Ruohonen J, Karhu J, Aronen HJ, Naatanen R and Katila T. (1997) Neuronal responses to magnetic stimulation reveal cortical reactivity and connectivity. *Neuroreport*, 8, 3537-40.

Jakes S and Hemsley DR. (1986) Individual differences in reaction to brief exposure of to unpatterned visual stimulation. *Personality and Individual Differences*, 7, 121-123.

Johns LC and van Os, J. (2001) The continuity of psychotic experiences in the general population. *Clinical Psychology Review*, 21, 1125-41.

Kamitani Y and Shimojo S. (1999) Manifestation of scotomas created by transcranial magnetic stimulation of human visual cortex. *Nature Neuroscience*, 2, 767-71.

Keel JC, Smith MJ and Wassermann EM. (2001) A safety screening questionnaire for transcranial magnetic stimulation. *Clinical Neurophysiology*, 112, 720.

Kravetz S, Faust M and Edelman A. (1998) Dimensions of schizotypy and lexical decision in the two hemispheres. *Personality and Individual Differences*, 25, 857-871.

Krummenacher P, Brugger P, Fathi M and Mohr C. (2002) *Dopamine, paranormal ideation and the detection of meaningful stimuli*. Poster presented at the Zentrum fur Neurowissenschaften Zurich, Zurich.

Leonhard D and Brugger P. (1998) Creative, paranormal, and delusional thought: a consequence of right hemisphere semantic activation? *Neuropsychiatry, Neuropsychology and Behavioural Neurology*, 11, 177-183.

Makarec K and Persinger MA. (1985) Temporal lobe signs: electroencephalographic validity and enhanced scores in special populations. *Perceptual and Motor Skills*, 60, 831-842.

Makarec K and Persinger MA. (1990) Electroencephalographic validation of a temporal lobe signs inventory in a normal population. *Journal of Research in Personality*, 24: 323-337.

McDonald C, Bullmore ET, Sham PC, Chitnis X, Wickham H, Bramon E and Murray RM. Association of genetic risks for schizophrenia and bipolar disorder with specific and generic brain structural endophenotypes. *Archives of General Psychiatry*, 61, 974-84.

Merckelbach H and van de Ven V. (2001) Another White Christmas: Fantasy proneness and reports of 'hallucinatory experiences' in undergraduate students. *Journal of Behavior Therapy and Experimental Psychiatry*, 32, 137-144.

Mills, KR. (1999) *Magnetic Stimulation of the Human Nervous System*. Oxford: Oxford University Press.

Mintz S and Alpert M. (1972) Imagery vividness, reality testing, and schizophrenic hallucinations. *Journal of Abnormal Psychology*, 79, 310-316.

Mohr C, Rohrenbach CM, Laska M and Brugger P. (2001) Unilateral olfactory perception and magical ideation. *Schizophrenia Research*, 47, 255-264.

Oldfield, RC. (1971) The assessment and analysis of handedness: the Edinburgh inventory. *Neuropsychologia*, 9: 97-113.

Pearlson GD, Barta PE, Powers RE, Menon RR, Richards SS, Aylward EH, Federman EB, Chase GA, Petty RG and Tien AY. (1997) Ziskind-Somerfeld Research Award 1996. Medial and superior temporal gyral volumes and cerebral asymmetry in schizophrenia versus bipolar disorder. *Biological Psychiatry*, 41, 1-14.

Persinger MA and Fisher SD. (1990) Elevated, specific temporal lobe signs in a population engaged in psychic studies. *Perceptual and Motor Skills*, 71, 817-818.

Persinger MA and Healey F. (2002) Experimental facilitation of the sensed presence: possible intercalation between the hemispheres induced by complex magnetic fields. *Journal of Nervous and Mental Disease*, 190, 533-541.

Persinger, M. A., Koren, S. A. (2005) A response to Granqvist et al. "Sensed presence and mystical experiences are predicted by suggestibility, not by the application of transcranial weak magnetic fields". *Neuroscience Letters*, 380, 346-347; author reply 348-350.

Persinger MA and Makarec, K. (1987) Temporal lobe epileptic signs and correlative behaviors displayed by normal populations. *Journal of General Psychology*, 114, 179-195.

Peters ER, Joseph S, Day S, Garety P. (2004) Measuring delusional ideation: The 21-item Peters et al. delusions inventory (PDI). *Schizophrenia Bulletin*, 30, 1005-1016.

Peters ER, Joseph SA, and Garety PA. (1999) Measurement of delusional ideation in the normal population: introducing the PDI (Peters et al. Delusions Inventory). *Schizophrenia Bulletin*, 25, 553-76.

Peters ER and Garety PA. (1996) The Peters et al. Delusions Inventory (PDI): new forms for the 21-item version. *Schizophrenia Research*, 18, 119.

Pizzagalli D, Lehmann D and Brugger P. (2001) Lateralized direct and indirect semantic priming effects in subjects with paranormal experiences and beliefs. *Psychopathology*, 34, 75-80.

Pizzagalli D, Lehmann D, Gianotti L, Koenig T, Tanaka H, Wackermann J and Brugger P. (2000) Brain electric correlates of strong belief in paranormal phenomena: intracerebral EEG source and regional Omega complexity analyses. *Psychiatry Research*, 100, 139-154.

Pourtois G and de Gelder B. (2002) Semantic factors influence multisensory pairing: a transcranial magnetic stimulation study. *Neuroreport*, 13, 1567-73.

Skirda RJ and Persinger MA. (1993) Positive associations among dichotic listening errors, complex partial epileptic-like signs, and paranormal beliefs. *Journal of Nervous and Mental Disease*, 181, 663-667.

Stewart LM, Battelli L, Walsh V and Cowey A. (1999) Motion perception and perceptual learning studied by magnetic stimulation. *Electroencephalography and Clinical Neurophysiology*, 51, 334-350.

Strauss JS. (1969) Hallucinations and delusions as points on continua function. Rating scale evidence. *Archives of General Psychiatry*, 21, 581-6.

Taylor KI, Zach P and Brugger P. (2002) Why is magical ideation related to leftward deviation on an implicit line bisection task? *Cortex*, 38, 247-252.

Terao Y, Ugawa Y, Sakai K, Miyauchi S, Fukuda H, Sasaki Y, Takino R, Hanajima R, Furubayashi T, Putz B and Kanazawa I. (1998) Localizing the site of magnetic brain stimulation by functional MRI. *Experimental Brain Research*, 121, 145-52.

Topper R, Mottaghy FM, Brugmann M, Noth J and Huber W. (1998) Facilitation of picture naming by focal transcranial magnetic stimulation of Wernicke's area. *Experimental Brain Research*, 121, 371-8.

Walsh V and Cowey A. (2000) Transcranial magnetic stimulation and cognitive neuroscience. *Nature Review Neuroscience*, 1, 73-79.

Walsh V and Pascual-Leone A. (2003) *Transcranial magnetic stimulation: A neurochronometrics of mind*. MIT: MIT Press.

Weinstein S and Graves RE. (2002) Are creativity and schizotypy products of a right hemisphere bias? *Brain and Cognition*, 49, 138-151.

Young HF, Bentall RP, Slade PD and Dewey ME. (1987) The role of brief instructions and suggestibility in the elicitation of hallucinations in normal and psychiatric subjects. *Journal of Nervous and Mental Disease*, 175, 41-48.

## **Chapter Seven**

### **Delusions, Anomalous Experience and Distress**

The following presentation has included material from this chapter:

Bell, V., Halligan, P.W. & Ellis, H.D. Delusions: A Cognitive / Emotional Approach.  
*Experimental Psychology Society*, April 2005.



## **7.1 Chapter outline**

Sections:

7.2 Abstract

7.3 Background

7.4 Method

7.5 Results

7.6 Discussion

7.7 Chapter summary and conclusion

7.8 References

It is now doubtful whether the current diagnostic criteria for delusions sufficiently capture the relevant factors that make a belief pathological. Distress is perhaps the most obvious omission from the current definition, which is a current clinical occurrence in people diagnosed as delusional. Following Chapter 5, which examined the role of anomalous perceptual experience in delusion formation, this chapter aims to examine the interaction of distress and anomalous experience in producing clinically relevant pathological beliefs.

## **7.2 Abstract**

Previous research has indicated that certain groups of people, particularly from recently created or revived religious movements, may experience high levels of psychosis-like experience but avoid significant levels of distress or impairment. Earlier studies have used psychometric scales that typically do not adequately differentiate between anomalous perceptual experience and schizotypy or delusional ideation. This study used the Cardiff Anomalous Perceptions Scale (CAPS) and the Peters et al. Delusions Inventory (PDI) to examine the relationship between perceptual distortion, delusional ideation and distress, in

groups of atheists, agnostics and humanists (N=26), Christians (N=32), Pagans (N=142), participants not selected for religious beliefs (N=36), deluded patients with hallucinations (N=24), deluded patients without hallucinations (N=24) and unselected psychotic patients (N=20). Two scoring methods were used, and the results suggest that the original CAPS and PDI scoring method overestimates the negative effects of anomalous experience in religious samples. Pagans were found to have similar levels of anomalous perceptual experience to psychotic inpatients and deluded patients with hallucinations, but suffer no more perceptual distortion-related distress than the general population. A comparison between the Pagan sample and deluded patients without hallucinations indicates a double-dissociation between anomalous perceptual experience and distress, suggesting that these factors can be experienced relatively independently, and that distress, rather than perceptual distortion, may be the more important factor in some types of delusion.

### **7.3 Background**

Diagnostic approaches to delusion (e.g. as described in the DSM; section 2.2) aim to provide the necessary conditions with which to classify a belief as pathological. A major drawback with this approach is that the given criteria have either not been shown to be necessary on empirical grounds, or not to apply on conceptual grounds (section 2.4).

Theoretical approaches have also attempted to give the necessary conditions for delusion formation. Both one-stage and two-stage models (sections 3.2.1; 3.2.2) argue that the presence of anomalous perceptual experience is a necessary condition. Other models, although not necessarily suggesting that anomalous experience is a necessary condition, still argue that it is one of the major influences on delusion formation and maintenance (e.g. IoP model, section 3.4; metacognitive models, section 3.5; aberrant salience models, section 3.7.2).

These approaches, however, largely ignore the pragmatic process by which a person becomes diagnosed as delusional (Bell et al., 2003). For example, a diagnosis entails that a person must come to the attention of a psychiatrist, mental health clinician or primary care practitioner. A person is only likely to come to the attention of such a clinician if they are experiencing distress, or causing social stress (they are a problem to other people), or, perhaps, both.

As psychiatrists typically only see those who fulfil these 'pragmatic criteria' for diagnosis, the written criteria for diagnosis (derived from descriptive psychopathology) are likely to be based on a sample bias. While there may be people who fulfil the diagnostic criteria and are in need of help, there may also be people who fulfil the diagnostic criteria who display no significant impairment or disability, and remain as virtually invisible counter-examples to the written criteria.

Recent studies based on the continuum model of psychosis (section 2.5.3) suggest there are likely to be a significant number of people in the population who would otherwise fulfil the diagnostic criteria, but do not come into contact with, or have no need of, clinical services.

Johns and van Os (2001) note that the psychosis continuum, conceptualised as the total level of anomalous beliefs and experiences, is unlikely to give a full understanding of what makes an experience pathological:

Therefore, as far as the patient - non patient distinction is concerned, it may be important to consider two interacting risks: one that determines which position a person is going to occupy along the psychosis continuum, and one that determines whether a person at a certain point is going to develop illness behaviour.

While high levels of psychosis-like experience may make it more likely that a person will display 'illness behaviour' (impairment or disability), the interaction between such phenomena and how a person appraises or assimilates these experiences, would be, according to Johns and van Os, the factor that mediates illness behaviour, of which distress is a prime component. This approach would also predict that there might be individuals who experience high levels of psychosis-like experience, but show no significant illness behaviour.

Indeed, psychometric studies of both the general population and groups likely to display high levels of psychosis-like experience have provided support for this conjecture. Two large-scale surveys indicated that 10-15% of the general population have experienced some form of hallucinatory experience in clear consciousness (Tien, 1991; Ohayon, 2000). Peters et al. (1999a; 2004) reported that 10-11% of the general population scored above the mean of a group of deluded inpatients on the Peters et al. Delusions Inventory (PDI), a figure that was closely matched by results from the Cardiff Anomalous Perceptions Scale (CAPS; section 5.5.1).

The principal components study of the CAPS (section 5.5.5) suggested that temporal lobe disturbance may be a contributory factor to anomalous perceptual experience (section 5.6). Notably, temporal lobe disturbance has also been linked to both perceptual distortion, religiosity and spiritual experience, both in temporal lobe epilepsy (Geschwind, 1983) as well as in non-clinical measure of temporal lobe lability (Makarec and Persinger, 1985; Persinger, 1983; Skirda and Persinger, 1993).

Previous work that has compared features of religious or spiritual experience and psychosis, has concluded that there are strong parallels in many cases (review in Clarke, 2001). One influential paper by Jackson and Fulford (1997) reported three individuals who fulfilled the diagnostic criteria for psychotic disorders, but whose experiences led to beneficial results. Although some authors have attempted to differentiate spiritual experience from psychosis, it is noteworthy that this has not been based on the Jasperian 'form' of the experience, used extensively in psychiatric diagnosis (Sims, 2003), as it is thought inadequately to differentiate the two states (Brett, 2003; Marzanski and Bratton, 2003; Pierre, 2001; section 2.4.4).

Peters and colleagues (Day and Peters, 1999; Peters et al., 1999b) took a psychometric approach and recruited members of 'New Religious Movements' (NRMs) (Druidry and Hare Krishna practitioners) on the basis of their holding anomalous spiritual beliefs and experiencing paranormal phenomena. The researchers found that members of NRMs scored significantly more highly than the general population on measures of schizotypy (Day and Peters, 1999) and were undifferentiated on PDI score in comparison with deluded inpatients (Peters et al., 1999b), although the religious sample were significantly less distressed and preoccupied with their beliefs.

One spiritual tradition sometimes classified as a NRM (although not without controversy) is Paganism (sometimes called Neo-Paganism). Paganism is an umbrella term for a number of spiritual traditions involving a polytheistic or pantheistic views of spirituality, typically involving nature-worshipping rituals and ceremonies (Harvey and Hardman, 1996). Many of the most popular forms of Paganism (such as Wicca) have been particularly influenced by ideas from the Victorian and early twentieth century occult revival, and involve the use of ceremonial magick and the belief in paranormal phenomena (Hutton, 2001). This suggests

that practising Pagans might score quite highly on psychosis-continuum measures owing to the strong 'magical thinking' component (section 6.3).

This is supported by evidence from a national survey of "witches and Neo-Pagans" conducted in the US (Berger et al., 2003). When compared to the general population, Pagans typically reported a 20-30% greater prevalence of the experiences 'thought you were somewhere you had been before', 'felt as though you were in touch with someone when they were far away from you', 'saw events that happened at a great distance as they were happening' and 'felt as though you were in touch with someone who had died'. The experience 'felt as though you were very close to a powerful spiritual force' was almost 50% more prevalent in the Pagan sample. Moreover, 64.3% of Pagan respondents reported being educated at least to college level or its equivalent (in comparison to 51% of the American public having only high school education); and only 2% reported being unemployed, suggesting a high level of social and occupational functioning.

These studies suggest that Pagans might represent the sorts of counter-examples to the written diagnostic criteria (and indeed, some theoretical models) that have been highlighted earlier as not being sufficiently considered when the necessary conditions for delusions were drawn up.

One problem with previous psychometric studies, however, has been the use of measures of schizotypy and delusional ideation that often conflate perceptual distortion, unusual thoughts and anomalous belief into a single measure; or are not sufficiently neutral to avoid potential biases from clinical psychiatry (section 5.3).

The development of the CAPS (Chapter 5) has produced a valid, reliable measure of anomalous perceptual experience, that also includes measures of distress, intrusiveness and frequency, allowing the interaction of perceptual distortion and aspects of illness behaviour to be examined in a single measure.

Consequently, this study aimed to examine the association between anomalous perceptual experience, distress and delusions by comparing psychotic inpatients, a general population sample, a group with mainstream spiritual beliefs (Christians), a group with unconventional spiritual beliefs (Pagans) and a group who were specifically non-religious (atheists, agnostics and humanists).

## **7.4 Method**

The study used a between subjects design with seven groups of participants. Three of the groups were clinical samples and four were non-clinical.

### **7.4.1 Participants**

Three clinical groups are the same samples reported in Chapter 5, namely inpatients with unselected psychotic symptoms ( $N = 20$ ), deluded inpatients with hallucinations ( $N = 24$ ) and deluded patients without hallucinations ( $N = 24$ ). Diagnoses was originally made by the responsible clinician, and confirmed by a review of the case notes and administration of the Psychosis Screening Questionnaire (Bebbington and Nayani, 1995) for the unselected psychotic samples, and the Scale for the Assessment of Positive Symptoms (SAPS; Andreasen, 1994) for the other clinical samples. See section 5.4.2.2 for further demographic details.

The four non-clinical groups were atheists, agnostics and humanists (AAH), Christians, Pagans and an 'unselected public' sample.

The atheist, agnostic and humanist group consisted of 26 participants (males = 20; females = 6) who had a mean age of 47.4 (SD = 15.2; range 20 – 70). They were recruited via an electronic circular distributed by an British Humanist Association, and were invited to take part in a study on “how people acquire and maintain their beliefs” with a focus on “beliefs in non-religious people”.

The Christian group consisted on 32 participants (males = 16; females = 16) who had a mean age of 45.8 (SD = 15.6; range 21 – 86). They were recruited via UK Christian internet message boards, notices in local churches in Cardiff, and copies were made available anonymously to students at a local Anglican theological college. Participants were invited to take part in a study on “how people acquire and maintain their beliefs” with a focus on “Christian beliefs”.

The Pagan group consisted of 142 participants (males = 56; females = 79; not disclosed = 7) who had a mean age of 38.0 (SD = 13.5; range 13 – 85). They were recruited via UK Pagan internet message boards and an advert in Pagan Dawn magazine, and were invited to take part in a study on “how people acquire and maintain their beliefs” with a focus on “Pagan beliefs”.

The unselected public group consisted of 36 participants (males = 18; females = 17; not disclosed = 1) who had a mean age of 33.2 (SD = 10.9; range 18-54). Of these participants, 32 were previously as part of the control group in Chapter 5 (section 5.4.2.1). They were recruited via UK internet message boards for 'miscellaneous topics' and general discussion



(such as the Usenet group uk.misc) and were invited to take part in a study on “how people acquire and maintain their beliefs”. No reference was made to religion or spiritual belief.

Several respondents asked for a number of packs to be sent so they could be anonymously distributed to friends or local religious groups, and some questionnaires were returned as photocopies, so it is not possible to accurately judge the response rate. From the figures available, it is estimated that the response rate was between 50-60% for all groups except the Christian sample, who responded at a rate of about 40%. This lower rate, however, may be due to a number of questionnaire packs being sent to the Anglican college, where they were given to all students in a class, rather than being individually requested by interested participants.

#### **7.4.2 Procedure**

The advertisements for the study invited participants to send their name and address to receive a questionnaire pack returnable in a pre-paid envelope. Separate email addresses were created to allow the researchers to identify which adverts participants were responding to, or, if they responded by phone, were asked where they had seen the advert. Participants were asked to complete and return the questionnaires anonymously.

All non-clinical participants received a copy of the 21-item Peters et al. Delusions Inventory (PDI-21; Peters et al., 2004), the Cardiff Anomalous Perceptions Scale (CAPS; Chapter 5, Bell et al., 2005) and a demographics page. This asked about age, gender, whether the participant was currently being treated for an active mental illness, was currently being treated for an active neurological illness or injury, had been treated for a mental illness in the past, or

had been treated for a neurological illness or injury in the past. These latter items were presented as yes / no questions, and no further details were requested.

Christian participants and a subgroup of Pagan participants (N=56) were also sent the Spiritual Transcendence Index (STIN; Seidlitz et al., 2002), a measure of the “perceived experience of the sacred that affects one’s self-perception, feelings, goals, and ability to transcend one’s difficulties” that has a possible range of scores from 6 to 48. Because the STIN asks specific question about “God” which may not be fully applicable to the Pagan sample, we prepended the following statement to the questionnaire given to both Pagan and Christian groups: “We realise that not everyone will use the word ‘God’ to best describe their own spirituality. However, we would appreciate it if you could interpret these questions in terms of your own personal understanding of the divine”.

The Christian group additionally received a copy of the short form of the Christian Orthodoxy Scale (COS; Hunsberger, 1989) that has a possible range of scores from 6 to 42.

As described in Chapter 5, the ‘unselected psychotic’ sample completed both the PDI-21 and the CAPS, whereas the deluded inpatients with hallucinations and deluded patients without hallucinations only completed the CAPS.

Both the PDI-21 and CAPS use a scoring method whereby responding ‘yes’ to an item, requires the participant to rate the experience on a scale from 1-5 for the subscales of distress, preoccupation and conviction (in the case of the PDI-21) or distress, intrusiveness and frequency (for the CAPS). Responding ‘no’ to an item automatically gives a score of zero on all three subscale ratings for that item.

It was hypothesised that this scoring method might substantially overestimate the level of distress in people with spiritual beliefs or experiences, as indicating the presence of a benign or beneficial psychosis-like experience automatically produces a distress score of 1, despite the fact that this is labelled as 'Not at all distressing' on the questionnaires. In contrast, someone not marking an experience or belief as present scores zero.

To test this hypothesis, we recoded the subscales scores on a scale of 0-4 during additional analyses of the results.

## **7.5 Results**

### **7.5.1 PDI scores**

#### *7.5.1.1 PDI total score*

Total score and subscale scores are displayed in table 7.1. When total score is compared using a one-way between subjects ANOVA there is a significant main effect for group ( $F_{(4,251)} = 23.938, p < .0005$ ). When individuals groups are compared using post-hoc Scheffe tests the Pagan group did not score significantly differently from the Christian group ( $p = .227$ ) but did score significantly differently from the AAH, unselected public and unselected psychotic groups (at least  $p < 0.005$ ). This suggests that Pagans and Christians are undifferentiated on levels of delusional ideation, although Pagans report significantly less delusional ideation than psychotic inpatients, but significantly more than non-religious people or the general public.

<i>Group</i>	<i>PDI Total</i>	<i>PDI Subscales</i>					
		<i>Original scoring</i>			<i>Revised scoring</i>		
		<i>Distress</i>	<i>Preoc</i>	<i>Conv</i>	<i>Distress</i>	<i>Preoc</i>	<i>Conv</i>
AAH	3.12 (2.37)	5.65 (6.25)	6.04 (5.92)	7.81 (6.84)	2.54 (4.25)	2.92 (3.81)	4.69 (4.72)
Public	5.05 (3.39)	10.92 (7.14)	9.76 (7.92)	14.68 (11.70)	5.89 (4.94)	4.73 (5.39)	9.65 (8.67)
Christian	5.97 (2.73)	11.48 (7.77)	14.52 (7.53)	20.69 (10.05)	5.52 (5.81)	8.55 (5.30)	14.76 (7.55)
Pagan	7.59 (3.39)	13.65 (9.20)	18.40 (11.42)	27.24 (12.99)	6.06 (6.52)	10.86 (8.65)	19.67 (9.95)
Psychotic	11.8 (4.64)	36.75 (20.81)	36.95 (22.46)	45.45 (24.23)	24.95 (16.88)	25.15 (18.51)	33.65 (20.26)

*table 7.1 PDI-21 scores using original and revised scoring*

#### *7.5.1.2 PDI subscale scores*

One-way between subjects ANOVAs also found significant effects for group for all PDI subscales for both the original scoring method (distress:  $F_{(4,251)} = 32.136, p < .0005$ ; preoccupation:  $F_{(4,251)} = 25.678, p < .0005$ ; conviction:  $F_{(4,251)} = 29.894, p < .0005$ ) and the revised scoring method (distress:  $F_{(4,251)} = 32.774, p < .0005$ ; preoccupation:  $F_{(4,251)} = 22.933, p < .0005$ ; conviction:  $F_{(4,251)} = 29.565, p < .0005$ )

Post-hoc Scheffe tests were conducted to examine the differences between the Pagan group and other groups on the PDI subscales. The results for both original and revised scoring are displayed in table 7.2. Non-significant differences are denoted by 'NSD', significant differences are denoted by the presence of an effect size. The key denotes significance level.

<i>Comparison groups</i>	<i>Pagan group PDI subscale scores</i>					
	<i>Original Scoring Method</i>			<i>Revised Scoring Method</i>		
	<i>Distress</i>	<i>Preoc</i>	<i>Conv</i>	<i>Distress</i>	<i>Preoc</i>	<i>Conv</i>
AAH	.86 <sup>b</sup>	1.06 <sup>c</sup>	1.38 <sup>c</sup>	NSD	.93 <sup>b</sup>	1.39 <sup>c</sup>
Public	NSD	.76 <sup>b</sup>	.92 <sup>c</sup>	NSD	.73 <sup>a</sup>	.95 <sup>c</sup>
Christian	NSD	NSD	NSD	NSD	NSD	NSD
Psychotic	-1.71 <sup>c</sup>	-1.28 <sup>c</sup>	-1.15 <sup>c</sup>	-1.81 <sup>c</sup>	-1.27 <sup>c</sup>	-1.12 <sup>c</sup>

NSD = No significant difference; <sup>a</sup> = significant difference at least  $p < .05$ ; <sup>b</sup> = significant difference at least  $p < .005$ ; <sup>c</sup> significant difference at least  $p = < .0005$ . Negative effect sizes indicate Pagan group scored less than other group, a positive effect size indicates the reverse.

*table 7.2 Effect sizes of significant post-hoc tests (Scheffe) between Pagans and other groups for both original and revised PDI scoring method*

As can be seen from table 7.2, the revised scoring method alters the pattern of significances for distress, causing the Pagan group to appear more pathological when using the original scoring method. When the revised scoring method is used, the Pagan group no longer reported significantly more PDI-related distress than the AAH group. Furthermore, the effect sizes for distress typically became larger when compared to the unselected psychotic group.

Percentage differences between original and revised distress scores are given in figure 7.1. It is clear from this graph that it is the explicitly religious groups (Pagan and Christian), and the explicitly non-religious group (AAH), who show the greatest difference in distress scores when the revised scores are used. The unselected psychotic group show the least change in distress scores. This further suggests that the original PDI scoring method over-estimates the distress caused by delusional ideation in Pagans.

Interestingly, despite the Pagan group being undifferentiated on reported distress (revised scoring method) they report significantly higher levels of preoccupation and conviction than

the AAH and unselected public group, suggesting that these factors may not necessarily be distressing in themselves.

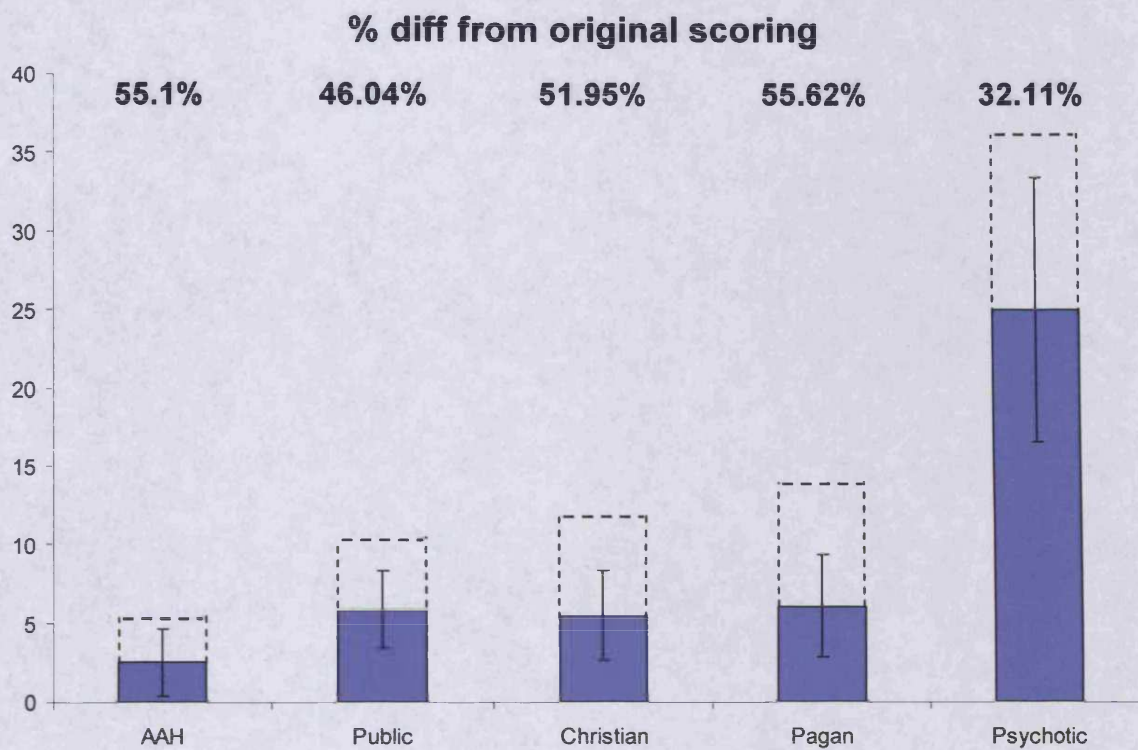


figure 7.1 Difference between original and revised PDI distress score as percentages. Original distress scores shown as outlines.

## 7.5.2 CAPS scores

### 7.5.2.1 CAPS total score

Two additional groups completed the CAPS who did not complete the PDI, namely a group of delusional inpatients with hallucinations, and a group of delusional inpatients without hallucinations. Total score and subscale scores for the CAPS are displayed in table 7.3.

When total score was compared using a one-way between subjects ANOVA there is a significant main effect for group ( $F_{(6,298)} = 13.648, p < .0005$ ). When individual groups were

compared using post-hoc Scheffe tests, the Pagan group did not score significantly differently from the unselected psychotic ( $p = .525$ ) and deluded patients with hallucinations groups ( $p = .999$ ), but did score significantly differently from the AAH, deluded without hallucinations, unselected public and Christian groups. This suggests that Pagans show equal levels of anomalous perceptual experience to deluded patients with hallucinations and psychotic inpatients, which is at a significantly greater level than mainstream religious, non-religious and a general public sample.

<i>Group</i>	<i>CAPS Subscales</i>						
	<i>CAPS Total</i>	<i>Original scoring</i>			<i>Revised scoring</i>		
		<i>Distress</i>	<i>Intrus</i>	<i>Freq</i>	<i>Distress</i>	<i>Intrus</i>	<i>Freq</i>
AAH	4.69 (4.47)	8.19 (9.7)	10.77 (12.19)	8.58 (10.12)	3.50 (6.26)	6.08 (8.57)	3.88 (6.13)
Deluded, no Hal.	6.0 (5.24)	19.08 (18.49)	17.17 (17.0)	17.42 (14.98)	12.04 (13.67)	11.13 (12.60)	11.38 (10.93)
Public	6.32 (5.67)	12.16 (12.43)	15.22 (14.72)	11.76 (12.17)	5.89 (7.61)	8.95 (9.53)	5.46 (7.23)
Christian	6.16 (5.67)	10.72 (13.26)	14.47 (18.32)	12.22 (13.84)	4.63 (9.04)	8.56 (13.17)	6.03 (8.63)
Deluded, with Hal.	11.42 (7.25)	32.08 (27.54)	32.08 (27.53)	40.17 (31.87)	20.67 (21.71)	20.67 (21.60)	28.75 (25.39)
Pagan	12.27 (7.09)	20.40 (13.38)	27.30 (19.53)	30.51 (24.53)	8.14 (8.46)	15.04 (13.86)	18.25 (18.32)
Psychotic	15.85 (8.45)	46.95 (36.77)	50.20 (35.82)	50.20 (40.08)	31.10 (30.76)	34.35 (29.38)	34.40 (32.86)

*table 7.3 CAPS scores using original and revised scoring*

7.5.2.2 CAPS subscale scores

One-way between subjects ANOVAs also found significant effects for group for all CAPS subscales for both the original scoring method (distress:  $F_{(6,298)} = 14.816, p < .0005$ ; intrusiveness:  $F_{(6,298)} = 11.306, p < .0005$ ; frequency:  $F_{(6,298)} = 13.408, p < .0005$ ) and the revised scoring method (distress:  $F_{(6,298)} = 15.261, p < .0005$ ; intrusiveness:  $F_{(6,298)} = 9.595, p < .0005$ ; frequency:  $F_{(6,298)} = 12.585, p < .0005$ )

Post-hoc Scheffe tests were conducted to examine the differences between the Pagan group and other groups on the CAPS subscales. The results for both original and revised scoring methods are displayed in table 7.4. As with table 7.2, non-significant differences are denoted by NSD, significant differences are denoted by the presence of an effect size. The key denotes significance level.

Comparison groups	Pagan group CAPS subscale scores					
	Original scoring method			Revised scoring method		
	Distress	Intrus	Freq	Distress	Intrus	Freq
AAH	NSD	.85 <sup>a</sup>	.91 <sup>b</sup>	NSD	NSD	.81 <sup>a</sup>
Deluded, no Hal.	NSD	NSD	NSD	NSD	NSD	NSD
Public	NSD	NSD	.79 <sup>b</sup>	NSD	NSD	.73 <sup>a</sup>
Christian	NSD	NSD	.76 <sup>a</sup>	NSD	NSD	.69 <sup>a</sup>
Deluded, with Hal.	NSD	NSD	NSD	-1.04 <sup>b</sup>	NSD	NSD
Psychotic	-1.34 <sup>c</sup>	-.98 <sup>b</sup>	-.71 <sup>a</sup>	-1.51 <sup>c</sup>	-1.1 <sup>c</sup>	-.76 <sup>a</sup>

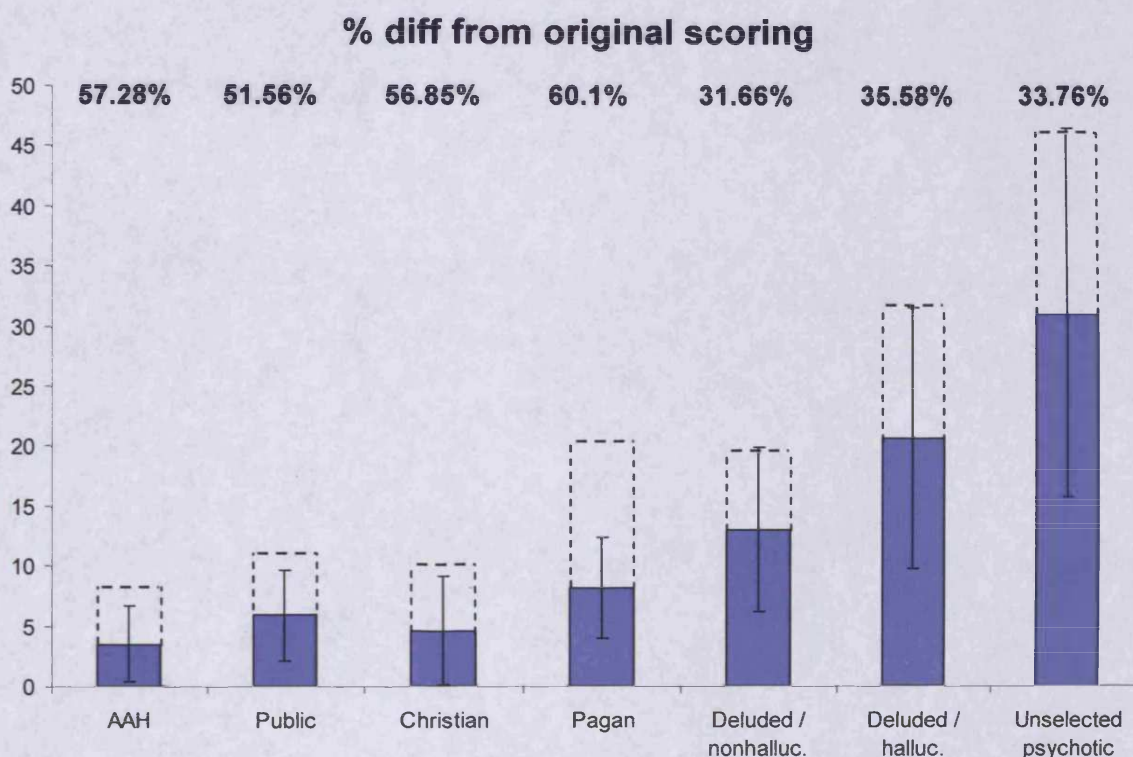
NSD = No significant difference; <sup>a</sup> = significant difference at least  $p < .05$ ; <sup>b</sup> = significant difference at least  $p < .005$ ; <sup>c</sup> significant difference at least  $p = < .0005$ . Negative effect sizes indicate Pagan group scored less than other group, a positive effect size indicates the reverse.

table 7.4 Effect sizes of significant post-hoc tests (Scheffe) between Pagans and other groups for both original and revised CAPS scoring method

As can be seen from table 7.4, the revised scoring method alters the pattern of significances for distress and intrusiveness, causing the Pagan group to appear more pathological when the



original scoring method is used. Conversely, with the revised scoring method, the Pagan group are no longer undifferentiated from the deluded patients with hallucinations on distress, and no longer report their experiences to be significantly more intrusive than the AAH group. Furthermore, the effect sizes typically become larger for significant differences when compared to the unselected psychotic group, but become smaller when the comparison involves non-clinical groups.



*figure 7.2 Difference between original and recoded CAPS distress score as percentages. Original distress scores shown as outlines.*

As can be seen from the percentage differences between original and revised scores given in figure 7.2, it is the explicitly religious groups (Pagan and Christian) and the explicitly non-religious group (AAH) who show the greatest difference in distress scores. The unselected psychotic group shows the least difference in distress scores. This is the same pattern as was

reported with regards to the PDI (section 7.5.1.1). Combined with the fact that Pagans become undifferentiated on distress scores to all non-clinical groups when the revised scoring method is used, this evidence suggests that the original CAPS scoring method significantly overestimates the distress caused by anomalous perceptual experience in Pagans.

Notably, the Pagan group is only differentiated on intrusiveness scores to the unselected psychotic group and reports significantly greater frequency of anomalous perceptual experience than the AAH, Public and Christian groups, but is undifferentiated on this measure to both the deluded patients with, and deluded patients without hallucinations. Notably, there are weak but significant correlations in the Pagan sample between STIN score and the CAPS total score (two-tailed;  $r = .29, p < .05$ ); and STIN score and the revised CAPS frequency score (two-tailed;  $r = .3, p < .05$ ). These correlations do not exist for the Christian group (the only other group to complete the STIN). This might suggest that even intrusive and frequent perceptual distortion is being appraised in positive spiritual terms by the Pagan group, rather than as a distressing experience, as by the psychotic and deluded patients with hallucinations groups.

### **7.5.3 Contribution of anomalous experience and distress to delusion formation.**

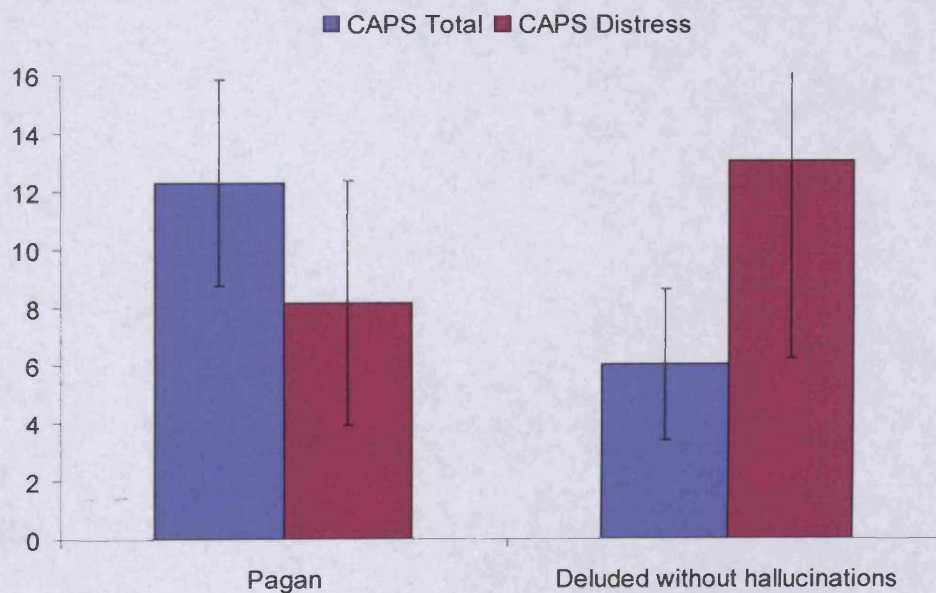
The results reported here suggest that Pagans show similar levels of anomalous perceptual experience to unselected psychotic and deluded patients with hallucinations, but report no more distress than other non-clinical groups.

In combination with the findings from section 5.5.3, that suggest that pathological levels of anomalous perceptual experience are not necessary for delusion formation, it might be argued that delusions arise from a necessary combination of distress and pathologically high levels of

perceptual distortion. In contrast, distress might itself be a central factor in delusion formation, independent of anomalous perceptual experience. This is in line with theories that argue for the central role of emotion in delusion formation (sections 3.2.3.2; 3.3; 3.4), and might provide some evidence for the validity of the ‘illness behaviour’ account of delusions put forward by Johns and van Os (2001), who argue that psychosis-like experience, in itself, is not sufficient to produce clinically significant experiences or behaviour.

As Pagans experience high levels of anomalous perceptual experience but low distress, it was predicted that the sample of deluded patients without hallucinations would display the reverse pattern, suggesting anomalous perceptual experience and distress might be independent, and that distress might be the more important factor in delusion formation.

Therefore, a planned comparison was performed to compare CAPS total score and CAPS distress score between the Pagan sample, and the sample of deluded patients without hallucinations.



*figure 7.3 CAPS total and distress scores in Pagan group and sample of deluded patients with hallucinations.*

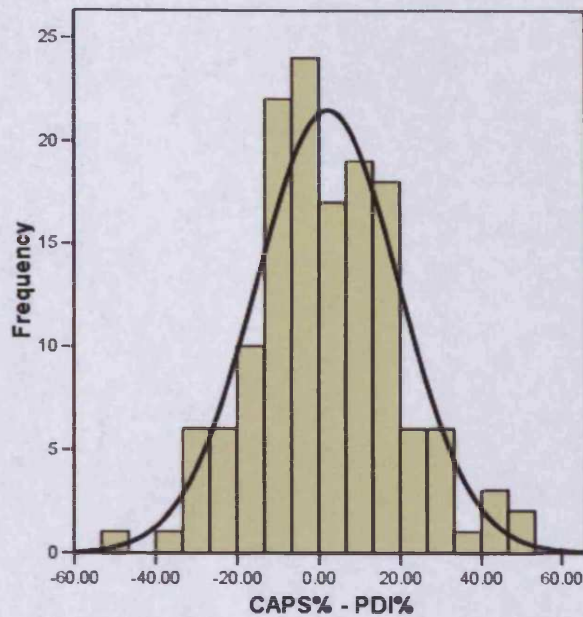
The means are graphed in figure 7.3 and display the hypothesised pattern. When compared using one-tailed independent samples t-tests, the Pagan group reported significantly higher levels of anomalous perceptual experience than the sample of deluded patients without hallucinations ( $t_{(164)} = 4.144, p < .0005$ ), and the sample of deluded patients without hallucinations reported significantly higher levels of distress than the Pagan group ( $t_{(164)} = -2.371, p < .001$ ). This suggests a double dissociation (of the trend type; Shallice, 1988, p227) between anomalous perceptual experience and distress. As can be seen from table 7.5, the pattern of associations and dissociations between these factors suggests that distress, rather than perceptual distortion is most association with the presence of delusions in the sampled participants.

<i>Group</i>	<i>Perceptual distortion</i>	<i>Distress</i>
Public	Low	Low
Pagan	High	Low
Deluded, no Hal.	Low	High
Deluded with Hal.	High	High

*table 7.5 Patterns of associations and dissociations between anomalous perceptual experience and distress*

#### **7.5.4 Relationship between anomalous perceptual experience and delusional ideation in the Pagan sample**

In an identical analysis to the one conducted in section 5.5.4, the CAPS total scores and PDI total scores were converted to a percentage, and the difference was calculated by taking the PDI percentage from the CAPS percentage. The distribution of scores for the Pagan sample is shown in figure 7.4



*figure 7.4 Frequency distribution of percentage difference between CAPS and PDI scores for Pagan sample (N = 142)*

The distribution has a skewness of .17 and a kurtosis of .231. When the skewness value is compared with the non-clinical sample (skewness = .043) and the unselected psychotic sample (skewness = -.157) from section 5.5.4, all can be seen to have a skewness value close to zero, showing a pattern similar to a normal distribution. In terms of kurtosis, the Pagan kurtosis value of .231 is part way between the peaked distribution of the non-clinical sample (kurtosis = .724) and the wider-based distribution of the unselected psychotic sample (kurtosis = -.062), although closer to the non-clinical sample. This suggests that the relationship between anomalous experience and delusional ideation varies throughout the Pagan sample, although the extreme discrepancies are not as prevalent as in the unselected psychotic sample, although a little more so than in the non-clinical sample.

### **7.5.5 CAPS item odds ratio analysis**

An odds ratio analysis was conducted (as described in 5.5.6) to identify those items that most distinguished the Pagan group from the combined unselected psychotic and deluded with

hallucinations groups. The results for individual items are displayed in table 7.6, and for

CAPS item groupings in table 7.7.

Item	Item Text	Non-clinical		Clinical		Odds ratio	95% CI	
		Freq N	Freq %	Freq N	Freq %		Low	High
28	Have you ever heard two or more unexplained voices talking with each other ?	17	11.97	23	52.27	8.05	3.70	17.55
7	Do you ever hear your own thoughts spoken aloud in your head, so that someone near might be able to hear them ?	27	19.01	24	54.55	5.11	2.47	10.57
11	Do you ever hear voices commenting on what you are thinking or doing ?	35	24.65	27	61.36	4.86	2.37	9.94
13	Do you ever hear voices saying words or sentences when there is no-one around that might account for it ?	48	33.80	26	59.09	2.83	1.41	5.66
2	Do you ever sense the presence of another being, despite being unable to see any evidence ?	59	41.55	28	63.64	2.46	1.22	4.95
3	Do you ever hear your own thoughts repeated or echoed ?	59	41.55	27	61.36	2.23	1.12	4.47
6	Do you ever hear noises or sounds when there is nothing about to explain them ?	67	47.18	29	65.91	2.16	1.07	4.38
16	Do you ever find that sounds are distorted in strange or unusual ways ?	28	19.72	15	34.09	2.11	1.00	4.45
30	Do you ever notice that food or drink seems to have an unusual taste ?	31	21.83	16	36.36	2.05	0.98	4.25
14	Do you ever experience unexplained tastes in your mouth ?	32	22.54	16	36.36	1.96	0.95	4.07
17	Do you ever have difficulty distinguishing one sensation from another ?	15	10.56	8	18.18	1.88	0.74	4.79
25	Do you ever find that common smells sometimes seem unusually different ?	22	15.49	10	22.73	1.60	0.69	3.71
22	Do you ever look in the mirror and think that your face seems different from usual ?	50	35.21	20	45.45	1.53	0.77	3.05
5	Do you ever experience unusual burning sensations or other strange feelings in or on your body ?	67	47.18	25	56.82	1.47	0.75	2.91
19	Do you ever find the appearance of things or people seems to change in a puzzling way, e.g. distorted shapes or sizes or colour ?	33	23.24	13	29.55	1.39	0.65	2.95
9	Do you ever have the sensation that your body, or a part of it, is changing or has changed shape ?	46	32.39	16	36.36	1.19	0.59	2.42
15	Do you ever find that sensations happen all at once and flood you with information ?	53	37.32	18	40.91	1.16	0.58	2.32
18	Do you ever smell everyday odours and think that they are unusually strong ?	47	33.10	16	36.36	1.16	0.57	2.34
26	Do you ever think that everyday things look abnormal to you ?	35	24.65	11	25.00	1.02	0.47	2.23
32	Do you ever hear sounds or music that people near you don't hear ?	71	50.00	22	50.00	1.00	0.51	1.97
21	Do you ever think that food or drink tastes much stronger than it normally would ?	39	27.46	12	27.27	0.99	0.46	2.12
1	Do you ever notice that sounds are much louder than they normally would be ?	59	41.55	17	38.64	0.89	0.44	1.77
4	Do you ever see shapes, lights or colours even though there is nothing really there ?	59	41.55	17	38.64	0.89	0.44	1.77
23	Do you ever have days where lights or colours seem brighter or more intense than usual ?	77	54.23	21	47.73	0.77	0.39	1.52
29	Do you ever notice smells or odours that people next to you seem unaware of ?	67	47.18	17	38.64	0.70	0.35	1.41
10	Do you ever have the sensation that your limbs might not be your own or might not be properly connected to your body?	46	32.39	11	25.00	0.70	0.32	1.50

table 7.6 [continued on next page] Comparison between Pagan group and combined unselected psychotic and deluded-with-hallucinations patients groups by odds ratio.

Item	Item Text	Non-clinical		Clinical		Odds ratio	95% CI	
		Freq N	Freq %	Freq N	Freq. %		Low	High
8	Do you ever detect smells which don't seem to come from your surroundings ?	74	52.11	18	40.91	0.64	0.63	2.22
31	Do you ever see things that other people cannot ?	87	61.27	22	50.00	0.63	0.50	2.57
12	Do you ever feel that someone is touching you, but when you look nobody is there ?	75	52.82	18	40.91	0.62	0.52	2.33
20	Do you ever find that your skin is more sensitive to touch, heat or cold than usual ?	80	56.34	18	40.91	0.54	0.56	1.99
27	Do you ever find that your experience of time changes dramatically ?	93	65.49	20	45.45	0.44	0.51	1.85

table 7.6 [continued from previous page] Comparison between Pagan group and combined unselected psychotic and deluded-with-hallucinations patients groups by odds ratio.

Table 7.7 also shows the data from the comparison between non-clinical and combined unselected psychotics and deluded with hallucinations sample, taken from table 5.7. The results from Pagan comparison show a remarkable similarity to the comparison between clinical and non-clinical participants from Chapter 5 (section 5.5.6). One notable difference stands out, however. Whereas the experience of having a non-shared sensory experience previously distinguished clinical from non-clinical groups (with the clinical group much more likely to experience a perceptual distortion as not shared by others), the Pagan group are undifferentiated from the clinical group on this dimension, suggesting they are equally as likely to experience an anomalous perceptual experience as one not shared by others. This may suggest that it is how this experience is appraised emotionally, rather than the experience of being the sole recipient of such an experience itself, which may mediate the likelihood of pathology.

Category	Non-clinical		Clinical		OR	95% CI	
	Freq N	Freq %	Freq N	Freq %		Low	High
By component from principal component analysis							
Component three 'clinical psychosis' (3,7,11,31)	52.00	15.48	25.00	56.82	2.28	1.15	4.53
Component one 'temporal lobe experience' (1,2,4,6,10,12,16,24,26,27,32)	60.09	17.88	18.00	40.91	0.94	0.47	1.88
Component two 'chemosensation' (8,14,18,20,21,25,29,30)	49.00	14.58	15.38	34.94	1.02	0.50	2.07
By sensory modality							
Audition (1,3,6,7,11,13,16,28,32)	45.67	13.59	23.33	53.03	2.38	1.20	4.74
Touch (5,9,10,12,20)	62.80	18.69	17.60	40.00	0.84	0.42	1.67
Vision (4,19,22,23,26,31)	56.83	16.91	17.33	39.39	0.97	0.49	1.95
Smell (8,18,25,29)	52.50	15.63	15.25	34.66	0.90	0.45	1.84
Taste (14,21,30)	34.00	10.12	14.67	33.33	1.59	0.76	3.31
By initial CAPS creation categories							
Verbal hallucinations (13,11,28)	33.33	9.92	25.33	57.58	4.42	2.17	9.02
Thought echo and hearing thoughts out loud (3,7)	43.00	12.80	25.50	57.95	4.49	2.20	9.17
Having a non-shared sensory experience (13,29,31,32)	68.25	20.31	21.75	49.43	1.06	0.54	2.08
Sensory experience from an unexplained source (4,6,8,12,14,28)	54.00	16.07	20.17	45.83	1.38	0.70	2.73
Distortion of form (size, shape) of own body and of external world (9,10,22,19)	43.75	13.02	15.00	34.09	1.16	0.57	2.38
Sensory flooding (15,17)	34.00	10.12	13.00	29.55	1.33	0.63	2.83
Inherently unusual or distorted (5,16,25,26,30)	36.60	10.89	15.40	35.00	1.55	0.75	3.20
Temporal Lobe (2,10,24,27)	66.75	19.87	17.25	39.20	0.73	0.36	1.45
Changes in levels of sensory intensity (1,18,20,21,23)	60.40	17.98	16.80	38.18	0.83	0.42	1.67

table 7.7 Odds ratios for mean frequency for CAPS items categories comparing between clinical and pagan populations. Contributory items listed in brackets.

The clinical group is much more likely to report 'first-rank' symptoms, although as this is the criteria for the diagnosis for many of the members of this group, this may be due to a sample bias.

### 7.5.6 Spiritual Transcendence and Christian Orthodoxy scales

The mean score for Christians on the STIN was 42.33 (SD = 5.18) compared to 39.93 (SD = 6.54) for the Pagan group. When compared using a two-tailed independent samples t-test,



there was no significant difference between the Christian and Pagan groups ( $t_{(89)} = 1.811, p = 0.074$ ). Although the  $p$  value suggests a trend toward significance, the small absolute difference between mean scores and the fact that they each reflects scores toward the high end of the scale's range suggest the Christian and Pagan groups can be considered equivalent in terms of the spiritual benefit they derive from their beliefs.

The Christian group had a mean score of 36.55 (SD = 9.53) on the COS, indicating a high degree of orthodoxy, being close to the maximum score of 42. This suggests the Christian group were representative of the mainstream of orthodox Christian beliefs.

### 7.5.7 Self-report of current and past mental / neurological illness

The frequencies of self-reported mental and neurological illness for each of the non-clinical groups are outlined in table 7.8. Four Pagan participants did not complete these questions. Chi-square tests suggest that the rate of self-report was not significantly different between groups for either current treatment of mental illness ( $\chi^2 = 5.766, p = .124$ ), past treatment of mental illness ( $\chi^2 = 2.864, p = .413$ ), current treatment of neurological illness ( $\chi^2 = 1.418, p = .701$ ) or past treatment of neurological illness ( $\chi^2 = 2.611, p = .456$ ). Although this assessment relied on self-report, it does provide some evidence that the presence of existing or past mental or neurological illness was not a confounding factor.

	<i>Christian</i> ( <i>N=32</i> )	<i>Pagan</i> ( <i>N=138</i> )	<i>AAH</i> ( <i>N=26</i> )	<i>Public</i> ( <i>N=36</i> )
Current mental illness	2	3	3	1
Past mental illness	5	18	5	2
Current neurological illness	0	3	0	1
Past neurological illness	0	5	2	1

*table 7.8 Frequencies of self reported mental and neurological illness*

## **7.6 Discussion**

This study compared the original and a revised scoring method for the PDI-21 and CAPS, and provides evidence that the original scoring method over-estimates the negative impact of psychosis-like experience in non-mainstream religious groups. The analysis, therefore, used a revised scoring method and found that Pagans have similar levels of anomalous perceptual experience to psychotic inpatients and deluded patients with hallucinations, but suffer no more perceptual distortion-related distress than the general population. A comparison between the Pagan sample and deluded patients without hallucinations indicated a double-dissociation between anomalous perceptual experience and distress, suggesting that these factors can be experienced relatively independently, and that distress rather than perceptual distortion may be the more important factor in delusion formation. An analysis of the difference between levels of anomalous perceptual experience and delusional ideation suggested a normal distribution, as has been found previously with both non-clinical and psychotic samples, but with the amount of distribution extremes part-way between the clinical and non-clinical groups. A frequency analysis comparing Pagan and clinical participants suggested that, unlike with the clinical / non-clinical comparison reported in Chapter 5 (section 5.5.6), Pagans experience perceptual distortions as not shared by others, in a more similar way to psychotic patients than the general population, suggesting this in itself is not necessarily a sign of pathology.

In contrast to Peters et al. (1999b), the Pagan group from this study reported significantly less delusional ideation than the clinical sample and were undifferentiated from a Christian control group. The unselected psychotic sample used in this study and the deluded inpatients sample from Peters et al. (1999) scored almost identically (both had a mean PDI-21 of 11.8). The

Pagan sample from this study scored lower (7.59) than the Peters' NRM sample (11.5), however, although there are some notable differences in sampling which may account for the discrepancies. Peters et al. recruited their participants from a Hare Krishna temple and an active Druid group, each of which may have contained members who might have consistently associated themselves more strongly with the religious ideas and spiritual experiences of the group, than the likely more diverse sample recruited by this study. Certainly, the Hare Krishna participants lived and worked at the temple, and so have made a considerable commitment to their spiritual practice.

Because both of these studies are cross-sectional, however, it is difficult to infer any causality. It may be that those who experience greater levels of anomalous experience and ideation are more likely to be attracted to, and make substantial commitments to, non-mainstream spiritual practices. The (admittedly weak) correlation between the total score and frequency subscale of the CAPS and the total score on the Spiritual Transcendence Index perhaps hints that this may be the case. As many spiritual practices result in altered states of consciousness (Vaitl et al., 2005), it may also be that such groups promote the likelihood of experiencing perceptual distortion and magical thinking.

The fact that the Pagan group reported similar CAPS total scores to two of the psychotic patient samples, but were no more distressed than the general population, and that a double dissociation can be seen between perceptual distortion and distress when compared to non-hallucinating deluded patients, suggests that anomalous perceptual experience is not necessarily associated with either distress or delusion formation.

Indeed, there is now increasing evidence that appraisal processes may be a key mediating factor in this regard. In an investigation of voice hearers, Birchwood and Chadwick (1997) noted that appraisal rather than the content of the hallucinated voices mediated distress. For example, voices that said seemingly innocuous comments could be experienced as very distressing, whereas some hearers regard their voices as benign, even when the content was hostile or derogatory. Close and Garety (1998), also working with voice hearers, reported negative reactions to voices were most associated with negative beliefs about the self. An appraisal about whether an unusual experience is a sign of 'madness' or mental illness has been cited as key in the delusion formation process (Birchwood, 1995) and plays a central role in the Institute of Psychiatry delusion formation model (section 3.4.1).

In contrast, certain appraisals of anomalous experiences have been found which might promote positive affect. Gauntlett-Gilbert and Kuipers (2005) reported that for patients who experience 'visions', positive affect was most associated with experiences appraised as a sign of being blessed or gifted.

A philosophy which accepts that, for example, magical or supernatural phenomena are a reality and that those who experience them are gifted, may be particularly comforting for someone who does have anomalous experiences but is unsure about how to interpret them. This framework might suggest to the person that they are not 'mad' (as such phenomena are thought genuinely to exist) and the fact that they personally experience the phenomena might indicate to the person that they are special in some way. It is, perhaps, unsurprising then, that people with anomalous experiences might be attracted to groups that interpret their experiences in ways that could simultaneously reduce negative affect and increase perceived self-worth, rather than potentially pathologise their experience as signs of mental instability.

Indeed, there is an increasing amount of convergent evidence that negative affect, rather than anomalous experience per se, increases the risk for developing psychosis, of which some of the most persuasive evidence comes from longitudinal studies. One study by Hanssen et al. (2005), found that participants experiencing distress in combination with an hallucinatory episode at the first assessment, had a fourfold chance of presenting with a delusion by the second. Further longitudinal studies have suggested that depressed mood and delusional ideation are risk factors for psychosis if they accompany an initial hallucinatory episode (Krabbendam and van Os, 2005; Krabbendam et al., 2005). In particular, Krabbendam and van Os (2005) found that neuroticism and worry were also risk factors, perhaps suggesting that long-term appraisal style is a significant influence.

What is not clear, however, is whether specific forms of experience may be particularly associated with psychosis, as the traditional diagnostic model maintains. Despite the Pagan group scoring similarly to the psychotic inpatients on total CAPS score, it is notable that there are differences in the prevalence of specific experiences, as can be seen from the odds ratio analysis outlined in tables 7.4 and 7.5. One problem with this analysis, is a potential sample bias from using a clinical sample which have largely been diagnosed on the basis of the presence of certain anomalous experiences ('first-rank' symptoms in particular).

Conceivably, the Pagan community might attract people with certain sorts of experiences, and this may also bias the sample to show a low prevalence of first-rank symptoms in a group with otherwise high levels of perceptual distortion. There are, however, examples of other non-clinical high-perceptual-distortion groups who may show far less of a difference in the prevalence of 'first-rank' symptoms. For example, the non-clinical voice hearers described by

Honig et al. (1998) reported voices of an identical form to the clinical group, but they did not feel alarmed or upset by their voices, and felt in control of the experience. Perhaps even some participants in the online 'mind control' community (identified in section 4.3) could also be candidates in this regard. Further studies, identifying other sub-groups of the population who might provide counter-evidence to the diagnostic model, therefore, are essential, if the ingredients of pathology, rather than the correlates of diagnosis, are to be discovered. Although this study has identified one sub-group of interest, it must be kept in mind that there are likely to be many others who may have differences in the qualitative colouring of their experiences, despite reporting similarly high levels of total perceptual distortion.

What this study does suggest, however, is that anomalous experience is not a necessary component of delusion formation, and neither is it necessarily linked to distress. This further suggests that appraisal is a key process in delusion formation and that non-mainstream spiritual beliefs may promote the adaptive incorporation of unusual experiences into a person's belief set.

## **7.7 Chapter summary and conclusion**

A comparison of different scoring methods of the PDI and CAPS showed that the original scoring methods over-estimate the extent of pathology in those with benign anomalous experiences and beliefs. A comparison between the Pagan sample and deluded patients without hallucinations indicates a trend double-dissociation between anomalous perceptual experience and distress, suggesting that these factors can be experienced relatively independently, and that distress rather than perceptual distortion may be the more important factor in delusion formation.

## 7.8 References

- Andreasen, NC. (1994) Scale for the assessment of positive symptoms (SAPS). Iowa City, University of Iowa.
- Bebbington PE, Nayani T. (1995) The psychosis screening questionnaire. *International Journal of Methods in Psychiatric Research*, 5, 11-19.
- Bell, V., Halligan, P.W., Ellis, H. (2003) Beliefs about delusions. *The Psychologist*, 16, 418-423.
- Bell, V., Halligan, P.W., Ellis, H.D. (2005) The Cardiff Anomalous Perceptions Scale (CAPS) and the Role of Anomalous Perceptual Experience in Delusion Formation. *Schizophrenia Bulletin* [Oct 19; Epub ahead of print]
- Berger, H.A., Leach, E.A., Shaffer, L.S. (2003) *Voices from the Pagan Census: A National Survey of Witches and Neo-Pagans in the United States*. Columbia: University of South Carolina Press.
- Birchwood, M. (1995) Early intervention in psychotic relapse: Cognitive approaches to detection and management. In G. Haddock and P. Slade(eds) *Cognitive Behavioural Interventions with Psychotic Disorders*. London: Routledge.
- Birchwood, M., Chadwick, P. (1997) The omnipotence of voices: Testing the validity of a cognitive model. *Psychological Medicine*, 27, 1345–1353.

Brett, C. (2003) Psychotic and mystical states of being: Connections and distinctions.

*Philosophy, Psychiatry and Psychology*, 9, 321-341.

Clarke, I. (2001) *Psychosis and Spirituality*. London: Whurr Publishers Ltd.

Close H, Garety P. (1998) Cognitive assessment of voices: further developments in understanding the emotional impact of voices. *British Journal of Clinical Psychology*, 37, 173-88.

Day, S., & Peters, E. (1999) The incidence of schizotypy in new religious movements.

*Personality and Individual Differences*, 27, 55-67.

Gauntlett-Gilbert, J., & Kuipers, E. (2005) Visual hallucinations in psychiatric conditions:

Appraisals and their relationship to distress. *British Journal of Clinical Psychology*, 44, 77-87.

Geschwind, N. (1983) Interictal behavioral changes in epilepsy. *Epilepsia*, 24 Suppl 1, S23-

30.

Hanssen, M., Krabbendam, L., de Graaf, R., Vollebergh, W., & van Os, J. (2005) Role of

distress in delusion formation. *British Journal of Psychiatry Supplement*, 48, s55-58.

Harvey, G., Hardman, C. (1996) *Pagan Pathways: A Guide to Ancient Earth Traditions*.

London: Thorsons.



Honig A, Romme MA, Ensink BJ, Escher SD, Pennings MH, deVries MW. (1998) Auditory hallucinations: a comparison between patients and nonpatients. *Journal of Nervous and Mental Disease*, 186, 646-51.

Hunsberger, B. (1989) A short version of the christian orthodoxy scale. *Journal for the Scientific Study of Religion*, 28, 360-365.

Hutton, R. (1002) *The Triumph of the Moon: A History of Modern Pagan Witchcraft*. Oxford: Oxford University Press.

Jackson, M., & Fulford, K. W. M. (1997) Spiritual experience and psychopathology. *Philosophy, Psychiatry and Psychology*, 4, 41-65.

Johns, L. C., & van Os, J. (2001) The continuity of psychotic experiences in the general population. *Clinical Psychology Review*, 21, 1125-1141.

Krabbendam, L., Myin-Germeys, I., Hanssen, M., de Graaf, R., Vollebergh, W., Bak, M., & van Os, J. (2005) Development of depressed mood predicts onset of psychotic disorder in individuals who report hallucinatory experiences. *British Journal of Clinical Psychology*, 44, 113-125.

Krabbendam, L., & van Os, J. (2005) Affective processes in the onset and persistence of psychosis. *European Archives of Psychiatry and Clinical Neuroscience*, 255, 185-189.

Makarec, K., & Persinger, M. A. (1985) Temporal lobe signs: electroencephalographic validity and enhanced scores in special populations. *Percept Mot Skills*, 60, 831-842.

Marzanski, M., Bratton, M. (2003) Psychopathological symptoms and religious experience: A critique of Jackson and Fulford. *Philosophy, Psychiatry and Psychology*, 4, 359-371.

Ohayon, M. M. (2000) Prevalence of hallucinations and their pathological associations in the general population. *Psychiatry Research*, 97, 153-164.

Persinger, M. A. (1983) Religious and mystical experiences as artifacts of temporal lobe function: a general hypothesis. *Percept Mot Skills*, 57, 1255-1262.

Peters, E., Day, S., McKenna, J., & Orbach, G. (1999b) Delusional ideation in religious and psychotic populations. *British Journal of Clinical Psychology*, 38, 83-96.

Peters, E., Joseph, S., Day, S., & Garety, P. (2004) Measuring delusional ideation: the 21-item Peters et al. Delusions Inventory (PDI). *Schizophrenia Bulletin*, 30, 1005-1022.

Peters, E. R., Joseph, S. A., Garety, P. A. (1999a) Measurement of delusional ideation in the normal population: introducing the PDI (Peters et al. Delusions Inventory). *Schizophrenia Bull*, 25, 553-576.

Pierre, J.-M. (2001) Faith or delusion: At the crossroads of religion and psychosis. *Journal of Psychiatric Practice*, 7, 163-172.

Seidlitz, L., Abernethy, A. D., Duberstein, P. R., Evinger, J. S., Chang, H. T., Lewis, B.  
(2002) Development of the Spiritual Transcendence Index. *Journal for the Scientific Study of Religion*, 41, 439-453.

Shallice, T. (1988) *From Neuropsychology to Mental Structure*. Cambridge: Cambridge University Press.

Sims A. (2003) *Symptoms in the mind: An introduction to descriptive psychopathology (3rd edition)*. Edinburgh: Elsevier Science Ltd.

Skirda, R. J., & Persinger, M. A. (1993) Positive associations among dichotic listening errors, complex partial epileptic-like signs, and paranormal beliefs. *J Nerv Ment Dis*, 181, 663-667.

Tien, A.Y. (1991) Distributions of hallucinations in the population. *Social Psychiatry and Psychiatric Epidemiology*, 26, 287-292.

Vaitl, D., Birbaumer, N., Gruzelier, J., Jamieson, G. A., Kotchoubey, B., Kubler, A.,  
Lehmann, D., Miltner, W. H. R., Ott, U., Putz, P., Sammer, G., Strauch, I., Strehl, U.,  
Wackermann, J., & Weiss, T. (2005) Psychobiology of altered states of consciousness.  
*Psychological Bulletin*, 131, 98-127.

## Chapter Eight

### Integration and Future Directions

Elements of this chapter are in publication as:

Bell, V., Halligan, P.W. & Ellis, H.D (in press) Explaining delusions: A cognitive perspective. *Trends in Cognitive Sciences*

## **8.1 Chapter outline**

Sections:

- 8.2 Summary of main findings
- 8.3 Implications for theories of delusions
- 8.4 Implications for theories of normal belief
- 8.5 Chapter conclusion
- 8.6 Concluding remarks
- 8.7 References

This chapter first summarises the findings and conclusions from previous chapters before examining the implications for theories of delusions and normal belief. Shortcomings and limitations of the evidence, in light of these theories, are also discussed, as are future research avenues for the development of this area.

## **8.2 Summary of main findings**

### **8.2.1 Chapter 1: Approaches to the Neuropsychology of Normal Belief**

There is no accepted definition of belief, and it is doubtful whether the concept can be identified with a unitary neuropsychological process. Therefore, what we call belief is likely to be supported by a number of potentially disparate neuropsychological processes and is not likely to be described in a monolithic model. Past experience of both neuropsychological studies on belief, and research on what seemed like similarly-nebulous constructs at the time, suggests that it is possible to successfully apply the techniques of cognitive neuropsychology to understanding belief, although the importance of converging evidence from phenomenology, single cases, neuropsychological correlates of belief states, and cognitive neuroscience is stressed. There are some attributes that have been consistently associated with

belief and will need to be explained in any comprehensive model: namely an explanation of both conscious and unconscious processes in belief formation, the influence of context and the 'web of belief', the role of affect in belief formation and confidence and authority in belief formation.

### **8.2.2 Chapter 2: Delusions: A Conceptual Review**

Although the diagnosis of a delusion can be made with acceptable reliability, it is clear that the current diagnostic criteria are not coherent and further work needs to be completed to test the boundaries of the concept. Furthermore, it is not clear that the traditional form / content dichotomy used in descriptive psychopathology is valid as a basis for scientific research.

There is some doubt in the literature as to whether delusions are beliefs as we would normally conceive of them and there is significant doubt about the status of delusions as a unitary phenomenon (in parallel with similar doubts about normal belief). Therefore, neuropsychological models must either be limited in their scope (not seeking to explain all delusions), or accept that phenomena currently labelled as 'delusional' may need to be explained by a number of processes, or be agnostic as to whether delusions are beliefs and derive correlates of the general phenomena.

### **8.2.3 Chapter 3: Delusions: Current Models and Empirical Evidence**

Functional models from cognitive, neuropsychological, neurobiological and computational traditions were summarised and discussed in terms of their explanatory power as general models of delusion formation. Several themes emerged from this analysis: despite the central role of anomalous perceptual experience in many delusion formation models, this aspect has been poorly specified and it is unclear whether it is a necessary condition; The functional neuroanatomy of delusional states is mostly understood at the level of very large brain

structures and needs refining. This understanding is also largely based on correlational studies, suggesting the need for research to allow causal processes to be inferred. Several models do not tackle the issue of why a belief might be pathological in pragmatic terms. Affect and emotion are likely to be key contributors to this process.

#### **8.2.4 Chapter 4: The Diagnostic and Phenomenological Boundaries of Delusions**

This chapter reported on two empirical studies that examined the influence of the internet on delusional beliefs to investigate the stability of the traditional psychopathological boundaries of delusion under the influence of a recent sociocultural development. The first study used a social network analysis approach to examine links between likely-delusional people who are part of the online ‘mind control’ community. The results suggest that there is an online community based around the content of likely-delusional beliefs, which is contrary to the diagnostic criterion that a delusion should not be a belief “accepted by other members of the person’s culture or subculture”. A further study conducted a literature review and presented a case series of people with internet-themed delusions, and concluded that the internet as a sociocultural phenomena could influence content, form, aetiology and prognosis of a delusional belief. The fact that the existing diagnostic criteria are challenged by a recent social development and clinical intervention, and that seemingly similar delusions could be markedly different in significant clinical features, provides further evidence that delusions are not a single unitary phenomena. Therefore, simply comparing clinical with non-clinical cases (as is common in cognitive neuropsychology) is not, in itself, likely to be adequate, if used as the sole approach to researching this area.

### **8.2.5 Chapter 5: The Cardiff Anomalous Perceptions Scale (CAPS) and the Role of Anomalous Perceptual Experience in Delusion Formation**

This chapter was inspired by the need to better specify and explore the role of perceptual distortion in delusion formation. It was noted that existing scales were often based on assumptions from clinical psychiatry that could either bias, or fail to take into account, certain aspects of anomalous experience when attempting to quantify the full range of relevant experiences. The chapter described the development of the CAPS, a valid, reliable scale for measuring anomalous perceptual experience, and its application to both clinical and non-clinical participants. A principal components analysis of data from the non-clinical population suggested a three component solution (the components were tentatively labelled 'chemosensation', 'temporal lobe experience' and 'clinical psychosis'). Comparisons with clinical participants suggested that pathologically high levels of anomalous perceptual experience are not a necessary condition for delusion formation, providing evidence against one-stage and two-stage models.

### **8.2.6 Chapter 6: The Role of the Lateral Temporal Lobes in Anomalous Perceptual Experience and Magical Thinking: A Transcranial Magnetic Stimulation Study**

One of the conclusions from Chapter 5 is that temporal lobe disturbance might be a contributory factor to anomalous perceptual experience. Furthermore, it was stressed in Chapter 3 that the functional neuroanatomy of delusional states needs refining and moving beyond purely correlational accounts. Transcranial magnetic stimulation (TMS) is capable of targeting relatively small areas of cortex and allowing causal inferences to be made. This study used TMS to disrupt the function of the lateral temporal lobes during an experiment where healthy participants were asked to detect non-existent patterns in visual noise.

Inhibition of the left lateral temporal area produced a significantly reduced tendency to report



meaningful information, suggesting that left lateral temporal activation may be more important in ‘magical thinking’ and, therefore, in producing and supporting anomalous beliefs and experiences. This provides evidence that refines the functional neuroanatomy of delusions, and provides some additional validation for the role of the temporal cortices in anomalous experience and magical thinking.

### **8.2.7 Chapter 7: Delusions, Anomalous Experience and Distress**

Many of the neuropsychological approaches to delusions have had little to say on what makes a delusion pathological, as opposed to lacking in consensual evidence, or being fixed or fantastical – all of which have been shown not to differentiate delusions from normal belief. The study reported in this chapter compared results from the Peters et al. Delusions Inventory (PDI) and CAPS from the following groups: atheists, agnostics and humanists, Christians, Pagans, an unselected public sample and groups of psychotic patients. Pagans are of particular interest, as they are known to have anomalous beliefs and experiences but are very functional, suggesting that they may be counter-examples to the traditional criteria upon which the diagnosis of delusion is based. A comparison of different scoring methods of the PDI and CAPS show that the original scoring method over-estimates the extent of pathology in those with benign anomalous experiences and beliefs. A comparison between the Pagan sample and deluded patients without hallucinations indicates a trend double-dissociation between anomalous perceptual experience and distress, suggesting that these factors can be experienced relatively independently, and that distress rather than perceptual distortion may be the more important factor in the formation of certain delusions.

## **8.3 Implications for theories of delusions**

### **8.3.1 Conceptual basis of delusions**

The traditional view of delusion as being a well-defined psychopathological symptom has been considerably weakened since the early 1990s (section 2.4). More recently, however, researchers are beginning to question whether delusions are really a unitary phenomenon, and, therefore, an indivisible symptom (Gilleen and David, 2005).

Critics of this position might suggest that the fact that the validity of the diagnostic construct is doubtful, does not necessarily mean that there is no circumscribed pathology to detect and define, but simply that it has not yet been fully understood. More troubling for the unitary view of delusions, however, would be evidence suggesting that the status of the underlying normal function was in doubt.

As can be seen from the analysis in Chapter 1, the status of belief as a unitary neuropsychological process is indeed doubtful. This suggests that any nosology based on such a concept must also be considered as heterogeneously represented and likely to correspond to nothing more than an umbrella term for various pathologies that result in either unlikely belief-claims, or the tendency for others to attribute unlikely beliefs to a person (by the person acting unusually, perhaps).

The status of the delusion concept as an 'umbrella term' is further supported by evidence reported in Chapter 4, which suggests that the traditional clinical boundaries of delusions are not stable under the influence of new social and technological developments. Therefore, theories of delusion (or indeed, belief) that argue for any specifically dedicated neuropsychological process are likely to be misleading.

### **8.3.2 The role of anomalous perceptual experience in delusion formation**

The results presented in Chapter 5 and 7 strongly suggest that pathological levels of anomalous perceptual experience are not necessary for delusion formation (most succinctly summarised in table 5.8). The strongest conclusion from this evidence would be that the one-stage / two-stage debate is dead, and that delusions are better understood as arising from an imbalance in the competing demands of two or more factors, rather than sequential stages.

This was first cogently proposed by Stone and Young (1997) and has now been incorporated into several of the more recent explicitly neuropsychological models of delusion formation (section 3.2.3). Notably, this view is also compatible with several other models, although they do not necessarily frame themselves in the same tradition or terminology.

For example, anomalous perceptual experience plays a barely-detectable role in either the ‘early’ Bentall model (section 3.3.2.1) or the revised ‘Attribution / Self-Representation’ version (3.3.2.2). Nevertheless, an hallucination (for example) could be considered as an event to which an attribution is made, potentially leading to a delusional interpretation, although there is no explicit need for perceptual distortion in this account, suggesting that delusions could arise without such occurrences. Nevertheless, this is only one interpretation of the model, and champions of the role of perceptual distortion might be tempted to reply that the model does not specifically exclude anomalous experience either.

The Institute of Psychiatry (IoP) model, however, does explicitly include anomalous experience as a component in its delusion formation and maintenance models (sections 3.4.1; 3.4.2), although it sees this as only one contributory factor, along with emotion, cognitive

biases and pre-existing beliefs, that contribute towards delusion formation. It is not entirely clear, however, whether the IoP model considers perceptual distortion as a necessary component. Certainly, it is depicted as the only route via which other factors can influence the 'search for meaning' in their diagram of the formation model (section 3.4.1). Freeman and Garety (2004, p117) seem to suggest, however, that the key experience is 'inner-outer confusion', which is described almost entirely in perceptual terms, although the also-included experience of abnormal "significance or reference" could be considered as post-perceptual or metacognitive in nature.

The critical question in this instance is 'does the IoP model allow for a delusion in the absence of pathological levels of anomalous perceptual experience?' On first sight, it seems this is indeed the case, as the inclusion of a 'direct' role for emotion suggests that abnormal affect or arousal could replace perceptual distortion in assuming the role of 'anomalous experience'. The established link between (particularly depression) and anomalous experience (Ohayon and Schatzberg, 2002) suggests, however, that if affect is a crucial aspect in the development of delusions, some perceptual distortion may occur in parallel and be interpreted in a delusional manner. Whether this necessarily occurs at a greater level than in other, non-delusional people, is doubtful though, suggesting that the IoP model could allow for delusions in the absence of significant pathologies of perceptual experience.

Notably, several of the metacognitive models (section 3.5) are either so specific as to only address certain delusions which are argued to arise from specific anomalous experiences (such as Frith and Blakemore motor-control model; sections 3.5.1.2; 3.5.2) or ignore the role of anomalous perceptual experience completely and argue that delusions arise from 'theory of mind' impairments (section 3.5.1.1). The 'theory of mind' approach, therefore, is compatible

with evidence suggesting pathological perceptual distortion is not necessary, but is significantly hampered by the equivocal supporting evidence.

Other, more general, metacognitive models (section 3.5.3) are more promising, in that they suggest a 'pure' cognitive dysfunction account could explain the presence of a delusion, and interestingly, that anomalous perceptions are more likely to be misidentified because of their distorted perceptual nature (Johnson and Raye, 2000). This would account both for the correlation between perceptual distortion and delusion, and the fact that it is unlikely to be a necessary condition. This is a promising area, although notably still limited in its scope and with little experimental evidence to add weight to the main proposals.

Notably, the approach taken by Johnson highlights the fact that it might be possible for a delusion to form from a pathologically high level of perceptual distortion, but *without* pathologically high levels of cognitive bias. Simply accepting an hallucinatory experience as veridical is enough (Sackeim, 1998), and indeed, this route is featured in the Davies et al. model (Davies et al. 2001; section 3.2.3.1). It is worth highlighting here that cognitive biases are the norm, rather than the exception, even in the general population (Gilovich, 1993), so no form of belief formation (either normal or pathological) is likely to occur in the complete absence of bias.

The hemispheric asymmetry models (section 3.6; with the exception of the McKay model, if it is included under this heading; section 3.2.3.2), the neurobiological (section 3.7) and computational models (3.8) typically do not make a distinction between explaining delusions and perceptual distortions; this could be used either to support or undermine the necessity of anomalous perceptual experience. These models undoubtedly provide advances for the project

of applying computational models to psychopathological problems, but in most cases they are not complete, well-supported, or in some cases, well-tested, and could barely be considered as comprehensive theories.

If it is assumed that perceptual distortion is not a necessary condition for delusion formation, although it is accepted that it may contribute towards it, then it is appraisal mechanisms that must be seen as central in producing delusions.

What is left unclear by the research covered in this thesis, however, is whether there are specific anomalous experiences that are more likely to be appraised in a delusional way and / or as distressing. Indeed, some authors have suggested that cognitive disorganisation may contribute to the formation of certain forms of anomalous experience that are most linked to pathological states (Serper et al., 2005). Unfortunately, it is not possible to test this hypothesis from the data presented here, partly because of the cross-sectional nature of the relevant studies, and partly because there is no objective measure of belief pathology.

This last point is important, as the diagnostic criteria used to select the clinical groups in Chapters 5 and 7, are being criticised by the study that uses them. Therefore, these groupings can only be considered as approximate ways of delineating pathology. As noted in section 7.6, there are likely to be further groups in the general population who might have experiences that are traditionally 'psychotic', but pragmatically benign. Different groups may have different distributions of experiences which still make them comparable to the a clinical group, so, for example, Pagans may be more likely to experience visual phenomena, whereas non-clinical members of the 'mind control' community might be perhaps more likely to experience passivity-like experiences. A better knowledge of the extent and diversity of these

groups, is essential to teasing out whether any particular experience or belief is specifically associated with pathology.

If through such comparisons, a particular experience is found only to occur in pragmatically clinical groups, it might be reasonable to try and frame the mechanism of this experience as cognitive pathology. If, however, an experience can occur benignly, a pathological account should focus on mechanisms that cause some people (perhaps, even the majority in some instances) to appraise it as distressing or disabling.

This would then raise the question: ‘What contributes to making these appraisals pathological?’. Further evidence from this thesis suggests that temporal lobe disturbance and the impact of negative affect may both make significant contributions.

### **8.3.3 The role of temporal lobe disturbance in magical thinking**

What is most striking when reading the psychosis continuum and temporal lobe literature is that both traditions have developed independently. This is particularly surprising as they have arrived at remarkably similar conclusions, particularly with regard to proposing a continuum of anomalous experiences and beliefs.

This thesis contributes to the understanding of these traditions and how they relate to delusion formation in two ways: firstly, by theoretically tying the two traditions together and noting their interconnections and secondly, by testing experimentally whether temporal lobe disturbance is related to forms of anomalous experience traditionally associated with the psychosis continuum and examining whether it plays a causal role in related cognitive processes. This is particularly important as it is not clear whether the two literatures are

discussing the same experiential phenomena from different angles, or whether temporal lobe disturbance makes any independent contribution to the psychosis continuum.

The principal components analysis of the CAPS (section 5.5.5) suggests that phenomena related to temporal lobe disturbance tend to occur together. This is a purely correlational result, however, and the hypothesis that these experiences are indeed related specifically to functional changes in the temporal cortex needs validating if it is to be taken seriously. The brief regression analysis presented in Appendix II provides preliminary data that these items predict the score on the well-validated Temporal Lobe Scale (Makarec and Persinger, 1985). Perhaps, more convincing in this regard is the TMS study reported in Chapter 6. This indicated that inhibiting the left lateral temporal lobe can alter the perception of non-existent meaningful information, suggesting that the temporal lobes play a causative role in producing anomalous experience.

This is particularly pertinent in light of the previous research which has examined the functional neuroanatomy of delusional states (section 3.7.1), specifically implicating the temporal and frontal lobes. What has not been clear from these studies is whether the differences in pathophysiology and function seen in these areas are the primary causes of delusions, or are simply a reflection of, for example, down-stream cognitive effects or compensation owing to pathology in other (perhaps more disparate) areas. The TMS paradigm deployed in Chapter 6 suggest that the temporal lobes may indeed play a causal role.

The nature of this role in the wider condition of psychosis is still far from clear, however. The interactions between the frontal and temporal cortices is thought to be key, although the cognitive aspects of this interaction have not been well described. Based on general findings



from normal cognitive neuroscience, it is possible to speculate that differences in the function of the frontal cortex may reflect attentional effects (Davidson and Heinrichs, 2003), whereas alterations in temporal cortex function would be associated with magical thinking and apophenia (section 6.3).

Indeed, in a review on the pathophysiology of schizophrenia, Siever and Davis (2004) suggested that there could be a compensatory relationship, in that the frontal cortex could compensate for temporal pathology. This view is not new, however, as Weinberger (1991) has previously suggested that schizophrenia may involve hypofrontality as a secondary effect from aberrant afferent temporal lobe connections, a model upon which Ruppin et al. (1996; section 3.8.3) based their connectionist model of delusion formation.

This suggests that explanations that localise functions (or indeed, dysfunctions) to individual brain areas, rather than functional networks, are likely to be unsustainable in the long-term. Notably, TMS may well be one of the more useful technologies in this regard, as it has near-millisecond resolution, potentially enabling network theories to be tested (with paired-pulse paradigms, for example), enabling the work presented in this thesis to be extended. Recent developments, such as the pairing of TMS with neuroimaging technologies, may even allow theories that propose a frontal-temporal interaction in psychosis to be tested with magical-thinking-like paradigm used in Chapter 6, allowing causal inferences to be drawn from combined behavioural and neurophysiological measurements.

One of the remaining caveats is that although the basic neurophysiology of TMS is now quite well specified (Walsh and Pascual-Leone, 2003), the wider cognitive effects and the effects on neural networks (as opposed to small areas directly under the coil's peak influence) are not

so fully understood. This makes it difficult to predict how the effect reported in Chapter 6 may differ, given different pulse timings and strengths. With some studies now using sub-threshold TMS to increase activation in both short-term (sub-second) and long-term (sub-hour) ranges, it would be also interesting to extend this research by aiming to excite, rather than inhibit, the lateral temporal areas, and look for the opposite pattern of results to provide some additional supporting evidence.

A further question concerns the level of resolution needed for an adequate description of the neural basis of psychosis and psychosis-like experience. Although this study has provided evidence that relatively small areas of the temporal cortex may be involved in magical thinking (compared to previous studies that have typically implicated whole lobes), evidence from a volumetric study by Sumich et al. (2005) found opposite correlations for adjacent areas of the left temporal lobe (namely Heschl's gyrus and the planum temporal) with reality-distortion symptoms. Although Sumich's study has yet to be replicated, it is still likely that the complexity of the temporal lobes support a multitude of processes, suggesting that a detailed understanding will be needed before an integrated cognitive neuroscience of delusions is possible.

In light of the conclusions from Chapters 1 and 2 (see also section 8.3.1), that belief and delusions are not likely to be explained by a unitary neuropsychological mechanism, it is important to bear in mind that many of the studies on 'delusional' patients may only be measuring shared variance from heterogeneous (but, it is hoped, overlapping) clinical phenomena. One way forward is to use interventionist paradigms on healthy participants, as deployed in the TMS study described in Chapter 6. Another approach is to sub-classify delusions, as suggested by Freeman and Garety (2004).

This method has produced some successful advances in understanding delusions (e.g. Blakemore and colleagues focus on delusions of control; section 3.5.2), but it is notable that many of these models focus on what causes the related anomalous experience, but not, as Davies et al. (2001) point out, why a delusional interpretation of such an experience is maintained. Many of the models avoid tackling this issue, and it is notable that those that do include a well-specified maintenance component (e.g. the Institute of Psychiatry model; section 3.4.2) produce a theme-specific explanation for this aspect of their model (in their case, focusing on persecutory delusions and the role of ‘safety behaviours’).

This might further suggest that, when refined to more complete models, there will be separate neuropsychological mechanisms (although with overlapping components) for each sub-type of delusion, that will explain the processes traditionally thought to be shared in belief formation and maintenance. Although various proposals have been offered (e.g. Freeman and Garety suggest emotional theme; Blakemore and colleagues focus on motor control) there is currently no standardised way of classifying these sub-types that is not wedded to the clinical categories derived from the DSM. One aim of the cognitive neuropsychiatric approach might be to be agnostic to the DSM classification, and distinguish delusions based on coherent explanations of the impaired cognitive systems that are identified, rather than assuming these might be different for each DSM classification.

Indeed, a similar approach, aiming to identify the ‘endophenotypes’ of psychiatric disorder has been recently championed by several authors (Gottesman and Gould, 2003; Weiser et al., 2005), who wish to break away from the traditional clinically-led diagnostic approach, to one led by the cognitive, genetic and neurosciences.

In practice, however, this is likely to involve a convergent approach, whereby ‘DSM agnostic’ research is meshed with ‘DSM driven’ research. The modulation of anomalous experience and ideation by TMS in healthy participants is likely to be a useful paradigm in this regard, as it can test hypotheses derived from the ‘driven’ approach in samples of healthy participants who are, therefore, ‘DSM agnostic’ in terms of their being without a psychiatric diagnosis.

#### **8.3.4 Affect and the pragmatic pathology of delusions**

One of the main aims of Chapter 7 was to examine the factors that make delusions pathological in the pragmatic sense. As enshrined in the diagnostic criteria, pathological beliefs are those assumed to be false, fixed, incorrigible and culturally incongruent, whereas pathological perceptions are those that occur without an environmental source. In contrast, Johns and van Os (2001) have noted that these attributes are only likely to represent a position on the psychosis continuum, and that illness behaviour is the factor most likely to mediate the pragmatic path by which a person comes to receive a psychiatric diagnosis or be the focus of clinical concern.

Chapter 7 particularly examined personal distress, an important factor in illness behaviour. It is important to note that this is not a complete account of illness behaviour, as individuals can be considered deluded or psychotic and not experience personal distress at the time (for example, with grandiose delusions, in which someone might positively delight). In these cases, it is social stress (their being perceived as being a problem to others) that is considered as illness behaviour.

It was argued in Chapter 5 that a pathological level of anomalous perceptual experience is not a necessary condition in delusion formation. It is possible, however, that perceptual distortion

combined with distress is a pathway to delusion. One way of investigating this relationship is to compare those who have been diagnosed as having pathological beliefs, to those who, on the surface at least, have personal experiences with similar themes, but are not disabled or impaired. Pagans were invited to participate for exactly this reason, and, when compared both with the general population and deluded patients with and without hallucinations, it was shown that diagnosed pathology was most associated with the presence of distress, which was doubly-dissociated from pathological levels of perceptual distortion.

One possible objection to the conclusions from this study is that it used a cross-sectional design, and so does not take into account the ontology of a delusion; and could, therefore, be missing the dynamic interaction between distress and anomalous experience. For example, delusional patients without hallucinations may not have been distressed when the delusion formed, but became so after a time of living with a potentially disabling belief.

It is unlikely this is the case, however. Although Chapman (1966) noted that initial perceptual changes in schizophrenia could initially be experienced as pleasant but later become a source of anxiety, recent studies on the prodrome of psychosis suggest that psychotic experiences are typically preceded by significant negative emotion, including depression, anxiety and anger (Gourzis et al., 2002). Recent longitudinal studies have also suggested that an anomalous experience accompanied by negative affect at first assessment increases the risk for delusion at the second (Krabbendam and van Os, 2005; Krabbendam et al., 2005).

One noteworthy point, is that in combination with such studies, these findings reported here provide further evidence against Maher's (1988) proposal that anomalous perceptual experience causes anxiety that is relieved by a delusional explanation (section 3.2.1). If

delusions are more likely to occur when perceptual distortion is accompanied by distress, and currently delusional patients with low levels of anomalous experience can show high levels of distress (section 7.5.2), it seems that a delusional explanation is not effectively defending against negative affect.

As a working hypothesis, this would, of course, need to be tested using a longitudinal study on a single sample of participants, rather than inferred from multiple studies, but it is a promising proposition nonetheless, and seems a viable avenue for future research. Following on from the caveats expressed in the last section about explanations being potentially specific to certain sub-types of delusion, it is possible, that the time course of the interaction between distress and unusual experience may be different for different emotions and psychotic themes.

Nevertheless, although this may provide evidence against Maher's (implicit) defence theory (section 3.2.1), it does not necessarily apply to other defence theories (in particular, the Bentall models; sections 3.3.2.1; 3.3.2.2) that argue that low self-esteem or negative self-evaluations are the principle factors which are being defended against, as these models do not include an explicit place for anomalous perceptual experience, and, therefore, have little to say about the detailed interaction between the form of perceptual distortion and distress in delusion formation.

A further conclusion from Chapter 7, was that the appraisal of an experience is likely to be key in mediating illness behaviour, and, therefore, is key in causing a belief to be considered clinically relevant. One problem in creating an integrated theory is a current lack of clarity in understanding the causal role of emotion in this process. If, as suggested by the studies mentioned above, negative affect proceeds and encourages delusional interpretation, it could

be thought of as a contributor to the appraisal process (e.g. trait anxiety increases the risk of a delusional interpretation; Krabbendam and van Os, 2005). Presumably, in line with classic appraisal theories of emotion (Arnold, 1960; Lazarus, 1991), emotion is also the result of an appraisal, including, one would expect, the occurrence of perceptual distortions and anomalous experience.

Notably, Phillips et al. (2003) have proposed a model of emotion perception (derived largely from the psychopathological literature) that might account for this feedback mechanism, as it includes a regulatory component which allows for an ongoing feedback of affect into the appraisal process. This suggests, in a similar fashion to the Attribution / Self-representation model (although not necessarily using the concept of self-esteem), that the regulatory process may be a central focus for explaining illness behaviour. Those whose affective regulatory system is highly reactive when anomalous experience occurs may be more susceptible to forming delusions.

Dynamic models are notoriously difficult to test, however, particularly on the cognitive and behavioural level where there is no objectively agreed phenomena to be measured. This is where the integration of cognitive neuroscience is likely to be a hugely beneficial, as convergent evidence from methods that have the possibility of measuring more objective phenomena (subjective analysis criteria in functional neuroimaging studies aside) are likely to constrain accounts relying purely on cognitive or phenomenological components.

#### **8.4 Implications for theories of normal belief**

Several approaches were outlined in sections 1.3.1 to 1.3.6 that suggested structures of belief and how they might be represented in the brain. The most compatible model with the results

and analysis presented in this thesis is undoubtedly the liberal dispositional account of belief primarily, proposed by Schwitzgebel (2002; section 1.5.2), specifically as applied to delusions by Bayne and Pacherie (2005; section 2.5.3).

The Bayne and Pacherie model is notable as it best fulfils the criteria outlined in section 1.5 (“What Must a Model of Belief Formation Include ?”): it includes both conscious and unconscious aspects, embracing both the unconscious influences of schemas and context on belief-based action (‘action’ includes mental states as well as externally observable behaviour); and the conscious influence of rationality, and, presumably, possible control of any conscious states that do arise. It also allows for a ‘web of belief’, and, almost uniquely, describes this as a well defined cognitive process, in this case, as the structure of long-term memory.

The role of affect, and motivational states in general, is explicitly included as having an influence on belief-based action (via, for example, affect driven memory retrieval biases). The ability to account for authority beliefs is well accounted for, owing to the reliance on long-term memory structures, and confidence or certainty in a belief is considered to be mediated by a disposition to feel a certain emotion also a possible form of belief-based action in Schwitzgebel’s account.

From this account, belief-claims or belief-based actions (including both internal mental events or externally observable motor acts) do not necessarily have to rely on pre-existing propositional representations residing somewhere in the brain. They are the output of the dispositional system which leads a person to make such a claim, carry out a certain act, or experience a certain form of mental phenomenon in response to a certain situation.



If a prior proposition did exist (such as a long-term semantic memory) it would, of course, influence this process, so the dispositional system would produce a proposition-congruent claim or action. It could be overridden, however, by contextual factors or emotion, so a person might (for example) believe house spiders to be harmless but still produce actions that would seem to be more in line with the belief that they were personally dangerous (and, perhaps, cause others to attribute this latter belief to them). In retrospect, any such action could be ascribed a propositional description, either by the person themselves, or by a third-party (not unlike in interpretationist accounts of belief; section 1.3.4) even if it did not have a propositional representation in long-term memory at the time the dispositional system was involved in producing the action or mental event.

Arguments that suggest delusions are not a unitary phenomenon are compatible with this approach, and it may account for why there is such disagreement over whether delusions are nothing more than 'empty speech acts' at one extreme (section 2.5.1), to traditional folk-psychological representations of belief at the other (section 2.5.4). With a liberal dispositional account, sometimes beliefs will seem like the folk-psychological description, in that they will primarily reflect propositional content stored in long-term memory structures; but at other times they will seem more like empty speech acts, when the dispositional process activates speech and other non-consequential acts, but is over-ridden by other factors that prevent the likelihood of more substantial actions.

Nevertheless, one potential criticism of this model is that it fails to distinguish between internal events experienced as thoughts and those experienced or communicated as beliefs. This is brought into stark relief when a comparison between obsessive thoughts (prevalent in

obsessive-compulsive disorder or OCD) and delusions are considered, as the current dispositional model does not seem able to adequately distinguish between the two. A similar break-down in a dispositional system could result in intrusive thoughts, although in the type that typically occur in OCD, they are experienced as alien and are resisted, rather than adopted as part of a personal epistemology.

Why this occurs is not clear, although there are clues from studies of metacognition in healthy participants. One concept which seems particularly relevant is the process called ‘feeling of knowing’, which has been studied extensively in the memory literature (review in Koriat, 2000) and is also used in face recognition models (Bruce and Young, 1986). The ‘feeling of knowing’ is an automatic process which is related to a conviction that an event has occurred or has been encountered before, regardless of whether a conscious memory can be retrieved (Rajaram, 1993; Koriat, 2000). One hypothesis suggested by this work is that delusions (or at least, some delusions) may be given their belief-like conviction by pathology of the ‘feeling of knowing’ system.

Interestingly, it is pathology to the ‘feeling of knowing’ aspect of the Bruce and Young (1986) face recognition model that is thought to go awry in Capgras delusion (Ellis and Lewis, 2001), and the idea that affect, or affect-like processes may provide this form of information exists in several models of affect and reasoning (Damasio, 1996; Clore and Gasper, 2000). Similarly, recent models of OCD suggest a pathology to ‘feeling of knowing’ (Szechtman and Woody, 2004; Woody et al., 2005), although the problem is thought to be an impairment of activation, leading to a pathological ‘doubting’, typically resulting in checking or washing, despite a conscious memory of having completed the act.

Although this explanation is still speculative and remains untested, from this perspective, both OCD and delusions could be seen as pathologies of belief, which may explain the continuum between OCD and delusion seen in clinical practice (O'Dwyer and Marks, 2000). Owing to the large amount of research on the 'feeling of knowing', particularly in the memory literature, this hypothesis does lead to some immediately testable hypotheses, as variables drawn from this tradition could be measured in delusional patients to look for substantial differences.

Despite these shortcomings, however, Bayne and Pacherie's model can be seen as a major advance, in that it suggests that belief as a circumscribed phenomenon represented in the brain does not necessarily exist, and that the notional mechanisms of belief formation can be reduced to other neuropsychological processes. What their model is light on, however, is elucidating how the competing demands of context, schema, emotion, rationality, and so on are balanced to produce the relevant output.

Clinical models that suggest contributory factors to delusion formation are likely to indicate the nature of some of these demands, particularly where the normal dispositional process can be seen to have broken down, or is subject to pathological bias. Indeed, such inferences are possible even when the clinical models are not explicitly framed as models of normal belief themselves (e.g. IoP model, section 3.4; Bentall's Attribution / Self-representation model, section 3.3.2.2).

It is important to note, however, that a careful distinction needs to be made between delusions, in the pragmatic clinical sense, and magical or anomalous beliefs. The evidence presented in Chapter 7 suggests that distress is a central component in delusion formation (at

least for some, and possibly the majority, of delusions). Purely cognitive accounts that leave little room for the role of affect (e.g. Langdon and Coltheart, 2000) are not able to make the distinction between delusional beliefs, and, for example, magical, anomalous, or even, spiritual beliefs.

Perhaps there needs to be two (perhaps overlapping) explanations, one which explains how 'magical' beliefs are formed, and another concerning how they may become associated with distress and disability in some people. As noted previously (section 8.3.4) the interaction between these factors is likely to take the form of a cyclic process that has a significant role for processes that feedback in both directions.

Although these aspects are likely to interact, it might be possible to identify neuropsychological processes that contribute more to one than the other. The evidence from this thesis suggests that temporal lobe disturbance may be more likely to contribute to magical thinking than distress, as it was shown to affect the perception of meaning in noise in Chapter 6, and was no more prevalent in the non-clinical than clinical groups in the CAPS item odds ratio analyses (tables 5.7; 7.5).

In terms of normal belief, magical thinking linked to temporal lobe function may contribute to creativity and originality. Certainly, temporal lobe function has been linked to cognitive flexibility and the experience of 'flow' (Dietrich, 2004) and single case studies have linked remarkable talent to temporal lobe epilepsy with a likely right-sided foci (Murai et al., 1998), and great right-than-left temporal atrophy to the new emergence of creative talent (Miller et al., 1996). Indeed, some authors (Leonhard and Brugger, 1998; Pizzagalli et al., 2000; Weinstein and Graves, 2002) have explicitly proposed a continuum of magical thinking from

creativity to frank delusional belief, although with the previous distinction in mind, this might be better considered as a continuum from creativity to wildly anomalous or magical belief.

It is possible that this magical thinking component is what is represented by the distribution of the difference between CAPS and PDI scores, represented in figure 5.3; although, admittedly, this is a preliminary result, and needs further investigation. The fact that it follows a normal distribution so closely though, does suggest that it is not a random result and merits further consideration.

In terms of distress, it is likely that this form of negative affect might reflect the ‘threshold’ discussed in certain models of psychosis (Johns and van Os, 2001; Vinogradov et al., 1992; section 3.8.4; Serper et al., 2005) and the component which seems to be the stumbling block for the hemispheric asymmetry models (section 3.6; McKay et al., 2005; section 3.2.3.2). The fact that distress and anomalous experience seem to be able to dissociate, and the fact that there is evidence that delusion-like belief and distress are subject to a similar dissociation (Peters et al., 1999), may provide some evidence for models of how emotion and belief combine structurally, particularly in light of Spicer’s (2004) models of this relationship (section 1.5.4).

To recap, Spicer has outlined three potential ways in which emotion and belief might interact: The ‘hybrid view’ is that emotions are beliefs and desires; the umbrella view is that emotions behave like beliefs and desires (i.e. they have a propositional content and intentionality); and the nomological view is that emotions carry beliefs and desires with them (i.e. they correlate). The evidence presented in this thesis suggests that the nomological view is most likely to be

correct, as although delusion and distress are correlated, they do not seem structurally bound or equivalent.

## **8.5 Chapter conclusions**

The psychiatric label of ‘delusion’ is an umbrella term for a variety of pathologies that result in a person making an unlikely belief-claim, or being ascribed a belief on the basis of their behaviour, in tandem with the person showing significant distress and / or causing social stress. The label of ‘belief’ is likely to reflect the output of a similarly diverse set of processes that result in a belief-claim, or a belief-ascription by a third party.

It is likely that the one-stage / two-stage debate is misleading, as both argue for the necessity of anomalous perceptual experience in delusion formation, and, instead, research should focus on finding non-sequential contributory factors.

It is important to distinguish between anomalous and delusional beliefs on pragmatic clinical grounds. Models that cannot distinguish between these two aspects have little to say about what makes a belief pathological. It is likely that temporal lobe disturbance may contribute towards forming magical beliefs, and, perhaps, in less obviously anomalous cases, to creativity. The role of appraisal in both forming beliefs and causing emotion suggests that a dynamic feedback system may be the key to understanding how distress and belief become pathologically associated.

The model that is most likely to support both the requirements of a theory of normal belief, and the evidence from the delusions literature, is the liberal dispositional account of Bayne and Pacherie (2005).

## **8.6 Concluding remarks**

Belief is a construct which has been largely neglected by neuropsychology, an omission which is becoming increasingly apparent as the understanding and importance of delusions as apparently pathological beliefs becomes highlighted by a growing literature. This increasing focus is leading to the development of effective therapeutic approaches that apply techniques derived largely from an understanding of normal belief and brain function. In light of this, the importance of appreciating the differences and similarities between benign beliefs and those that cause distress or impairment is becoming increasingly apparent, particularly when framing clinical therapies and deploying them for the benefit of affected individuals. It is hoped that this thesis successfully contributes towards such an understanding, and has the potential to complement both the academic research in this area and the science of clinical practice.

## 8.7 References

- Arnold, M.B. (1960) *Emotion and Personality*. New York: Columbia University Press.
- Bayne, T., Pacherie, E. (2005) In Defence of the Doxastic Conception of Delusions. *Mind and Language*, 20, 163-188.
- Bruce, V., Young, A. (1986) Understanding face recognition. *The British Journal of Psychology*, 77, 305-327.
- Chapman J. (1966) The early symptoms of schizophrenia. *British Journal of Psychiatry*, 112, 225-51
- Clore, G.L., Gasper, K. (2000) Some affective influences on beliefs. In Frijda, N.H., Manstead, A.S.R., Bem, S.(eds) *Emotions and Beliefs: How Feelings Influence Thoughts*. Cambridge: Cambridge University Press.
- Damasio AR. (1996) The somatic marker hypothesis and the possible functions of the prefrontal cortex. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, 351 (1346), 1413-20.
- Davidson, L. L. Heinrichs, R. W. (2003) Quantification of frontal and temporal lobe brain-imaging findings in schizophrenia: a meta-analysis. *Psychiatry Research*, 122, 69-87.
- Davies, M., Coltheart, M., Langdon, R., & Breen, N. (2001) Monothematic delusions: Towards a two-factor account. *Philosophy, Psychiatry, and Psychology*, 8, 133-158.



Dietrich A. (2004) Neurocognitive mechanisms underlying the experience of flow.

*Consciousness and Cognition*, 13, 746-61.

Ellis, H. D., Lewis, M. B. (2001) Capgras delusion: a window on face recognition. *Trends in Cognitive Sciences*, 5, 149-156.

Freeman, D., Garety, P. (2004) *Paranoia: The Psychology of Persecutory Delusions*. Hove: Psychology Press.

Gilleen, J., David, A. S. (2005) The cognitive neuropsychiatry of delusions: from psychopathology to neuropsychology and back again. *Psychological Medicine*, 35, 5-12.

Gilovich, T. (1993) *How we know what isn't so: The fallibility of human reason in everyday life*. New York: Free Press.

Gottesman II, Gould TD. (2003) The endophenotype concept in psychiatry: etymology and strategic intentions. *American Journal of Psychiatry*, 160, 636-45.

Gourzis P, Katrivanou A, Beratis S. (2002) Symptomatology of the initial prodromal phase in schizophrenia. *Schizophrenia Bulletin*, 28, 415-29.

Johns, L. C., van Os, J. (2001) The continuity of psychotic experiences in the general population. *Clinical Psychology Review*, 21, 1125-1141.

Johnson, M.K. Raye, C.L. (2000) Cognitive and brain mechanisms of false memories and beliefs. In D.L. Schacter and E. Scarry (eds) *Memory, Brain and Belief*. Cambridge Massachusetts: MIT Press.

Koriat A. (2000) The feeling of knowing: some metatheoretical implications for consciousness and control. *Consciousness and Cognition*, 9, 149-71.

Krabbendam, L., Myin-Germeys, I., Hanssen, M., de Graaf, R., Vollebergh, W., Bak, M., & van Os, J. (2005) Development of depressed mood predicts onset of psychotic disorder in individuals who report hallucinatory experiences. *British Journal of Clinical Psychology*, 44, 113-125.

Krabbendam, L., van Os, J. (2005) Affective processes in the onset and persistence of psychosis. *European Archives of Psychiatry and Clinical Neuroscience*, 255, 185-189.

Langdon, R. Coltheart, M. (2000) The cognitive neuropsychology of delusions. In M. Coltheart and M. Davies (eds) *Pathologies of Belief*. Oxford: Blackwell Publishing.

Lazarus, R.S. (1991) Cognitive and motivation in emotion. *American Psychologist*, 46, 352-367.

Leonhard D, Brugger P. (1998) Creative, paranormal, and delusional thought: a consequence of right hemisphere semantic activation? *Neuropsychiatry, Neuropsychology and Behavioural Neurology*, 11, 177-183.

- Maher, B. (1988) Anomalous experience and delusional thinking: The logic of explanations. In T.F. Oltmanns, B.A. Maher (eds) *Delusional beliefs*. Chichester: Wiley.
- Makarec, K., Persinger, M. A. (1985) Temporal lobe signs: electroencephalographic validity and enhanced scores in special populations. *Perceptual and Motor Skills*, 60, 831-842.
- McKay, R., Langdon, R., Coltheart, M. (2005) "Sleights of mind": Delusions, defences and self-deception. *Cognitive Neuropsychiatry*, 10, 205-326.
- Miller BL, Ponton M, Benson DF, Cummings JL, Mena I. (1996) Enhanced artistic creativity with temporal lobe degeneration. *Lancet*, 348 (9043), 1744-5
- Murai T, Hanakawa T, Sengoku A, Ban T, Yoneda Y, Fujita H, Fujita N. (1998) Temporal lobe epilepsy in a genius of natural history: MRI volumetric study of postmortem brain. *Neurology*, 50, 1373-6.
- O'Dwyer, A. M., Marks, I. (2000) Obsessive-compulsive disorder and delusions revisited. *British Journal of Psychiatry*, 176, 281-284.
- Ohayon, M. M., & Schatzberg, A. F. (2002) Prevalence of depressive episodes with psychotic features in the general population. *American Journal of Psychiatry*, 159, 1855-1861.
- Peters, E., Day, S., McKenna, J., Orbach, G. (1999) Delusional ideation in religious and psychotic populations. *British Journal of Clinical Psychology*, 38, 83-96.

Phillips ML, Drevets WC, Rauch SL, Lane R. (2003) Neurobiology of emotion perception I: Basis of normal emotion perception. *Biological Psychiatry*, 54 (5), 504-514.

Pizzagalli D, Lehmann D, Gianotti L, Koenig T, Tanaka H, Wackermann J and Brugger P. (2000) Brain electric correlates of strong belief in paranormal phenomena: intracerebral EEG source and regional Omega complexity analyses. *Psychiatry Research*, 100, 139-154.

Rajaram, S. (1993) Remembering and knowing: Two means of access to the personal past. *Memory and Cognition*, 21, 89-102.

Ruppin, E., Reggia, J. A., Horn, D. (1996) Pathogenesis of schizophrenic delusions and hallucinations: a neural model. *Schizophrenia Bulletin*, 22, 105-123.

Sackeim HA. (1998) The meaning of insight. In: Amador XF, David, AS, eds. *Insight and Psychosis (1st ed)*. Oxford: Oxford University Press.

Schwitzgebel, E. (2002) A phenomenal, dispositional account of belief. *Nous*, 36, 249-275.

Serper, M., Dill, C. A., Chang, N., Kot, T., Elliot, J. (2005) Factorial structure of the hallucinatory experience: continuity of experience in psychotic and normal individuals. *Journal of Nervous and Mental Disease*, 193, 265-272.

Siever LJ, Davis KL. (2004) The pathophysiology of schizophrenia disorders: perspectives from the spectrum. *American Journal of Psychiatry*, 161, 398-413.

Spicer, F. (2004) Emotional behaviour and the scope of belief-desire explanation. In D. Evans and P. Cruse (eds) *Emotion, Evolution and Rationality*. Oxford: Oxford University Press.

Stone, T., Young, A.W. (1997) Delusions and brain injury: The philosophy and psychology of belief. *Mind and Language*, 12, 327-364.

Sumich, A., Chitnis, X. A., Fannon, D. G., O'Ceallaigh, S., Doku, V. C., Faldrowicz, A., Sharma, T. (2005) Unreality symptoms and volumetric measures of Heschl's gyrus and planum temporal in first-episode psychosis. *Biological Psychiatry*, 57, 947-950.

Szechtman H, Woody E. (2004) Obsessive-compulsive disorder as a disturbance of security motivation. *Psychological Review*, 111, 111-27.

Vinogradov S, King RJ, Huberman BA. (1992) An associationist model of the paranoid process: application of phase transitions in spreading activation networks. *Psychiatry*, 55, 79-94.

Walsh V and Pascual-Leone A. (2003) *Transcranial magnetic stimulation: A neurochronometrics of mind*. MIT: MIT Press.

Weinberger, D.R. (1991) Anteromedial temporal-prefrontal connectivity: A functional neuroanatomical system implicated in schizophrenia. In B.J. Carroll and J.E. Barrett (eds) *Psychopathology and the Brain*. New York: Raven Press.

Weinstein S and Graves RE. (2002) Are creativity and schizotypy products of a right hemisphere bias? *Brain and Cognition*, 49, 138-151.

Weiser M, van Os J, Davidson M. (2005) Time for a shift in focus in schizophrenia: from narrow phenotypes to broad endophenotypes. *British Journal of Psychiatry*, 187, 203-5.

Woody EZ, Lewis V, Snider L, Grant H, Kamath M, Szechtman H. (2005) Induction of compulsive-like washing by blocking the feeling of knowing: An experimental test of the security-motivation hypothesis of obsessive-compulsive disorder. *Behavioral and Brain Functions*, 1, 11.

## **Appendix I**

### **The Cardiff Anomalous Perceptions Scale (CAPS)**

### Introduction

This questionnaire asks questions about sensations and perceptions you may have experienced. Some of the experiences are unusual, some of them are more everyday.

We realise circling answers may not always represent your experience as accurately as you might like. However, we would ask you to circle the answers that most closely match your experience and avoid missing any questions out.

We would appreciate it if you could be as honest as possible when giving your answers.

*The only experiences we are not interested in are those that may have occurred whilst under the influence of drugs.*

### Instructions

Each item has a question on the left hand side. Please read the question and circle either YES or NO

- If you circle **NO** please move straight on to the next question.
- If you circle **YES** please rate the experience *in all of the three boxes* on the right hand side of the item by circling a number between 1 and 5.

These ask about how distressing you found the experience, how distracting you found it, and how often the experience occurs.

### Example questions

You do not need to answer these questions, they are just examples to illustrate the instructions.

Do you ever notice that lights seem to flicker on and off for no reason ?

**NO**     **YES**

If YES please rate on right hand side.

Not at all distressing	1	2	3	4	Very distressing
Not at all distracting	1	2	3	4	Completely intrusive
Happens hardly at all	1	2	3	4	Happens all the time

Do you ever feel that the sound on the TV or radio seems unusually quiet ?

**NO**     **YES**

If YES please rate on right hand side.

Not at all distressing	1	<input checked="" type="radio"/> 2	3	4	Very distressing
Not at all distracting	1	2	<input checked="" type="radio"/> 3	4	Completely intrusive
Happens hardly at all	1	<input checked="" type="radio"/> 2	3	4	Happens all the time



**1) Do you ever notice that sounds are much louder than they normally would be ?**

NO YES

If YES please rate on right hand side.

Not at all distressing	1	2	3	4	Very distressing
Not at all distracting	1	2	3	4	Completely intrusive
Happens hardly at all	1	2	3	4	Happens all the time

**2) Do you ever sense the presence of another being, despite being unable to see any evidence ?**

NO YES

If YES please rate on right hand side.

Not at all distressing	1	2	3	4	Very distressing
Not at all distracting	1	2	3	4	Completely intrusive
Happens hardly at all	1	2	3	4	Happens all the time

**3) Do you ever hear your own thoughts repeated or echoed ?**

NO YES

If YES please rate on right hand side.

Not at all distressing	1	2	3	4	Very distressing
Not at all distracting	1	2	3	4	Completely intrusive
Happens hardly at all	1	2	3	4	Happens all the time

**4) Do you ever see shapes, lights or colours even though there is nothing really there ?**

NO YES

If YES please rate on right hand side.

Not at all distressing	1	2	3	4	Very distressing
Not at all distracting	1	2	3	4	Completely intrusive
Happens hardly at all	1	2	3	4	Happens all the time

**5) Do you ever experience unusual burning sensations or other strange feelings in or on your body ?**

NO YES

If YES please rate on right hand side.

Not at all distressing 1	2	3	4	Very distressing 5
Not at all distracting 1	2	3	4	Completely intrusive 5
Happens hardly at all 1	2	3	4	Happens all the time 5

**6) Do you ever hear noises or sounds when there is nothing about to explain them ?**

NO YES

If YES please rate on right hand side.

Not at all distressing 1	2	3	4	Very distressing 5
Not at all distracting 1	2	3	4	Completely intrusive 5
Happens hardly at all 1	2	3	4	Happens all the time 5

**7) Do you ever hear your own thoughts spoken aloud in your head, so that someone near might be able to hear them ?**

NO YES

If YES please rate on right hand side.

Not at all distressing 1	2	3	4	Very distressing 5
Not at all distracting 1	2	3	4	Completely intrusive 5
Happens hardly at all 1	2	3	4	Happens all the time 5

**8) Do you ever detect smells which don't seem to come from your surroundings ?**

NO YES

If YES please rate on right hand side.

Not at all distressing 1	2	3	4	Very distressing 5
Not at all distracting 1	2	3	4	Completely intrusive 5
Happens hardly at all 1	2	3	4	Happens all the time 5

9) Do you ever have the sensation that your body, or a part of it, is changing or has changed shape ?

NO YES

If YES please rate on right hand side.

Not at all distressing	1	2	3	4	Very distressing
Not at all distracting	1	2	3	4	Completely intrusive
Happens hardly at all	1	2	3	4	Happens all the time

10) Do you ever have the sensation that your limbs might not be your own or might not be properly connected to your body?

NO YES

If YES please rate on right hand side.

Not at all distressing	1	2	3	4	Very distressing
Not at all distracting	1	2	3	4	Completely intrusive
Happens hardly at all	1	2	3	4	Happens all the time

11) Do you ever hear voices commenting on what you are thinking or doing ?

NO YES

If YES please rate on right hand side.

Not at all distressing	1	2	3	4	Very distressing
Not at all distracting	1	2	3	4	Completely intrusive
Happens hardly at all	1	2	3	4	Happens all the time

12) Do you ever feel that someone is touching you, but when you look nobody is there ?

NO YES

If YES please rate on right hand side.

Not at all distressing	1	2	3	4	Very distressing
Not at all distracting	1	2	3	4	Completely intrusive
Happens hardly at all	1	2	3	4	Happens all the time

**13) Do you ever hear voices saying words or sentences when there is no-one around that might account for it ?**

NO YES

If YES please rate on right hand side.

Not at all distressing	1	2	3	4	Very distressing
Not at all distracting	1	2	3	4	Completely intrusive
Happens hardly at all	1	2	3	4	Happens all the time

**14) Do you ever experience unexplained tastes in your mouth ?**

NO YES

If YES please rate on right hand side.

Not at all distressing	1	2	3	4	Very distressing
Not at all distracting	1	2	3	4	Completely intrusive
Happens hardly at all	1	2	3	4	Happens all the time

**15) Do you ever find that sensations happen all at once and flood you with information ?**

NO YES

If YES please rate on right hand side.

Not at all distressing	1	2	3	4	Very distressing
Not at all distracting	1	2	3	4	Completely intrusive
Happens hardly at all	1	2	3	4	Happens all the time

**16) Do you ever find that sounds are distorted in strange or unusual ways ?**

NO YES

If YES please rate on right hand side.

Not at all distressing	1	2	3	4	Very distressing
Not at all distracting	1	2	3	4	Completely intrusive
Happens hardly at all	1	2	3	4	Happens all the time

**17) Do you ever have difficulty distinguishing one sensation from another ?**

NO YES

If YES please rate on right hand side.

Not at all distressing 1	2	3	4	Very distressing 5
Not at all distracting 1	2	3	4	Completely intrusive 5
Happens hardly at all 1	2	3	4	Happens all the time 5

**18) Do you ever smell everyday odours and think that they are unusually strong ?**

NO YES

If YES please rate on right hand side.

Not at all distressing 1	2	3	4	Very distressing 5
Not at all distracting 1	2	3	4	Completely intrusive 5
Happens hardly at all 1	2	3	4	Happens all the time 5

**19) Do you ever find the appearance of things or people seems to change in a puzzling way, e.g. distorted shapes or sizes or colour ?**

NO YES

If YES please rate on right hand side.

Not at all distressing 1	2	3	4	Very distressing 5
Not at all distracting 1	2	3	4	Completely intrusive 5
Happens hardly at all 1	2	3	4	Happens all the time 5

**20) Do you ever find that your skin is more sensitive to touch, heat or cold than usual ?**

NO YES

If YES please rate on right hand side.

Not at all distressing 1	2	3	4	Very distressing 5
Not at all distracting 1	2	3	4	Completely intrusive 5
Happens hardly at all 1	2	3	4	Happens all the time 5

21) Do you ever think that food or drink tastes much stronger than it normally would ?

NO YES

If YES please rate on right hand side.

Not at all distressing	1	2	3	4	Very distressing
1	2	3	4	5	
Not at all distracting	1	2	3	4	Completely intrusive
1	2	3	4	5	
Happens hardly at all	1	2	3	4	Happens all the time
1	2	3	4	5	

22) Do you ever look in the mirror and think that your face seems different from usual ?

NO YES

If YES please rate on right hand side.

Not at all distressing	1	2	3	4	Very distressing
1	2	3	4	5	
Not at all distracting	1	2	3	4	Completely intrusive
1	2	3	4	5	
Happens hardly at all	1	2	3	4	Happens all the time
1	2	3	4	5	

23) Do you ever have days where lights or colours seem brighter or more intense than usual ?

NO YES

If YES please rate on right hand side.

Not at all distressing	1	2	3	4	Very distressing
1	2	3	4	5	
Not at all distracting	1	2	3	4	Completely intrusive
1	2	3	4	5	
Happens hardly at all	1	2	3	4	Happens all the time
1	2	3	4	5	

24) Do you ever have the feeling that of being uplifted, as if driving or rolling over a road while sitting quietly ?

NO YES

If YES please rate on right hand side.

Not at all distressing	1	2	3	4	Very distressing
1	2	3	4	5	
Not at all distracting	1	2	3	4	Completely intrusive
1	2	3	4	5	
Happens hardly at all	1	2	3	4	Happens all the time
1	2	3	4	5	

**25) Do you ever find that common smells sometimes seem unusually different ?**

NO YES

If YES please rate on right hand side.

Not at all distressing	1	2	3	4	Very distressing
Not at all distracting	1	2	3	4	Completely intrusive
Happens hardly at all	1	2	3	4	Happens all the time

**26) Do you ever think that everyday things look abnormal to you ?**

NO YES

If YES please rate on right hand side.

Not at all distressing	1	2	3	4	Very distressing
Not at all distracting	1	2	3	4	Completely intrusive
Happens hardly at all	1	2	3	4	Happens all the time

**27) Do you ever find that your experience of time changes dramatically ?**

NO YES

If YES please rate on right hand side.

Not at all distressing	1	2	3	4	Very distressing
Not at all distracting	1	2	3	4	Completely intrusive
Happens hardly at all	1	2	3	4	Happens all the time

**28) Have you ever heard two or more unexplained voices talking with each other ?**

NO YES

If YES please rate on right hand side.

Not at all distressing	1	2	3	4	Very distressing
Not at all distracting	1	2	3	4	Completely intrusive
Happens hardly at all	1	2	3	4	Happens all the time

**29) Do you ever notice smells or odours that people next to you seem unaware of ?**

NO YES If YES please rate on right hand side.	Not at all distressing 1      2      3      4      5 Very distressing
	Not at all distracting 1      2      3      4      5 Completely intrusive
	Happens hardly at all 1      2      3      4      5 Happens all the time

**30) Do you ever notice that food or drink seems to have an unusual taste ?**

NO YES If YES please rate on right hand side.	Not at all distressing 1      2      3      4      5 Very distressing
	Not at all distracting 1      2      3      4      5 Completely intrusive
	Happens hardly at all 1      2      3      4      5 Happens all the time

**31) Do you ever see things that other people cannot ?**

NO YES If YES please rate on right hand side.	Not at all distressing 1      2      3      4      5 Very distressing
	Not at all distracting 1      2      3      4      5 Completely intrusive
	Happens hardly at all 1      2      3      4      5 Happens all the time

**32) Do you ever hear sounds or music that people near you don't hear ?**

NO YES If YES please rate on right hand side.	Not at all distressing 1      2      3      4      5 Very distressing
	Not at all distracting 1      2      3      4      5 Completely intrusive
	Happens hardly at all 1      2      3      4      5 Happens all the time



## **Appendix II**

**Which factors on the Cardiff Anomalous Perceptions Scale (CAPS) are related to temporal lobe disturbance?**

One of the extracted components from the principal components analysis of the Cardiff Anomalous Perceptions Scale (CAPS; Bell et al., in revision; Chapter 5) items was interpreted as reflecting experiences related to temporal lobe disturbance (section 5.5.5). This conclusion is tentative, however, and needs to be better validated before it can be accepted as a firm conclusion.

Makarec and Persinger's (1985) Temporal Lobe Scale (TLS) is a measure of experiences linked to both clinical and non-clinical temporal lobe disturbance, which has been well validated using both electrophysiological measures, and in groups with high levels of anomalous experiences and beliefs (Makarec and Persinger, 1985; 1990; Persinger and Fisher, 1990).

If the temporal lobe factor of the CAPS (table 5.7) was genuinely predicting temporal lobe disturbance, rather than simply reflecting, for example, an attenuated or sub-clinical range of experiences, it should predict the score on the TLS to a far greater degree than the other CAPS factors.

This was tested on a sample of 39 undergraduate students (males = 11, females = 28; mean age = 19.57, SD = 1.49, range 18-25), who completed both the CAPS and TLS as part of a wider study on anomalous experience and cognitive performance, conducted by Caroline Dietrich.

The CAPS temporal lobe factor consisted of the items specified in table 5.7, except for two items (CAPS items 2 and 24) which were directly taken from the TLS during the development of the CAPS.

The standard multiple regression was carried out with the three CAPS factors (chemosensation, clinical psychosis and the modified-temporal lobe) as independent variables, and the TLS score as a dependent variable. Results are given in table A.1 below.

<i>CAPS subscale</i>	<i>Beta</i>	<i>t</i>	<i>p</i>
Temporal lobe	.600	4.306	< .0005
Chemosensation	.234	1.738	.091
Clinical psychosis	.104	1.016	.317

*table A.1 Multiple regression results*

As can be seen from table A.1, the modified temporal lobe factor on the CAPS is the greatest contributor to the variance of the TLS, and the only one that reaches significance for the test of unique contribution to the TLS score. This provides evidence that that the temporal lobe factor on the CAPS is reflecting temporal lobe disturbance, although the small sample size means these results must be considered preliminary.

## References

Bell, V., Halligan, P.W. & Ellis, H.D. (2006) The Cardiff Anomalous Perceptions Scale (CAPS) and the Role of Anomalous Perceptual Experience in Delusion Formation.

*Schizophrenia Bulletin*, 32, 366-77.

Makarec, K., & Persinger, M. A. (1985) Temporal lobe signs: electroencephalographic validity and enhanced scores in special populations. *Perceptual and Motor Skills*, 60, 831-842.

Makarec, K., & Persinger, M. A. (1990) Electroencephalographic validation of a temporal lobe signs inventory in a normal population. *Journal of Research in Personality*, 24, 323-337.

Persinger, M. A., & Fisher, S. D. (1990) Elevated, specific temporal lobe signs in a population engaged in psychic studies. *Perceptual and Motor Skills*, 71, 817-818.

