Cognitive mechanisms associated with clinical and non-clinical psychotic experiences

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Thesis submitted for the degree of Doctor of Philosophy

Durham University, 2009

6 MAY 2009
Declaration

The research contained in this thesis was carried out by the author between 2005 and 2008 while a postgraduate student in the Department of Psychology at Durham University. None of the work contained in this thesis has been submitted in candidature for any other degree. The Rasch analyses reported in Chapter 3 were performed by David Meads of the Galen Institute. The author was assisted by Lee de-Wit in some of the data collection reported in Chapter 6.

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Acknowledgements

Firstly, I would like to thank Dr Charles Fernyhough for his generosity with his time, humouring my work cycle, and going beyond what I could ever have expected from a supervisor. Thanks are also due to my second supervisors, Prof Findlay and Dr John. Many other staff at Durham University have provided fantastic support for me over the past three years. Elaine Stanton, Richard Stock, Bob Metcalf, and Dr Mike Burt have given me invaluable computing support, and Drs Steve Muncer and Bob Williams have given me valuable statistical guidance. I am also very grateful to Dr Vincent Reid for the significant effort, time and patience he put into teaching me electrophysiology.

I was fortunate to receive an Economic and Social Research Council and Medical Research Council Studentship which allowed my work to take place. Thanks also go to Dr Robyn Langdon and Prof Max Coltheart for inviting me to, and hosting me at, Macquarie University, Sydney, Australia, where I spent an extremely enjoyable research visit.

Over the past three years I have been privileged to meet many people through the Hearing Voices Network, including Peter Bullimore, Rufus May, and Jacqui Dillon, among many others. Talking with them has radically changed my views of how we should approach hallucinations. Before I met them I was interested in hallucinations. Now I am interested in the person behind the hallucination. They humbled and inspired me.

I must also thank my Mum, Rosemary, for teaching me to read, and my Dad, David, for teaching me to drive. I literally wouldn’t have have got to Durham otherwise. Last but not least I must thank, my now fiancée, Roseline. I couldn’t have got through my PhD without her patience, support, love and understanding.
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Appendix A


Appendix B

Last year's words belong to last year's language/

And next year's words await another voice.

T. S. Eliot (1888–1965)

Accountants rampant/ Oh, you know I couldn't last

Morrissey (1959–)
ABSTRACT

The studies reported in this thesis were designed to address several important issues in symptom-specific cognitive models of psychosis. The design of these studies was guided by a commitment to the continuity hypothesis of psychosis, which holds that psychotic experiences exist on a continuum stretching into the healthy population. The thesis firstly examines a two-factor cognitive model of persecutory ideation, focusing primarily on the roles of thought suppression, intrusive thoughts, anxiety, social rank, and the jumping to conclusions bias. The thesis then turns to an examination of cognitive factors involved in hallucinations and, in particular, auditory verbal hallucinations. Chapters in this section describe a series of experimental and theoretical studies of the relations between intrusive thoughts and hallucinations, agency and hallucinations, and the role of inner speech in auditory verbal hallucinations.

Two-factor models of persecutory delusion (PD) formation propose that in the first stage of PD formation an initial implausible idea is triggered. The second stage of PD formation is then the uncritical adoption of such a thought as a belief, which may be due to cognitive biases such as the jumping to conclusions (JTC) bias often present in those with PDs. The first study (Chapter 1) investigated whether thought suppression, and its interaction with anxiety, was associated with levels of non-clinical persecutory delusion-like beliefs (PDLBs). It was hypothesised that thought suppression could play a role in the formation and maintenance of PDLBs through its tendency to lead to intrusive thoughts, and to trigger initial implausible ideas. Consistent with this proposal, thought suppression was positively associated with PDLBs only when anxiety was high.
The second study (Chapter 2) examined a prediction of the two-factor model, namely that a second-stage factor, the jumping to conclusions (JTC) bias, should interact with first-stage factors, specifically social anxiety, social rank, anomalous experiences and thought suppression. Consistent with the two-factor model, the JTC bias was found not to be an independent predictor of PDLB levels, but its interaction with social rank was a significant predictor of PDLBs. It was concluded that although evidence was found for the two-factor model, the presence of the JTC bias was neither a sufficient nor necessary condition for PDLB formation.

In addition to being postulated to play a role in persecutory delusion formation, intrusive thoughts have been implicated in the formation of hallucinations, and particularly auditory verbal hallucinations (AVHs). The third study (Chapter 3) created a new tool for assessing hypnagogic and hypnopompic (H&H) hallucinations, and showed that the presence of auditory H&H hallucinations, but not visual or felt-presence H&H hallucinations, was associated with a greater tendency to experience intrusions. The fourth study (Chapter 4) developed an extended model of AVHs in which rumination, as well as thought suppression, were proposed to be involved in the formation of AVHs, through their creation of intrusive thoughts. This model was tested in a healthy sample of individuals using structural equation modelling, and the proposed model was found to be a good fit to the data.

The study on rumination and AVHs highlighted that agency disruption factors are likely to be involved in leading these self-generated cognitions to be experienced as alien. A theoretical analysis (Chapter 5) was made of the mechanisms likely to be involved in this disruption of agency, involving the concept of a neurocognitive action self-monitoring system (NASS) and a breakdown in the processes leading to the illusion of conscious will.
A consideration was also given to how a Vygotskian conception of inner speech could contribute to inner speech models of AVHs. The next study (Chapter 6) then performed an empirical test of the proposal that the disruption of agency in AVHs is associated with a faulty NASS. Subliminal primes were used as a proxy for the predicted state proposed to exist in the NASS, which leads to the experience of agency. It was proposed that those prone to hallucinations would be less able to use primes to enhance their experience of agency, due to deficits in their NASS. A statistically significant trend was found for the more hallucination-prone to be less able to use subliminal primes, but this effect was only found in women. It was concluded that although this was a promising finding, the effect was too small and gender-specific to be practical to test in a clinical sample of patients with AVHs.

The proposal that AVHs result from a breakdown in the NASS, specifically a corollary discharge deficit between speech production and reception areas, has been claimed to be supported by electrophysiological event-related potential (ERP) studies. However, only a simplistic conception of inner speech has thus far been investigated in ERP studies, and the potential confounding effects of attention have not been considered. The N1 ERP component response to auditory stimuli during inner speech was studied in a sample of healthy volunteers (Chapter 7). Although dampening of the N1 response was found during all types of inner speech, as compared to a silent baseline condition, dampening was also found during a mental rotation task. It was concluded that dampening of the N1 ERP component during inner speech is due to attentional factors, and is not indicative of a corollary discharge mechanism. Finally, a theoretical analysis considers whether inner speech models of AVHs are able to satisfactorily account for the phenomenology of the experience (Chapter 8). It is concluded that subcategorisation of
auditory hallucinations may be necessary, with memory-based, inner speech-based, and ictal-based models each accounting for a subcategory of auditory hallucinations. The concept of the dynamic developmental progression of AVHs is introduced and avenues for future research in this area highlighted.
Introduction

The term ‘schizophrenia’ may well be the most feared and least understood in mental health. Given that those diagnosed with this condition have a life expectancy 20% less than the general population (Newman & Bland, 1991), and face the stigma associated with this “sacred symbol of psychiatry” (Szasz, 1979, p. 1), there is reason for trepidation. However, in the past two decades in particular, there have been some profound and wide-ranging critiques of the concept of schizophrenia, which have led clinicians and researchers to rethink how we should approach and study the experiences associated with psychotic illnesses.

Complaint-based approaches to psychopathology

An extensive scientific literature has arisen which has questioned the reliability and validity of the diagnosis of schizophrenia (e.g., Bentall, 2003, 2006; Boyle, 1990). For example, Bentall, Jackson and Pilgrim (1988) reviewed data on the symptoms, course, and outcome of psychotic disorders and concluded that the diagnosis of schizophrenia lacked predictive validity. Other studies have accumulated evidence against the assumption that psychotic experiences are symptoms of discrete diseases such as schizophrenia (Bentall & Fernyhough, 2008). For example, Johnstone et al. (1988) found that response to medication was predicted not by the diagnosis of patients (such as schizophrenia and bipolar disorder) but by the complaints they reported. As Frith (1992) notes, such studies “provide support at the biological level for the proposal that it is a more fruitful research strategy to study
patients with certain symptoms rather than patients in particular diagnostic categories" (p. 12)

A similar symptom-oriented research strategy has been strongly advocated by Bentall (2003, 2006). However, in line with the argument that experiences such as hallucinations are not necessarily ‘symptoms’ of mental illness (Romme & Escher, 1993) Bentall uses the term ‘complaint’ rather than symptom. Bentall (2006) has suggested that we should “abandon psychiatric diagnoses altogether and... instead attempt to explain the specific complaints (‘symptoms’) that patients bring to our attention” (p. 220), and that “once these complaints have each been explained... there will be no ‘schizophrenia’... left over to account for” (p. 224). Such an approach has resulted in a burgeoning literature on the cognitive mechanisms underlying specific complaints such as hallucinations (e.g., Frith, 1992; David, 2004; Seal, Aleman & McGuire, 2004; Leudar & Thomas, 2000) and delusions (e.g., Freeman, Garety, Kuipers, Fowler, & Bebbington, 2002; Garety & Freeman, 1999; Langdon, Corner, McLaren, Ward, & Coltheart, 2006).

The importance of such an approach has been underscored by resulting advances in cognitive-behavioural interventions for those distressed by such experiences (e.g., Rector & Beck, 2001; Dickerson, 2000). A series of randomised controlled trials of cognitive behavioural therapy for hallucinations and delusions have found that interventions targeted at these specific complaints can decrease symptom frequency and associated distress (e.g., Sensky et al., 2006). In line with the utility of a complaint-based approach, this thesis will focus on two specific complaints often associated with patients with a diagnosis of schizophrenia, namely persecutory delusions and auditory verbal hallucinations.
General Introduction

Persecutory delusions

Freeman and Garety (2000) define a belief as being persecutory if the individual believes that “harm is occurring, or is going to occur, to him or her” and that “the persecutor has the intention to cause harm” (p. 412). Such beliefs are more likely to be considered delusions the more they are implausible, unfounded, strongly held, not shared by others, distressing and preoccupying (Freeman, 2007). This reflects the American Psychological Association’s (DSM-IV-TR, 2000) definition of delusion as a “false belief based on incorrect inference about external reality that is firmly sustained despite what almost everyone else believes and despite what constitutes incontrovertible and obvious proof or evidence to the contrary” (p. 821). Persecutory delusions are traditionally associated with schizophrenia. Indeed, Sartorius et al. (1986) found that such delusions occur in nearly 50% of patients with a diagnosis of schizophrenia. However, persecutory delusions are also found in other conditions such as bipolar disorder (Goodwin & Jamison, 1990) and post-traumatic stress disorder (Butler, Mueser, Sprock, & Braff, 1996).

The continuity hypothesis of persecutory delusions

Many have argued that, rather than understanding persecutory delusions as discrete, categorical entities, qualitatively different to more common persecutory ideation, they should be understood as existing on a continuum with normal experiences (e.g., Van Os & Verdoux, 2003). In the realm of physical health, Rose and Barker (1978) have argued that characteristics such as blood pressure and glucose tolerance exist on a continuum in the general population, and that an essentially arbitrary line is drawn in clinical practice, above which the individual is said to belong in the categorical disease states of hypertension and diabetes respectively. Johns and van Os (2001) draw a parallel between this and psychotic
experiences, arguing that “that psychosis exists as a continuum of experiences with a
distribution in the general population” (p. 1137). Indeed, a recent systematic review of
studies assessing subclinical psychotic experiences in the general population found a
median prevalence rate of delusions of 6% and a median incidence rate of around 4% (van
Os, Linscott, Myin-Germeys, Delespaul, & Krabbendam, 2009), although data on
specifically persecutory delusions was not reported.

By this argument, persecutory delusions commonly found in patients with
schizophrenia would represent the extreme form of experiences also found in a milder form
in the general population (Freeman, 2007). Freeman illustrates this by suggesting that a
clinically relevant persecutory delusion about government attempts to kill the person could
represent an amplification of a non-clinical delusion about neighbours trying to get at the
person, which could in turn be considered as related to everyday suspicions about the
intentions of others.

The continuity assumption is consistent with findings that persecutory ideation is
not an uncommon experience in the general population. For example, Eaton, Romanoski,
Anthony, and Nestadt (1991), using the Diagnostic Interview Schedule (Robins, Helzer,
Croughan, & Ratcliff, 1981), found that 5% of healthy adults endorsed an item indicating
that they believed people had been trying to hurt them during the past month. This
experience was rated clinically relevant in a fifth of these 5% of participants. Similarly,
Olsson et al. (2002) found that, in a sample of over 1000 healthy adults, 10.6% expressed
the belief that others were spying on or following them and 6.9% believed that people were
plotting or trying to poison them. In a student sample, Freeman et al. (2005) found that 5%
of respondents thought (with at least a weekly frequency) that there was the possibility of a
conspiracy against them, and that 8% had a suspicion that someone ‘had it in for them’, and
that the same percentage of students believed someone would harm them if given the opportunity. In an influential recent review, Freeman (2007) concluded that a “conservative estimate is that 10–15% of the general population regularly experience paranoid thoughts, though such figures hide marked differences in content and severity” (p. 430). Freeman (2007) further observed that “It is also likely that the studies underestimate the true frequency of paranoid thoughts because large epidemiological studies from a psychiatric perspective are unlikely to record more plausible fleeting everyday instances of paranoid thinking” (p. 430).

Auditory verbal hallucinations

The second experience this thesis will focus on is auditory verbal hallucinations, colloquially referred to as ‘hearing voices’. Auditory verbal hallucinations (AVHs), the experience of perceiving speech without corresponding external stimulation (Stephane, Barton, & Boutros, 2001), have been documented in humankind throughout recorded history (Leudar & Thomas, 2000). Again, such experiences are traditionally associated with schizophrenia. Studies have found that between 60 and 74% of those with schizophrenia report AVHs (Wing, Cooper, & Sartorius, 1974; Slade & Bentall, 1988). Furthermore, Sartorius et al. (1986) found that the monthly prevalence of AVHs in such patients was also within this range. In contrast to persecutory delusions, a number of rich and detailed studies have been made of the occurrence of AVHs throughout history (e.g., Leudar & Thomas, 2000; Schmidt, 2002). A consideration of this plentiful history shows that not only are they not necessarily a sign of mental illness but, as with persecutory delusions, they appear to exist on a continuum stretching into the general population.
A brief history of hearing voices

In Western Europe, between around 500 and 1500 A.D., individuals who reported hearing voices or seeing visions were not typically considered mad, but regarded by their peers as having actual perceptual experiences (Kroll & Bachrach, 1982). Such experiences could be understood as communications from spiritual beings (God, the Devil, etc.), as mere imaginings, or as resulting from illness (Watkins, 1998). As late as the seventeenth century, the explanation of hallucinations as religious madness was still common (Porter, 2002). However, by the start of the eighteenth century, opinion was shifting towards a conception of hallucinations as illness. Works such as William Battie’s Treatise on Madness (1758) were formative in identifying lunacy with false perceptions such as reported conversations with angels (Schmidt, 2002). As Schmidt (2002) puts it, “Enlightenment epistemologies... demanded the disciplining of religious enthusiasm, a confinement of those ‘unguarded fancies of a man’s own brain’ within a secure domain from which reason and the state might avoid contamination” (p. 191)

In 1817 experiences such as hearing voices and seeing visions were subsumed into the first medical definition of ‘hallucination’ by Esquirol. It was during this period of history that, for the first time, medicine claimed as part of its domain experiences that had traditionally fallen under the authority of the church (James, 1995). Indeed, Schmidt (2002) has argued that medical psychiatry was created precisely in order politically to contain delusions of religious fervour. A number of events around the end of the nineteenth-century provided an impetus for considering hallucinations as non-pathological. Firstly, large-scale

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empirical investigations of hallucinatory experiences in the normal population began. In a survey of 17,000 normal participants, the Report on the Census of Hallucinations (Sidgwick et al., 1894, p. 33) asked the question ‘Have you ever, when believing yourself to be completely awake, had a vivid impression of seeing or being touched by a living being or inanimate object, or of hearing a voice: which impression, so far as you could discover, was not due to any external physical cause?’ Ten percent of respondents replied in the affirmative, though approximately half reported these experiences occurring either as they were falling asleep or waking up. In terms of the modality of the hallucinations, 56% were solely visual, 23% solely auditory, and 6% involved combined visual and auditory hallucinations.

Secondly, William James’s publication of The Varieties of Religious Experience in 1902 argued for alternative ways to understand religious experiences. James (1902, p. 35) noted that what he termed medical materialism “finishes up Saint Paul by calling his vision on the road to Damascus a discharging lesion of the occipital cortex, he being an epileptic... sniffs out Saint Teresa as an hysterical, [and] Saint Francis of Assisi as an hereditary degenerate”. James claimed such an approach was simple-minded, and argued that experiences such as hallucinations should be dealt with not by “superficial medical talk” but by an inquiry into “their fruits for life” (p. 398).

*Not a necessary signifier of mental illness*

Today, it is now generally accepted that hallucinations do not necessarily imply mental illness. The contemporary reconstruction of the nineteenth-century concept of hallucinations in the sane (de Boismont, 1860) may be traced back to the interactions of one psychiatrist, Marius Romme, with a particular patient, Patsy Hague. Romme was led to
investigate the presence of specifically auditory hallucinations (voice-hearing) in the
general population after a discussion with Hague who herself heard voices. These meetings
directly led to a television appearance by Romme requesting voice-hearers to contact him.
Of the 173 respondents who agreed to complete a questionnaire, 76 of these were not in
psychiatric care (Romme, Honig, Noorthoom, & Escher, 1992). This and later work led to
Romme being confronted by a large number of “well-balanced, healthy people, who
happened to hear voices” (Romme & Escher, 1993, p. 59).

This work by Romme and Escher (1993) provided the impetus for a series of studies
into psychosis-like experiences in the general population, similar to those at the end of the
19th century. These studies provided further evidence that otherwise healthy individuals
may experience hallucinations in the absence of the social dysfunction or distress
associated with clinical psychosis (Johns & van Os, 2001; Posey & Losch, 1983; Tien,
1991). As a result of such work, a growing worldwide movement exists today based around
the view that hallucinating does not necessarily imply mental illness (Romme & Escher,
1993). For example, in the United Kingdom this has led to the creation of a large user-
movement, the Hearing Voices Network, with parallel organisations now existing
throughout the world. Although such individuals’ experiences are predominantly auditory,
members of the Hearing Voices Network also experience hallucinations in a range of other
modalities, for example, visual and tactile (ibid). This movement has resulted in a large
number of such individuals rejecting the label of ‘patient’, and the reinvention by such
individuals, partly in partnership with psychiatrists, of the paradigm of hallucinations in the
sane, and the development of the concept of the healthy voice-hearer (ibid).

There is a gathering consensus that whether an individual experiencing voices
and/or visions receives a psychiatric diagnosis depends much on the emotional content of
General Introduction

their experience, and their emotional response to it. For example, voice-hearers’ interpretation of their voices, not just the experience of the voice-hearing per se, is an important determinant of the amount of resulting distress and social/occupational disability (Krabbendam et al., 2004). In this framework, social representations (Moscovici, 1988) of voices and visions as being coterminous with madness and insanity may act as self-fulfilling prophecies through their impact on how individuals who have these experiences interpret them. Today many individuals who experience voices and visions are able to lead productive lives, and encounter their experiences in a non-psychiatric, non-mental illness discourse. Contemporary examples of this include a prominent Professor of Mathematics who reports receiving beneficial mathematical insights from her voices (Malone, 2006) and a leading pianist who hears a voice that illuminates his playing as well as experiencing a number of anomalous visual experiences (ibid). In line with such examples, Leudar has argued that “hearing voices (and the experiences we may categorize as hallucinations) should be judged as sane or insane in terms of their consequences for life. They are not in themselves signs of madness, any more than, say, thinking and remembering” (David & Leudar, 2001, p. 256).

The continuum hypothesis of hallucinations

Aleman and Laroi (2008) have identified four key components of the assumption of continuity in hallucinatory experiences. The first is the distributional component. This refers to the assumption that hallucinations “should be present in not only subjects identified as clinical cases but also in a proportion of subjects from the general population who do not fulfil the clinical criteria of a patient” (p. 83). There is substantial evidence for this proposal. Firstly, in the recent systematic review of incidence and prevalence studies of
population rates of subclinical psychotic experiences highlighted earlier in this Introduction, van Os et al. (2009) found the median prevalence rate of hallucinations (data on specific modalities was not reported) to be 4% and a median incidence rate of around 2%. This was in-line with earlier studies that found that the annual prevalence of hallucinatory experiences in the general population (in both the visual and auditory modality) to be 4% (Johns et al., 1998). Secondly there is good evidence that specifically AVHs are a not uncommon experience in the general population. For example, Posey & Losch (1993) found brief AVHs had been experienced in wakeful situations by 71% of university students. In addition to simple AVHs such as hearing one’s name called (experienced by 36% of participants), more complex AVHs were also reported, including AVHs offering advice (experienced by 10% of participants). In non-student samples significant rates of AVHs have also been reported. For example, Verdoux et al. (1998) found that 5% of patients attending a GP surgery (who did not have a diagnosis of a psychotic disorder) reported having experienced hearing voices conversing or giving commands. Other studies have found that 2% of the general population annually experience AVHs when completely awake (Tien, 1991). Such prevalences are comparable to the prevalence of persecutory ideation identified in the previous section.

The second component of the continuum hypothesis identified by Aleman and Laroi (2008) is the phenomenological component. This stipulates that there should be phenomenological similarity between hallucinations in clinical and non-clinical samples. Consistent with this proposal, AVHs in clinical samples, aside from their increased probability of being the voice of a public figure and their tendency to cause distress, have been found to be very similar, phenomenologically, to those voices reported in non-clinical samples (Leudar et al., 1997). Thirdly, Aleman and Laroi identify the developmental
component of the continuum hypothesis. This entails that factors identified as important risk factors in clinical cases of hallucinations be associated with the prevalence of hallucinations in non-clinical samples too. Importantly, Aleman and Larøi note that “this would suggest a developmental mechanism genesis, which is applicable to both clinical and non-clinical samples” (p. 83). This also receives empirical support. For example, trauma has been identified as an important precursor to hallucinations in both clinical (Read & Argyle, 1999) and non-clinical (Morrison & Peterson, 2003) samples. In line with this, Johns and van Os (2001) note that there is “evidence that psychological mechanisms associated with psychotic symptoms also operate in non-patient samples, and further study of these could greatly contribute to our understanding of psychosis” (p. 1134). Finally, there is what Aleman and Larøi term the etiological component. This entails that clinical and non-clinical populations should “share common ground in terms of the cognitive and emotional mechanisms underlying hallucinations” (p. 83). In considering this, Aleman and Larøi conclude that a cognitive approach is “particularly pertinent and fruitful in the context of hallucinations” (p. 84). In conclusion, the available evidence appears to strongly favour the continuum hypothesis of hallucinations, and an approach to these experiences that examines cognitive mechanisms underlying hallucination-proneness in non-clinical subjects.

Ethical considerations

All studies reported in this thesis received approval from the Durham University Ethics Advisory Sub-Committee in the Department of Psychology. The studies were performed in accordance with the British Psychological Society (BPS) Code of Ethics and Conduct (BPS, 2006), the Economic and Social Research Council Research Ethics
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Framework, and the Medical Research Council Ethics and Research Guidance. Studies conducted through on-line questionnaires present a new ethical challenge, considered in the BPS guidelines on internet-mediated research (BPS, 2007). As the BPS (2007) notes, obtaining informed consent on-line is problematic as "it is both possible and indeed relatively usual for individuals to access web sites without reading instructions, explanations or terms and conditions" (p. 4). Hence, wherever possible, websites used for data collection were designed so that participants could not proceed to take part in the study until they had clicked radio buttons to indicate that, 1) they consented to take part in the study, 2) they understood that they were free to withdraw from the study at any point, and to stop answering the questionnaires, and 3) they understood that all their answers were given anonymously. When this was not possible, participants were told via text on the first page of the website, and in the invitational e-mail, that they were free to stop answering the questionnaires at any point, in which event their data would not be used in the study. It was also made clear that participation was voluntary and that all answers were given anonymously, with the exception of basic demographic data. At the end of the on-line studies participants were presented with a wide range of sources of support that they could contact about any issues that were likely to have been raised by the study. They were also given the author’s e-mail address in case of any further questions about the study’s aims, design, and results.

The electrophysiological study reported in Chapter 7 raised additional ethical issues, as it involved the placement of conducting gel in the participant’s hair, and was a lengthy process. Potential participants were given a detailed information sheet (Appendix D) before consenting. This gave information on key features of the study such as the potential risks of taking part, what participants should expect to happen to them and in what order, and what
they would be paid for participation. Participants all gave signed informed consent to take part in the study.

Chapters 2 and 6 both involved a mild degree of deception. In the study reported in Chapter 2, participants were told that they would be shown beads that were being randomly drawn from a jar. In fact, as is standard practice in this widely used task (e.g., Freeman, Pugh, & Garety, 2008), the beads were always presented to the participants in a predefined non-random order. This was done in order to provide a consistent sequence of beads and to avoid the confounding effects of different participants encountering different sequences of beads. It was considered that this deception was minimal and unlikely to lead to the participants experiencing any distress. In the study reported in Chapter 6, participants were asked to guess whether a square shown on a computer screen represented the end-point of their own action, or whether it was a result of the computer’s actions. However, in order for priming to take place, the final landing position of the square had to be predefined (in accordance with standard practice in this task), such that participants were misled about the causal efficacy of their actions. That said, the degree of deception was again considered to be mild. Participants were fully debriefed immediately after the experiment and no distress found to result.
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References


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Sartorius, N., Jablensky, A., Korten, A., Ernberg, G., Anker, M., Cooper, J. E., et al. (1986). Early manifestations and first-contact incidence of schizophrenia in different cultures. *Psychological Medicine, 16*, 909-928.


PERSECUTORY IDEATION
Chapter 1: Thought suppression and PDLBs

Chapter 1

Thought suppression and persecutory delusion-like beliefs in a non-clinical sample

ABSTRACT

Thought suppression may play a role in the formation and/or maintenance of persecutory delusions, although this possibility has not yet been empirically studied. We investigated thought suppression levels in relation to the presence of persecutory delusion-like beliefs (PDLBs), and hypothesized that only when levels of anxiety or negative affect were high would thought suppression predict levels of PDLBs. Thought suppression, anxiety, negative affect, social desirability, and persecutory ideation were assessed in a non-clinical sample (N = 184) using online questionnaires. When gender, anxiety, and negative affect were controlled, the interaction between thought suppression and anxiety predicted levels of PDLBs. Further analysis of this interaction showed that thought suppression was positively associated with PDLBs only when anxiety was high. Neither thought suppression by itself, nor the two-way interaction between negative affect and thought suppression, nor the three-way interaction between negative affect, anxiety, and thought suppression, were predictors of PDLB levels. The results are consistent with a proposed interaction between thought suppression and anxiety in the development of PDLBs. Possible causal mechanisms underlying this relation are considered further, future research in the area proposed, and potential clinical implications examined.

Chapter 1: Thought suppression and PDLBs

Introduction

Persecutory delusions (PDs) are one of the most commonly held types of delusional belief (Garety, Everitt, & Hemsley, 1988; Jorgensen & Jensen, 1994), and are found cross-culturally (Stompe et al., 1999). As Freeman, Garety, Kuipers, Fowler, and Bebbington (2002) note, possession of persecutory beliefs may have severe consequences, with their presence being a predictor of admission to hospital (Castle, Phelan, Wessely, & Murray, 1994). Successful cognitive modelling of the formation and maintenance of PDs is an important step towards the improvement of treatments for such distressing beliefs (Garety & Freeman, 1999).

Two-factor models of the formation of delusions (e.g., Davies, Coltheart, Langdon, & Breen, 2001) suggest a two-stage process with each stage in turn being associated with specific cognitive deficits/biases. Langdon, Corner, McLaren, Ward, and Coltheart (2006) suggest that the first of these stages, in the case of PDs, is the triggering of “an initial implausible thought” (p. 700), such as ‘X hates me’, which is not justified in the circumstances. Once this thought is triggered, the second stage of PD formation is the uncritical adoption of such a thought as a belief. The uncritical acceptance of implausible thoughts as delusional beliefs may be due to cognitive biases such as the jumping-to-conclusions bias often present in those with PDs (Freeman, 2007). One limitation of this model is that it does not address the multidimensional nature of PDs. Freeman (2007) has argued that such a multidimensional understanding of delusional experience is needed, allowing for the possibility that each dimension (the content of the PD, the conviction with which the PD is held, the distress caused by the PD, and the resistance to change of the PD) may be influenced by different factors.
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Affective processes undoubtedly play a key role in PDs. Freeman and colleagues’ cognitive models of PDs emphasise that they are direct reflections of emotional concerns (e.g., Freeman et al., 2002; Freeman et al., 2004; Green et al., 2006). Two specific emotional factors that appear likely to be involved in many of the dimensions of PDs are anxiety and negative affect. Negative affect has been found to be associated with PDs, and more strongly so than with other types of delusion types (Appelbaum, Robbins, & Roth, 1999). However, it is anxiety that has been proposed to be the key emotion with regard to PD formation (Freeman et al., 2002, Freeman, 2007). Extensive empirical evidence has been provided for a relation between anxiety and PDs (Freeman & Garety, 1999; Startup, Freeman, & Garety, 2007), with anxiety being associated with the persistence of PDs at a three-month follow-up (Startup et al., 2007). Furthermore, the content of paranoid ideation in non-clinical samples has been found to build upon an individual’s own interpersonal anxieties (Freeman, et al., 2005).

The way in which an individual deals with anxiety and negative affect is also likely to be an important factor in both the generation and maintenance of PDs, as well as in the distress and conviction with which they are held. In Wells and Matthews’ (1994, 1996) Self-Regulatory Executive Function (S-REF) model, vulnerability to, and maintenance of, many psychological disorders are understood as resulting from an individual’s metacognitive beliefs which provide a framework for interpreting cognitive events. In line with this proposal, Freeman and Garety (1999) used the Thought Control Questionnaire (TCQ; Wells & Davies, 1994) to examine the relation between PDs (in patients with schizophrenia) and strategies used to try and suppress or control unwanted thoughts. None of the thought-control strategies assessed by the TCQ, namely distraction, social control (asking friends if they have the same thoughts), worry (focusing on other negative
thoughts), punishment, and re-appraisal (trying to reinterpret the thought), were found to differentiate a control group consisting of patients with Generalized Anxiety Disorder from patients with PDs. Levels of anxiety were also found not to differ between the two groups. However, it was found that the presence of meta-worry (assessed by the single question ‘Do you worry that you cannot control your thoughts about the belief [the PD] as well as you would like?’) was related to the amount of delusional distress experienced by the patients. Freeman and Garety interpret this to mean that PDs become most upsetting when “the individual has worries about not being able to control his or her thoughts about the belief” (p. 59). Similarly, Freeman (2007) has noted that “Worry may keep the suspicions in mind and develop the content in a catastrophising manner” (p. 450).

Another cognitive strategy that may, paradoxically, lead to such suspicions staying in mind is attempting to suppress such thoughts. Laboratory studies have shown that the process of attempting to suppress a thought can lead to the suppressed thought rebounding and becoming chronically accessible (Clark, Winton, & Thynn, 1993; Lavy & van den Hout, 1990; Wegner, Schneider, Carter, & White, 1987). In one such study two groups of participants were asked to think about a white bear. One group suppressed thoughts of the bear, while the other group did not. It was found that the group that had suppressed thoughts of the bear reported a greater number of occurrences of thoughts of the bear in the post-suppression period, compared to the group who had not suppressed thoughts of the bear (Wegner et al., 1987). This finding of increased frequency of thoughts about the target following thought suppression is termed post-suppression rebound (Wenzlaff & Wegner, 2000). Although other laboratory groups have failed to replicate such findings (e.g., Kelly & Kahn, 1994), when longitudinal studies have been performed outside the laboratory over a period of days, post-suppression rebound for personally relevant intrusive thoughts has
been found (Borton & Casey, 2006). It has also been demonstrated that, when thought suppression occurs under cognitive load, the thought becomes more accessible than if it is not being suppressed (Wegner & Erber, 1992; Wegner, Erber & Zanakos, 1993). This effect is termed the hyperaccessibility of suppressed thoughts (Wenzlaff & Wegner, 2000).

Given such effects of thought suppression there are a number of good reasons to believe that this concept may be important within a multidimensional model of PDs. Firstly, based on a range of studies such as those showing that thought suppression is predictive of post-traumatic stress disorder symptoms following trauma (Aaron, Zaglul, & Emery, 1999) and perseverance of PTSD symptoms (Ehlers, Mayou, & Bryant, 1998), Wegner and Smart (1997) have proposed that “when thought suppression accompanies psychological disorders, it may exacerbate them and magnify their symptoms” (p. 992). This is consistent with the findings that use of thought suppression is associated with anxiety (Muris, Merckelbach, & Horselenberg, 1996; Wegner & Zanakos 1994) and psychological distress (e.g., Cheavens et al., 2005; Rosenthal, Rasmussen Hall, Palm, Batten, & Follette, 2005).

Wegner and Smart’s (1997) proposal opens up the possibility that thought suppression may play a role in the development and/or maintenance of PDs. Firstly, attempting to suppress fledgling persecutory thoughts may lead to their amplification through their resultant hyperaccessibility and through post-suppression rebound. As chronically accessible thoughts serve as filters through which we see the world (Newman Duff, & Baumeister, 1997), an individual suppressing fledgling persecutory thoughts may, through these thoughts becoming chronically hyperaccessible, become more likely to interpret events and actions as persecutory. When coupled with increased occurrences of the thought due to post-suppression rebound, the result may be an increased intensity of the
Chapter 1: Thought suppression and PDLBs

associated persecutory beliefs. Secondly, attempting to suppress fully-formed PDs may act as a maintenance factor for such beliefs, through the twin effects of hyperaccessibility and post-suppression rebound. Such a method of dealing with such thoughts may also be associated with increased distress, increased conviction, and resistance to change of the PD.

There are therefore several (potentially overlapping) reasons for expecting a relation between thought suppression and PDs. Although Freeman and Garety (1999) have already noted that attempts to suppress worry may increase the frequency of worry, the relation of thought suppression to PDs has not yet been empirically examined. The only evidence suggestive of a relation comes from the findings that a) persecutory ideation in the healthy population is related to metacognitive beliefs about the uncontrollability and dangerousness of thoughts (Larøi & van der Linden, 2005a) and b) beliefs about the uncontrollability and dangerousness of thoughts are associated with higher levels of thought suppression (Jones & Fernyhough, 2006). However, Larøi and van der Linden’s study was not replicated when anxiety levels were controlled (Garcia-Montes, Cangas, Perez-Alvarez, Hidalgo, & Gutierrez, 2005). Furthermore, Larøi and van der Linden used a measure of persecutory ideation that has been argued not to be psychometrically valid (Jones & Fernyhough, 2007).

We hence designed our study to perform the first, preliminary, investigation of the relation between persecutory ideation and thought suppression. In line with evidence that PDs exist on a continuum into the healthy population (Fenigstein, 1997; Johns & van Os, 2001; Peters, Joseph, Day, & Garety, 2004), we examined relations between persecutory delusion-like beliefs (PDLBs), thought suppression, anxiety and negative affect in a non-clinical sample. If thought suppression is associated with clinically relevant persecutory delusions, such a relationship is likely to be found in relation to PDLBs in the non-clinical population. Given Freeman’s (2007) proposal that anxiety is key to the formation of PDs,
as well as the potential role of negative affect discussed above, we proposed that thought suppression would only predict levels of PDLBs when anxiety and/or negative affect were high. This led to a number of specific hypotheses. We firstly predicted that, when anxiety and negative affect were controlled using a multiple linear regression, thought suppression levels would not explain any unique variance in PDLBs. Our next two hypotheses were that the two-way interactions between anxiety and thought suppression (Hypothesis 2), and between negative affect and thought suppression (Hypothesis 3) would be predictors of PDLBs. Our fourth hypothesis was that the three-way interaction between anxiety, negative affect, and thought suppression would be a predictor of PDLBs, with thought suppression predicting PDLB levels most strongly when both anxiety and negative affect were high. Such hypotheses meet Freeman’s (2007, p. 451) call for the “testing for interactions between variables” in PD research.

We assessed levels of PDLBs using the Persecutory Ideation Questionnaire (McKay, Langdon, & Coltheart, 2006). No evidence yet exists on the extent to which participants' responses on this instrument are related to what they believe are socially appropriate responses. In comparison, scores on other psychopathology questionnaires (such as the Launay-Slade Hallucination Scale) have been shown to have no relation to participants' tendencies to give socially appropriate responses (Young, Bentall, Slade, & Dewey, 1987; Larør & van der Linden, 2005b). The study was hence designed to examine whether individuals' levels of social desirability bias predicted their scores on the PIQ, as a means of ensuring that any relation between PIQ and thought suppression was not confounded by this variable. Our final hypothesis (Hypothesis 5) was thus that participants' levels of social desirability bias would not predict their PIQ scores.
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Method

Participants

An e-mail was sent to undergraduates enrolled on a range of different programs at a British university informing them of a website where they could take part in a study. No incentive was offered for participants to take part. As a result of this, 183 undergraduates (76 males and 107 females) participated. The mean age of participants was 18.5 years ($SD = 1.3$).

Measures

Thought suppression: The White Bear Suppression Inventory (WBSI; Wegner & Zanakos, 1994) is a frequently used research tool to measure the “conscious desire to suppress thoughts” (p. 637). However, it has been argued that the WBSI taps levels of intrusive thoughts as well as thought suppression (Muris et al., 1996; Rassin, 2003). Thus, Muris et al. created a “corrected” version of the WBSI, the WBSI$_{sup}$, which constitutes the items remaining on the original WBSI after items 2, 3, 4, 5, and 9 have been removed. The remaining 10-item scale includes items such as “I always try to put problems out of mind”. This measure has been shown to have satisfactory reliability and validity (Jones & Fernyhough, 2006; Muris et al., 1996). The WBSI$_{sup}$ was used as a measure of the conscious desire to suppress thoughts. Items are rated on a five-point Likert scale ranging from “Strongly Agree” (5) to “Strongly Disagree” (1). Total scores on the WBSI$_{sup}$ can hence range from 10 to 50.

Proneness to persecutory delusion-like beliefs: The Persecutory Ideation Questionnaire (PIQ; McKay, Langdon, & Coltheart, 2006) is a 10-item questionnaire designed to measure persecutory ideation in both clinical and non-clinical samples. Items are rated on a five-point Likert scale ranging from “Very True” (4) to “Very Untrue” (0).
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The total PIQ score can hence range from 0 to 40. This measure has been shown to have good reliability and validity (ibid).

**Negative affect:** Negative affect was assessed using the 10 items assessing negative affective states on the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988). We refer to this 10-item subscale of the PANAS as PANAS\(_N\). Participants rate the extent to which they have experienced such affective states in the past week on a five-point Likert scale ranging from “Extremely” (5) to “Very slightly or not at all” (1). Total scores on the PANAS\(_N\) can hence range from 10 to 50. This measure has been shown to have good reliability and validity (ibid).

**Anxiety:** Anxiety was assessed using the Manifest Anxiety Scale (TMAS; Taylor, 1953). This is a 50-item self-report questionnaire with response options of true or false available for each question. Total scores can range from 0 to 50, with higher scores representing greater levels of anxiety. This has been found to possess satisfactory psychometric properties (ibid).

**Social desirability:** This was assessed using the Marlowe–Crowne Social Desirability Scale (Crowne & Marlowe, 1960). This is a 33-item self-report scale which assesses participants’ tendencies to give socially appropriate responses. A series of items, such as “I never resent being asked to return a favor” are rated as true or false. Total scores can range from 0 to 33 with higher scores representing a greater tendency to give socially appropriate responses. This has been found to have satisfactory psychometric properties (ibid).
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Results

Mean scores, standard deviations, ranges and Cronbach’s alphas for all scales are presented in Table 1.1. Alphas for all scales were greater than 0.7. The descriptive statistics for the PIQ data reported here were previously published in Jones, Fernyhough, de-Wit, and Meins (2008).

Table 1.1

Descriptive statistics for variables under investigation

<table>
<thead>
<tr>
<th></th>
<th>Mean (SD)</th>
<th>Range</th>
<th>Cronbach’s alpha</th>
</tr>
</thead>
<tbody>
<tr>
<td>PIQ</td>
<td>7.21 (5.14)</td>
<td>0-24</td>
<td>.87</td>
</tr>
<tr>
<td>WBSI_{sup}</td>
<td>32.87 (6.97)</td>
<td>11-50</td>
<td>.85</td>
</tr>
<tr>
<td>TMAS</td>
<td>19.79 (10.06)</td>
<td>0-48</td>
<td>.91</td>
</tr>
<tr>
<td>PANAS_{N}</td>
<td>22.74 (8.43)</td>
<td>10-49</td>
<td>.87</td>
</tr>
<tr>
<td>Soc-Des</td>
<td>15.75 (4.83)</td>
<td>6-28</td>
<td>.73</td>
</tr>
</tbody>
</table>

PIQ = Persecutory Ideation Questionnaire, WBSI_{sup} = White Bear Suppression Inventory – Corrected, PANAS_{N} = Positive and Negative Affect Scale – negative items only, TMAS = Taylor Manifest Anxiety Scale, Soc-Des = Marlowe–Crowne Social Desirability Scale

The mean PIQ score of 7.23 was somewhat lower than the previous administration of this questionnaire to university students, which found a mean score of 9.11 (McKay et al., 2006). Bivariate zero-order correlations among the variables are presented in Table 1.2. PIQ scores correlated positively with TMAS, PANAS_{N}, and WBSI_{sup}, but negatively with social desirability.
Parametric statistical analysis was performed using a hierarchical multiple linear regression with PIQ score as the dependent variable. The assumption of independence of residuals was assured, as the Durbin-Watson value of the model was 2.00. A Kolmogorov-Smirnov test indicated that the standardized residuals did not deviate significantly from normality ($D = 0.06, p > 0.05$). Examination of a plot of standardized residuals against standardized predicted values suggested no violation of the assumption of homoscedasticity.

Table 1.2
Bivariate correlations between variables under investigation

<table>
<thead>
<tr>
<th></th>
<th>PIQ</th>
<th>WBSI$_{sup}$</th>
<th>TMAS</th>
<th>PANAS$_N$</th>
<th>Soc-Des</th>
</tr>
</thead>
<tbody>
<tr>
<td>PIQ</td>
<td>-</td>
<td>.40*</td>
<td>.55*</td>
<td>.43*</td>
<td>-.39*</td>
</tr>
<tr>
<td>WBSI$_{sup}$</td>
<td>-</td>
<td>.58*</td>
<td>.52*</td>
<td>-.44*</td>
<td></td>
</tr>
<tr>
<td>TMAS</td>
<td>-</td>
<td>-</td>
<td>.67*</td>
<td>-.51*</td>
<td></td>
</tr>
<tr>
<td>PANAS$_N$</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-.50*</td>
<td></td>
</tr>
<tr>
<td>Soc-Des</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

* $p < .001$

The variables known to be associated with persecutory ideation (TMAS and PANAS$_N$) were entered in a first step (Field, 2000). Gender was also entered in the first step as paranoid ideation is more common in young men than women (APA, 2000; Reich, 1987). Age was not entered due to the homogeneous nature of the sample, and its non-significant correlation with PIQ scores ($r = -.04$, n.s.). This model was a significant predictor of PIQ.
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scores, $R^2 = .34, F(3,179) = 30.28, p < .001$. Both TMAS, $\beta = .48, p < .001$, and gender (higher scores in males), $\beta = .18, p < .01$, were significant predictors of PIQ scores, but PANASN was not, $\beta = .14, n.s.$

Table 1.3

Results of hierarchical multiple linear regression of PIQ scores

<table>
<thead>
<tr>
<th>Variable</th>
<th>$\beta$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>.14</td>
<td>$&lt; .05$</td>
</tr>
<tr>
<td>TMAS</td>
<td>.39</td>
<td>$&lt; .001$</td>
</tr>
<tr>
<td>PANASN</td>
<td>.08</td>
<td>.42</td>
</tr>
<tr>
<td>WBSI$_{sup}$</td>
<td>.11</td>
<td>.21</td>
</tr>
<tr>
<td>WBSI$_{sup} \times$ TMAS</td>
<td>.26</td>
<td>$&lt; .01$</td>
</tr>
<tr>
<td>WBSI$_{sup} \times$ PANASN</td>
<td>-.14</td>
<td>.16</td>
</tr>
<tr>
<td>WBSI$_{sup} \times$ PANASN$\times$ TMAS</td>
<td>.01</td>
<td>.98</td>
</tr>
<tr>
<td>Soc-Des</td>
<td>-.12</td>
<td>.11</td>
</tr>
</tbody>
</table>

Note: Bold type indicates significant result.

The remaining variables under investigation, namely WBSI$_{sup}$, its two-way interaction with TMAS, its two-way interaction with PANASN, its three-way interaction with both TMAS and PANASN, and social desirability, were then entered in a second step (Field, 2005). WBSI$_{sup}$, TMAS and PANASN scores were centered prior to forming the multiplicative term as recommended by Jaccard, Turrisi, and Wan (1990) in order to remove multicollinearity from the data. This step was significant, $\Delta R^2 = .045, \Delta F(3,173) = 2.51, p$
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< .05. The overall model was also significant, $F(9,173) = 13.40, p < .001$. The results for this overall model are presented in Table 1.3.

Figure 1.1

Analysis of interaction between anxiety (TMAS) and thought suppression (WBSI<sub>sup</sub>) on persecutory ideation (PIQ).
In this model gender and TMAS remained significant predictors of PIQ, with PANAS$_N$ still not predicting PIQ scores. This model also showed WBSI$_{sup}$, its two-way interaction with PANAS$_N$, and its three-way interaction with PANAS$_N$ and TMAS, not to be significant predictors of PIQ. Social desirability was not a predictor of PIQ scores. However, the two-way interaction between WBSI$_{sup}$ and TMAS was a significant predictor of PIQ scores.

Figure 1.1 illustrates the significant two-way interaction between WBSI$_{sup}$ and TMAS. In preparing this figure, the model's regression equation was used over the range of TMAS scores, for high, average and low values of thought suppression (defined as mean score plus one standard deviation, mean score, and mean score less one standard deviation, respectively) with all other predictors at mean levels. Figure 1.1 demonstrates that thought suppression has a greater effect on PIQ scores when anxiety levels are high.

Discussion

The study reported here was designed as a preliminary investigation of the relation between thought suppression and persecutory ideation. Zero-order correlations showed that both anxiety and negative affect correlated positively with levels of PDLBs. However, regression analysis showed that only anxiety explained a significant amount of unique variance in PDLB levels with anxiety, but not negative affect, predicting PDLB levels. This supports Freeman and colleagues’ (Freeman et al., 2002, Freeman, 2007) contention that anxiety is the key emotion in PDs. Gender was also found to be a significant predictor of PDLBs, with males reporting higher PIQ levels than females. This is in line with the greater prevalence of paranoid personality disorder in males than females (APA, 2000).

Although a positive zero-order correlation was found between thought suppression and PDLBs, in line with our first hypothesis thought suppression was found not to predict any unique variance in levels of PDLBs. Our second hypothesis was also supported, with
the two-way interaction between anxiety and thought suppression being a significant predictor of PDLBs. Further analysis showed that it was only when anxiety levels were high that thought suppression had a significant effect on PDLBs. Our third and fourth hypotheses, that there would be a two-way interaction between negative affect and thought suppression, and a three-way interaction between negative affect, anxiety and thought suppression, were not supported. Finally, in line with our fifth hypothesis, participants’ levels of social desirability bias did predict their PDLB levels.

The present study was thus able for the first time to establish a relation between thought suppression and PDLBs. That said, only a relatively small amount of variance (4.5%) in persecutory ideation was accounted for by the interaction between thought suppression and anxiety. The statistical significance of this variance component presumably results from the relatively large sample size employed. If thought suppression does play a role in PDLBs, it clearly must do so in conjunction with a range of other factors. Some possible relevant factors identified by Freeman (2007) include anomalous experiences (such as hallucinations, which may in turn be related to illegal drug use), interpersonal sensitivity, reasoning biases (e.g., the jumping to conclusions bias), isolation and trauma. Thought suppression is hence likely to have a role only as part of a multi-factorial explanation of PDs.

Some caveats need to be made about these findings. Firstly, this was a correlational study, which could not determine whether thought suppression, via its putative twin effects of post-suppression rebound and hyperaccessibility of the thought, plays a role in the development of PDs, in their maintenance, or both. Secondly, although it is possible that our use of online questionnaires may have led to unrepresentative patterns of responding, studies have shown that data collected by such questionnaires (including the measure of
persecutory ideation used in this study) are consistent with those collected by more traditional methods (e.g., Jones et al., 2008).

The present findings suggest a number of avenues for future research. Firstly, the models of PDs discussed in the Introduction suggest that, in addition to an initial implausible thought, cognitive biases such as the jumping-to-conclusions (JTC) bias are also needed for PDs to form. It would hence be profitable to replicate our study whilst concurrently assessing levels of the JTC bias. That said, no relation between the JTC bias and paranoid thinking was found in a recent study with non-clinical participants (Freeman et al., 2005). This may be due to biases in reasoning being more subtle outside of acute delusional states, and/or the small size of the sample (N = 30). Alternatively, there may be no direct relation between levels of JTC bias and PDLBs; instead, JTC bias may only predict levels of PDLBs through its interaction with other variables. On the basis of the present findings, we would expect two-way interactions between the JTC bias and both anxiety and thought suppression in predicting PDLBs. Specifically, the JTC bias should only predict PDLBs when anxiety is high, and thought suppression should only predict PDLBs when the JTC bias is high. In terms of the three-way interaction, thought suppression should have its strongest predictive value in relation to PDLBs when both anxiety and JTC bias levels are high.

It is also possible that the relation between thought suppression and PDLBs may be due to other mechanisms than those discussed above. One possibility is that thought suppression is associated with PDLBs due to its potential to lead to defensive projection, defined by Newman et al. (1997) as "the act of perceiving in other people those characteristics that one wishes to deny in oneself" (p. 980). It has been empirically demonstrated that suppressing one's negative traits leads to the perception of increased
levels of such traits in other people (Newman et al., 1997). This finding suggests that a contributing factor to the generation of the initial implausible or unjustified thought with persecutory content, highlighted in Davies et al.'s (2001) two-factor model of PDs discussed above, might be suppression of ego-dystonic thoughts (negative thoughts about the self that are threatening to one's self-image). Specifically, suppression of an ego-dystonic thought (e.g., "I am not trustworthy"), resulting in that thought becoming hyperaccessible, could in turn predispose an individual to perceive the associated trait in other people's behaviour, resulting in thoughts with a persecutory flavour. Such a hypothesis could be tested through investigating whether suppressing thoughts about specific negative traits in the self leads to increased attributions of persecutory intent to ambiguous behaviours.

The present findings will also need to be replicated in a clinical population. Such a study could attempt to establish whether thought suppression plays a causal role in PD formation. This could be done by examining levels of thought suppression in patients with PDs and patients with generalised anxiety disorder. If thought suppression plays a causal role in PD formation, we would expect patients with PDs to have higher thought suppression levels than controls matched for anxiety levels (when controlling for the JTC bias). Another approach would be to examine longitudinally these variables in individuals at high risk of developing PDs, with a view to determining whether the proposed causal factors in PD formation are present before the onset of the dysfunctional beliefs.

In conclusion, the present study provided preliminary evidence that thought suppression is associated with PDLBs. Future research is needed to establish whether thought suppression plays a role in the development and/or maintenance of clinically relevant PDs. Any such findings would have clear implications for treatment. One
therapeutic possibility might be for patients to be encouraged openly to express the
thoughts they have been suppressing. In light of Sparrow and Wegner's (2006) findings
that intrusive thoughts may be deactivated by merely expressing them, this would be
expected to have beneficial effects for the patients. Such studies should help to test the
validity of our proposal that there is a role for thought suppression in cognitive models of
PD formation.
Chapter 1: Thought suppression and PDLBs

References


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Chapter 2

Anxiety, anomalous experiences, jumping to conclusions and persecutory delusion-like beliefs

ABSTRACT

Two-factor models of persecutory delusions suggest that they are formed in two stages. The first involves the generation of an initial implausible thought that is subsequently uncritically accepted as a belief through the operation of second-stage factors, such as the jumping to conclusions (JTC) bias. Drawing on a healthy non-clinical sample (N = 165), the present study tested the hypothesis that the JTC bias would not be an independent predictor of persecutory delusion-like beliefs (PDLBs), but that it would predict such experiences through its interaction with first-stage factors (social anxiety, anomalous experiences, thought suppression, and social rank). In support of this hypothesis, no main effect of the JTC bias was found on PDLB levels. The only significant interaction between the JTC bias and a first-stage factor was with social rank. Social rank had a greater effect on levels of PDLBs when the JTC bias was present. It was concluded that, although these results offered some support for two-factor models of PD formation, the JTC bias was not a necessary condition for PD formation. Avenues for future research and clinical implications are discussed.
Chapter 2: Jumping to conclusions and persecutory ideation

Introduction

Two-factor models of the formation of delusions (e.g., Davies, Coltheart, Langdon, & Breen, 2001; Langdon & Coltheart, 2000; see Chapter 1) suggest a two-stage process of persecutory delusion (PD) formation with each stage in turn associated with specific cognitive deficits and biases. The first stage, in the case of PDs, is the triggering of an initial implausible thought which, once triggered, is uncritically adopted as a belief as a result of the operation of second-stage factors such as the jumping to conclusions (JTC) bias. This bias has been frequently documented in those with delusions (Garety & Freeman, 1999) and, in the only study to look specifically at PDs in relation to the JTC bias, Startup, Freeman, and Garety (2008) found that 50% of patients with schizophrenia with PDs demonstrated a JTC bias, compared to only 10% of healthy, non-clinical controls.

A number of factors are likely to play a role in the first stage of PD formation. One important candidate factor which may seed initial persecutory ideas is anxiety (Freeman, 2007; Jones & Fernyhough, 2008; see Chapter 1). Furthermore, as paranoid thoughts build upon common interpersonal anxieties and worries (Freeman et al., 2003), social anxiety in particular is likely to play a key role in the generation of initial implausible thoughts. Secondly, Freeman (2007) suggests that social rank may be an important element in persecutory ideation formation. For example, greater levels of paranoid ideation in healthy individuals have been found to be associated with lower social rank (Freeman et al., 2005). This study found that suspiciousness was also associated with low social rank. It hence seems plausible that low social rank may make it more likely for individuals to be suspicious and to generate initial implausible thoughts.

A third factor that may be involved in the generation of initial implausible ideas is the presence of anomalous experiences such as heightened perceptions of colour, increased
Chapter 2: Jumping to conclusions and persecutory ideation

salience of events, hypersensitivity to sound, and hallucinatory experiences. This proposal stems from the work of Maher (1974), who proposed that delusional experiences result from rational interpretations of unusual experiences. The evidence linking anomalous experiences and persecutory ideation is limited. However, in a series of studies using a methodology involving participants encountering avatars in a virtual reality environment, Freeman and colleagues (Freeman et al., 2003, 2005) found that a predisposition to anomalous experiences made persecutory ideation more likely. In these studies participants wore helmets, immersing them in a virtual reality environment. Participants were presented with a scenario in which other individuals ('avatars') were presented to them as other passengers on a underground train. Although the avatars were programmed to act in a neutral manner, around a third of participants experienced the avatars' behaviour as persecutory. Individuals were more likely to experience persecutory ideation, as opposed to simply social anxiety, if they had high levels of hallucination-proneness (as assessed by self-report questionnaire). Finally, a fourth factor implicated in the creation of initial implausible ideas is thought suppression, which may make thoughts hyperaccessible and amplify suppressed thoughts (Jones & Fernyhough, 2008; see Chapter 1).

The two-factor model of PD formation predicts that such initial implausible ideas are likely to be transformed into beliefs of a delusional intensity when they are accompanied by biases in reasoning, such as the JTC bias. However, interactions between factors associated with the first and second stages of the two-factor model have not yet been experimentally investigated. The present study examined the hypothesis that biases in reasoning, specifically the JTC bias, would not in themselves predict levels of PDLBs, unless there were also high accompanying levels of the factors proposed to create the initial implausible thought. Specific hypotheses (see below) involved two-way interactions among
potential first-stage factors highlighted above and the second-stage factor of the JTC bias. Support for these hypotheses would constitute strong evidence in favour of a two-factor model. Conversely, evidence of the JTC bias independently predicting PDLBs would run counter to two-factor models.

The study set out to test the following hypotheses. Firstly, it was predicted that the JTC bias would not be an independent predictor of levels of PDLBs (Hypothesis 1). Secondly, it was predicted that there would be two-way interactions between the JTC bias and social anxiety (Hypothesis 2a), social rank (Hypothesis 2b), anomalous experiences (Hypothesis 2c), and thought suppression (Hypothesis 2d). It was predicted that all these first-stage factors would have a stronger effect on levels of PDLBs when the JTC bias was present, as opposed to absent. The study also took the novel step of utilising two separate measures of persecutory ideation in order to attempt to achieve convergent validity for any conclusions.

Method

Participants

Undergraduate and postgraduate students (N = 165, 89 women) at a United Kingdom university, with a mean age of 20.2 years (SD = 1.3, range = 18-25) participated in the study. Participants were recruited through e-mail invitation. There was no financial incentive to participate. Answers were given anonymously, with only age and gender being requested. Response rates were approximately 20%, and comparable to previous studies employing on-line questionnaires of similar length (e.g., Kaplowitz, Hadlock & Levine, 2004). On-line questionnaires have been shown to be a reliable method of data collection (Jones, Fernyhough, de-Wit, & Meins, 2008; see Appendix A).
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Measures

Jumping to conclusions bias: This was assessed using an on-line version of the probabilistic reasoning task known as the ‘beads task’; a methodology based on earlier studies of probabilistic reasoning (e.g., Phillips & Edwards, 1966). Participants are asked to obtain as many pieces of evidence (in the form of coloured beads) as they need in order to make a decision (about which of two jars the beads came from) with certainty. The number of beads requested before making a decision is termed the number of ‘draws to decision’. Participants who take only a few draws to decision are seen to be making decisions based on only a small amount of the available evidence, and are hence said to demonstrate a jumping to conclusions bias. Participants were first given instructions, on-line. They were next shown pictures of two jars, one with 85 black beads and 15 yellow beads, the other with 15 black beads and 85 yellow beads. The participant was then shown a jar (with a blue cloth around it) and told it was one of the two jars they had just seen. They next had to use the mouse to click on a button on screen which said “Show me a bead”. This bead was then shown coming out of the jar, and a picture of this bead was shown on the bottom of the screen. Participants were told in the initial instructions that the bead was actually placed back in the jar after it was drawn, and that the picture on screen was to help them recall what beads had been seen. The participant could then either click “Show me another bead” or “I am completely certain which jar it is”. If the participant chose to see another bead, this was shown on the screen, alongside the previous bead. This on-screen presentation of already-selected beads was intended to reduce working memory demand aspects of the task. If the participant clicked “I am completely certain which jar it is”, then they were taken to a new screen where they were shown pictures of the ‘mostly black’ and ‘mostly yellow’ jars, and invited to click on the picture of the jar they thought the beads had come
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The order of the beads drawn was fixed and was always BBBBBBYBBBBYYBBBBBBBBYB (B refers to a black bead and Y to a yellow bead). This order was previously employed by McKay, Langdon, and Coltheart (2006a).

Persecutory ideation: Persecutory ideation was assessed using the Persecutory Ideation Questionnaire (PIQ; McKay, Langdon, & Coltheart, 2006b). This is a 10-item questionnaire designed to measure persecutory ideation in both clinical and non-clinical samples. Items are rated on a five-point Likert scale ranging from “Very True” (4) to “Very Untrue” (0). This measure has been shown to have good reliability and validity (McKay et al., 2006b). A second measure of persecutory ideation was also employed to enable convergent validity to be obtained for any conclusions. This was the Persecution and Deservedness Scale (PADS: Melo, Corcoran, Shryane, & Bentall, in press). This is a 10-item self-report measure of persecutory ideation, which also assesses the perceived deservedness of the experience (although the latter is not reported on here). Items are rated on a five-point Likert scale ranging from “Certainly false” (0) to “Certainly true” (4). This has been found to have satisfactory psychometric properties (ibid).

Social Anxiety: This was assessed by the Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987), a 24-item self-report measure in which participants first rate the amount of fear a social situation causes them. This is scored on a four-point Likert scale ranging from “none” (0) to “severe” (3). Participants then rate how much they avoid such situations, and respond on a 4-point Likert scale ranging from “never” (0) to “usually” (3). An overall total score is calculated by summing the total fear and total avoidance scores. The LSAS has been shown to be a valid and reliable tool (Baker, Heinrichs, Kim, & Hofmann, 2002).

Anomalous experiences. This was assessed using the Unusual Experiences subscale of the short form version of the Oxford and Liverpool Inventory of Feelings and
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Experiences (O-LIFE: Mason, Linney, & Claridge, 2005). This is a 15-item self-report measure, in which participants respond ‘Yes’ (1) or ‘No’ (0) to items such as “Have you sometimes sensed an evil presence around you, even though you could not see it?” This has been shown to have satisfactory psychometric properties (ibid).

Thought suppression: This was assessed using Muris et al.’s (1996) 10-item corrected version of the White Bear Suppression Inventory (WBSI; Wegner & Zanakos, 1994), the WBSI_{sup} (Chapter 1). This measure has been shown to have satisfactory reliability and validity (Jones & Fernyhough, 2006; Muris et al., 1996). The WBSI_{sup} was used as a measure of the conscious desire to suppress thoughts. Items are rated on a five-point Likert scale ranging from “Strongly Agree” (5) to “Strongly Disagree” (1). Total scores on the WBSI_{sup} can hence range from 10 to 50.

Social Rank: This was assessed using the Social Comparison Scale (Allan & Gilbert, 1995), an 11-item scale in which participants rate themselves on a 10-point Likert scale anchored in two bipolar constructs such as left out–accepted and unattractive–more attractive. For example, on the first item participants are asked to rate on a ten-point scale anchored in ‘inferior’ (1) and ‘superior’ (10) how they typically feel in relation to others. A higher score represents a higher perceived social rank. The Social Comparison Scale has been shown to have satisfactory psychometric properties (ibid).

Results

Initial analyses

Descriptive statistics are presented in Table 2.1. Due to a coding error, data for item 11 on the Social Comparison Scale was not recorded. However, the resulting 10-item scale still had satisfactory internal reliability (alpha = .90). Kolmogorov-Smirnov tests indicated that PIQ and PADS scores were non-normally distributed, \( D(165) = .15, p < .001 \), and
Non-parametric correlational analyses were therefore utilised and relations between the continuous variables under investigation are presented in Table 2.2. Notably, a significant negative correlation was found between both PIQ and PADS scores and social rank. Additionally, the positive correlation between thought suppression and persecutory ideation found by previous research (Jones & Fernyhough, 2008; see Chapter 1) was replicated. Jones and Fernyhough (2008) previously found a positive relation between anxiety and thought suppression; the results in Table 2.2 show that thought suppression is also related to specifically social anxiety (see also Chapter 4).

Table 2.1
Descriptive statistics

<table>
<thead>
<tr>
<th></th>
<th>Mean (SD)</th>
<th>Range</th>
<th>Cronbach’s alpha</th>
</tr>
</thead>
<tbody>
<tr>
<td>PIQ</td>
<td>7.13 (5.08)</td>
<td>0-23</td>
<td>.82</td>
</tr>
<tr>
<td>PADS</td>
<td>13.35 (7.81)</td>
<td>0-33</td>
<td>.83</td>
</tr>
<tr>
<td>Beads</td>
<td>9.18 (6.77)</td>
<td>2-20</td>
<td>n/a</td>
</tr>
<tr>
<td>SocAnx</td>
<td>42.13 (20.45)</td>
<td>6-103</td>
<td>.80</td>
</tr>
<tr>
<td>O-LIFEUE</td>
<td>3.80 (2.70)</td>
<td>0-12</td>
<td>.74</td>
</tr>
<tr>
<td>Social Rank</td>
<td>57.67 (13.71)</td>
<td>25-91</td>
<td>.90</td>
</tr>
<tr>
<td>WBSI\text{sup}</td>
<td>34.40 (7.86)</td>
<td>13-50</td>
<td>.80</td>
</tr>
</tbody>
</table>

Note. PIQ = Persecutory Ideation Questionnaire, PADS = Persecution and Deservedness Questionnaire, Beads = Draws to Decision on Beads Task, SocAnx = Liebowitz Social Anxiety Scale, O-LIFE\text{UE} = Unusual Experiences subscale of the Oxford and Liverpool Inventory of Feelings and Experiences, Social Rank = Social Comparison Scale, and WBSI\text{sup} = White Bear Suppression Inventory – Corrected.
### Table 2.2

Non-parametric bivariate correlational analysis between continuous variables

<table>
<thead>
<tr>
<th></th>
<th>PIQ</th>
<th>PADS</th>
<th>SocAnx</th>
<th>O-LIFE\textsubscript{UE}</th>
<th>Social rank</th>
<th>WBSI\textsubscript{sup}</th>
</tr>
</thead>
<tbody>
<tr>
<td>PIQ</td>
<td>1</td>
<td>.65*</td>
<td>.31*</td>
<td>.24*</td>
<td>-.32*</td>
<td>.30*</td>
</tr>
<tr>
<td>PADS</td>
<td>-</td>
<td>1</td>
<td>.40*</td>
<td>.24*</td>
<td>-.35*</td>
<td>.41*</td>
</tr>
<tr>
<td>SocAnx</td>
<td>-</td>
<td>-</td>
<td>1</td>
<td>.13</td>
<td>-.54*</td>
<td>.30*</td>
</tr>
<tr>
<td>O-LIFE\textsubscript{UE}</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>1</td>
<td>.04</td>
<td>.27*</td>
</tr>
<tr>
<td>Social rank</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>1</td>
<td>-.26*</td>
</tr>
<tr>
<td>WBSI\textsubscript{sup}</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>1</td>
</tr>
</tbody>
</table>

* p < .003

### Table 2.3

Non-parametric bivariate correlational analysis between interval level measures for JTC-(JTC+) group

<table>
<thead>
<tr>
<th></th>
<th>PIQ</th>
<th>PADS</th>
<th>SocAnx</th>
<th>O-LIFE\textsubscript{UE}</th>
<th>Social rank</th>
<th>WBSI\textsubscript{sup}</th>
</tr>
</thead>
<tbody>
<tr>
<td>PIQ</td>
<td>1</td>
<td>.66* (.52*)</td>
<td>.24 (.47*)</td>
<td>.23 (.27)</td>
<td>-.25 (-.37)</td>
<td>.24 (.36)</td>
</tr>
<tr>
<td>PADS</td>
<td>-</td>
<td>1</td>
<td>.34* (.45)</td>
<td>.33* (-.09)</td>
<td>-.22 (-.64*)</td>
<td>.41* (.41)</td>
</tr>
<tr>
<td>SocAnx</td>
<td>-</td>
<td>-</td>
<td>1</td>
<td>.13 (.15)</td>
<td>-.46* (-.65*)</td>
<td>.28* (.28)</td>
</tr>
<tr>
<td>O-LIFE\textsubscript{UE}</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>1</td>
<td>.04 (.18)</td>
<td>.26 (.29)</td>
</tr>
<tr>
<td>Social rank</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>1</td>
<td>-.30* (-.13)</td>
</tr>
<tr>
<td>WBSI\textsubscript{sup}</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>1</td>
</tr>
</tbody>
</table>

* p < .003
Distribution of draws to decision

It has previously been suggested that the draws to decision (DTD) variable on the beads task should not be treated as an interval scale, as the beads differ in informational value (Freeman, Pugh, & Garety, 2008). This has led such data typically being dichotomised (i.e., JTC bias present, JTC bias absent) in order to identify extreme responding styles. The distribution of draws to decision in the present study is shown in Figure 2.1. The clustering of responses observed in Figure 2.1 suggested a trimodal distribution of responses. Although no participants made their decision after seeing just one draw, a distinct group of early responders making a decision after 2 or 3 draws was discernable ($n = 39, 24\%$ of sample). Although, Freeman et al. have suggested that the cut-off for a JTC bias be set at 2 beads or less, others have used a criterion of 3 beads (e.g., van Dael et al., 2005). For the purposes of the present study, the presence of a JTC bias was defined as those making a decision after 3 or fewer beads. This group ($n = 39$) is henceforth referred to as JTC+.

A second cluster of individuals chose after 5, 6, or 7 beads ($n = 61, 37\%$ of sample). At the other end of the spectrum of responses, a clearly distinguishable group of individuals ($n = 39, 24\%$ of total sample) took the full 20 possible draws to decide. Previous research using face-to-face administrations of the beads task shows that taking 10 or more draws is only usually observed in 1\% of participants (e.g., Dudley, John, Young, & Over, 1997). It may be that the absence of face-to-face interaction with a real experimenter, and presumably therefore a reduced social pressure to make a decision, enabled the participants to feel freer to select more beads in this on-line format. This point will be returned to in the Discussion. For the purposes of the present study, and to avoid unnecessary reductions to
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the power of the analyses, all those taking more than 3 beads to reach a decision were classed as not showing a JTC bias. This group ($n = 126$) is henceforth referred to as JTC-.

The mean DTD for the total sample ($N = 165$) was 9.18 (6.77). This was significantly higher than previous face-to-face administrations of the 85:15 ratio Beads Task in non-clinical samples. For example, Dudley et al. (1997) found a mean DTD of 4.9 (SD = 1.9) beads. As noted above, the higher mean DTD score in the present sample was due to a significant number of participants requesting the full number of available beads. The mean (SD) DTD in the JTC+ group ($n = 39$) was 2.72 (.46), and the mean (SD) DTD in the JTC- group ($n = 126$) was 11.17 (6.55).

The relations between the continuous variables in the JTC+ and JTC- groups are presented in Table 2.3. Differing patterns of correlations for the two JTC bias groups were followed up by the testing of interactions between the JTC bias and the continuous variables. This was done through multiple linear regression analyses.

Multiple linear regression analyses

Multiple linear regression (MLR) analyses were used to analyse the data. Separate MLRs were conducted with PIQ and PADS as the respective dependent variables. Age and gender were entered in a first step to control for the potential influence of these variables. The continuous variables of social anxiety, social rank, anomalous experiences and thought suppression were entered in a second step, in addition to JTC group which was coded as a dummy variable (JTC+ = 1, JTC- = 0). In order to examine interaction effects between first- and second-stage factors, 2-way interactions between JTC group and social anxiety, social rank, anomalous experiences, and thought suppression were also entered in this step.
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Figure 2.1

Distribution of draws to decision for participants (N = 165)
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Beads group, social anxiety, social rank, anomalous experiences, and thought suppression scores were all mean-centered in order to remove multicollinearity from the data, as recommended by Jaccard, Turrisi, and Wan (1990). This initial analysis showed that age and gender were not significant predictors of PIQ or PADS scores, and hence the analyses were repeated with these variables excluded, a process recommended by Tabachnick and Fidell (2007) in order to maximize the power of analyses.

For the MLR with PADS score as the DV, a Kolmogorov-Smirnov test indicated the residuals were normally distributed, $D(165) = .04$, n.s. However, in order to meet the assumption of normally distributed residuals for the MLR with PIQ score as the DV, a square root transformation had to be applied. After this transformation a Kolmogorov-Smirnov test indicated that the residuals were normally distributed, $D(165) = .05$, n.s.

Results of the MLRs are presented in Table 2.4.

For both MLRs, O-LIFEUE, social rank, and WBSI$_{sup}$ were all independent predictors of levels of PDLBs. However, JTC group and social anxiety were not. The 2-way interaction between social rank and JTC group was significant in predicting PADS scores, although only a trend towards significance was found for the equivalent interaction when predicting PIQ scores. No other 2-way interactions were significant predictors of either PADS or PIQ scores. Figure 2.2 illustrates the significant 2-way interaction between JTC group and social rank in predicting PADS scores. In preparing this figure, the model’s regression equation was used for both JTC groups (JTC+, JTC-) over the observed range of centered social rank scores with all other predictors at mean levels. Figure 2.2 demonstrates that social rank has a greater effect on PADS scores in the JTC+ group.
Table 2.4

Results of multiple linear regressions

<table>
<thead>
<tr>
<th>Variable</th>
<th>PIQ [Sqrt]</th>
<th>PADS</th>
</tr>
</thead>
<tbody>
<tr>
<td>β, sig</td>
<td>β, sig</td>
<td></td>
</tr>
<tr>
<td>$R^2$ = .23</td>
<td>$R^2$ = .36</td>
<td></td>
</tr>
<tr>
<td>$F(9, 155) = 5.09, p &lt; .001$</td>
<td>$F(9, 155) = 9.49, p &lt; .001$</td>
<td></td>
</tr>
<tr>
<td>SocAnx</td>
<td>.12, $p = .18$</td>
<td>.16, $p = .05$</td>
</tr>
<tr>
<td>O-LIFE$_{UE}$</td>
<td>.17, $p &lt; .05$</td>
<td>.15, $p &lt; .05$</td>
</tr>
<tr>
<td>Social rank</td>
<td>-.20, $p &lt; .05$</td>
<td>-.20, $p &lt; .05$</td>
</tr>
<tr>
<td>WBSI$_{sup}$</td>
<td>.20, $p &lt; .01$</td>
<td>.30, $p &lt; .001$</td>
</tr>
<tr>
<td>JTC Group</td>
<td>-.02, $p = .84$</td>
<td>-.08, $p = .25$</td>
</tr>
<tr>
<td>JTC Group * SocAnx</td>
<td>.06, $p = .55$</td>
<td>.13, $p = .14$</td>
</tr>
<tr>
<td>JTC Group * O-LIFE$_{UE}$</td>
<td>-.04, $p = .57$</td>
<td>.10, $p = .16$</td>
</tr>
<tr>
<td>JTC Group * Social rank</td>
<td>.18, $p = .08$</td>
<td>.31, $p &lt; .001$</td>
</tr>
<tr>
<td>JTC Group * WBSI$_{sup}$</td>
<td>-.04, $p = .64$</td>
<td>-.06, $p = .44$</td>
</tr>
</tbody>
</table>

Note. Bold font indicates significant result, italics indicate a trend toward significance.
The present study aimed to test a range of predictions stemming from two-stage models of PD formation. Bivariate correlational analyses found higher levels of social anxiety, anomalous experiences and thought suppression were all associated with greater levels of PDLBs. Lower levels of social rank were also associated with greater levels of PDLBs. However, multiple linear regression analyses showed that although anomalous experiences, social rank, and thought suppression were significant predictors of levels of PDLBs, the interaction effect between social rank and JTC group further added to the prediction of PADS scores.
Chapter 2: Jumping to conclusions and persecutory ideation

PDLBs, social anxiety was not. The finding that thought suppression was a significant predictor of levels of persecutory ideation was surprising as previous studies (Jones & Fernyhough, 2008; see Chapter 1) found thought suppression not to be an independent predictor of levels of PDLBs.

In terms of specific hypotheses, the first hypothesis, that there would be no main effect of JTC bias on levels of PDLBs, was supported. Hypotheses 2a, 2c, and 2d, that there would be 2-way interactions between the JTC bias and social anxiety, anomalous experiences and thought suppression respectively, were not supported. In contrast, support was found for Hypothesis 2b, that there would be a 2-way interaction between the JTC bias and social rank. In line with the direction of this hypothesis, analysis of this interaction showed that social rank had a greater effect in the group where the JTC was present. In those with a JTC bias, lower social rank was associated with greater levels of PDLBs.

In summary, these findings are broadly in line with two-factor models of PD formation which propose that, in addition to cognitive biases such as the JTC bias, factors causing an initial implausible thought are required. That said, the only factor found to interact with the JTC bias was social rank. This could be taken to indicate that social rank is the only factor of those investigated to create the type of thoughts which, when combined with a JTC bias, can lead to persecutory ideation. There is now the need for more detailed studies of the role of social rank in persecutory ideation, what the experiences associated with low social rank are, and how they can seed initial implausible thoughts. It is plausible that lower social rank leads to initial implausible ideas due to the greater social threat anticipation associated with such a status. It has already been found that patients with schizophrenia with PDs show greater threat anticipation than controls (Corcoran et al., 2006). It would hence be profitable to examine the relation between social rank and minute-
to-minute social cognitions. Specifically it would be interesting to assess whether those low in social rank have greater numbers of thoughts about other’s intentions towards them, and whether these tend to be more negative than controls. One methodology that is particularly suited to studying this is the experience sampling method (ESM). In ESM participants wear a watch that beeps a fixed number of times each day, at random intervals. At these beeps, participants are required to report their current affective states and cognitions when this beep occurs. This methodology has already been employed with patients with schizophrenia and PDs, finding that they had more unstable levels of self-esteem than controls (Thewissen, Bentall, Lecomte, van Os, & Myin-Germeys, 2008).

The finding that anomalous experiences, social rank, and thought suppression were all independent predictors of levels of PDLBs could be taken to indicate that such factors are a sufficient cause of PDLBs, without the need for accompanying cognitive biases. A comparable line of reasoning would also suggest that the presence of the JTC bias is unlikely to be a sufficient cause of PDLBs. Thus, the present findings are consistent with a view that there are multiple routes to PDLBs, one operating directly through anomalous experiences, social rank, and thought suppression, without necessitating any cognitive biases, and another in which PDLBs arise through a two-factor process in which a JTC bias operates on initial implausible ideas created by social rank.

A number of limitations of the present study need to be acknowledged. Firstly, as this was a correlational study, neither causation, nor direction of potential relations can be established. Secondly, no previous published studies have reported results of administering the beads task in an on-line format, rather than face-to-face. Although the on-line method has the advantage of being able to administer the task to large numbers of participants cheaply and efficiently, hence conferring greater statistical power, it also has potential
Chapter 2: Jumping to conclusions and persecutory ideation

limitations. It is firstly prudent to consider the possibility that on-line administration may have affected patterns of responding. One way in which the on-line administration of the beads task may have affected the results is through the possibility (which we cannot completely eliminate) that participants may have failed to understand the written instructions, or have skipped over these, paying them only cursory attention. Such a possibility could be reduced in future on-line studies by displaying the instructions for a fixed minimum interval, and/or only letting participants proceed to the study proper after correctly answering on-line questions about how the study will work and what they are to do. A second way in which on-line administration may have affected the results is due to the anonymous nature of the task. The finding that a significant number of individuals took more than ten beads to reach a decision in this on-line version of the task raises an interesting question about the role of social pressures in choice-making on the beads task. One obvious difference between the online and face-to-face administration formats is that the former includes little social pressure to produce an answer. It could be argued that the on-line version actually assesses 'off-line' decision-making abilities, which are unlike those required in real-time, real-world situations. This may be addressed in future studies, as Fernyhough, Jones, Whittle, and Waterhouse (2008) have suggested, by introducing an element of a social interactional context to on-line tasks, such as a virtual 'experimenter' asking for responses. Such a procedure might potentially result in a greater load on online processing. However, ultimately, this again highlights the need to replicate these findings in a face-to-face administration format.

In conclusion, this study offers some evidence consistent with a two-factor model of PD formation. Evidence was found that social rank, a possible source of initial implausible ideas, had a stronger effect on levels of PDLBs when the JTC bias was also present.
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However, the JTC bias appeared to be neither a sufficient nor a necessary cause of PDLBs, as anomalous experiences, social rank and thought suppression (but not the presence of the JTC bias) all independently predicted levels of PDLBs. These findings would be consistent with a one-factor model, in which single factors (anomalous experiences, social rank and thought suppression) are sufficient to lead to PDLBs, without the necessary presence of reasoning biases.

The present findings therefore suggest the need for further research into the impact of the JTC bias on persecutory ideation. For example, PDs have been argued to be a multidimensional phenomenon (Freeman, 2007) with dimensions such as distress and conviction. Given that the JTC bias has been proposed to be associated with the uncritical acceptance of initial implausible ideas, it would be interesting to examine whether the JTC bias impacts specifically upon PD conviction. It is also important that the findings of the present study be replicated in a clinical population. Direct tests of the two-factor model would presumably be quite feasible in such clinical groups. For example, one could compare levels of anxiety and the presence of the JTC bias in patients with persecutory delusions, and patients with generalised anxiety disorder (but no persecutory ideation). The two-factor model would predict that although levels of anxiety may not differ between these two groups, greater levels of the JTC bias would be found in the patients with persecutory ideation. Such studies would offer greater insight into the processes behind PDs, which should be extremely valuable to allowing further development of cognitive behavioural therapies for individuals with these frequently distressing belief systems.
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References


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66
HALLUCINATIONS AND INTRUSIONS
FOREWORD TO CHAPTERS 3 AND 4

The studies reported in Chapters 1 and 2 examined the potential of thought suppression to contribute to the formation of persecutory delusion-like beliefs. It was noted that this relation was thought to pertain, in part, due to the potential of thought suppression to lead to intrusive thoughts. Chapter 1 found evidence that thought suppression in those with high levels of anxiety may contribute to the formation or maintenance of persecutory delusion-like beliefs. Chapter 2 went beyond this to examine the relation between factors thought to cause the initial implausible thought (first-stage factors), and factors which lead to the uncritical acceptance of such a thought as a belief, specifically the jumping to conclusions bias.

The next two chapters examine how intrusive thoughts, which thought suppression may contribute to, may form part of the cognitive processes that lead to hallucinations. Chapter 3 describes the development and evaluation of a new tool for the assessment of hallucinations in the hypnagogic and hypnopompic states. The relation of such experiences to the general tendency to experience intrusive thoughts is examined. Chapter 4 describes the testing of a model of auditory verbal hallucinations involving intrusions, thought suppression and rumination. This chapter then proposes an extended model which highlights a role for trauma and disrupted agency mechanisms in the formation and maintenance of these experiences.
Chapter 3

In a dark time: Development, validation, and correlates of the
Durham Hypnagogic and Hypnopompic Hallucinations
Questionnaire

ABSTRACT

One factor limiting research involving hypnagogic and hypnopompic (H&H) hallucinations is the lack of a brief, valid and reliable self-report measure of such experiences. The present paper reports on the development of the Durham Hypnagogic and Hypnopompic Hallucinations Questionnaire (DHQ) which consists of three unidimensional subscales assessing the presence of auditory, visual, and felt-presence experiences in the H&H state. In a sample of 18-29 year olds ($N = 365$) this scale was found to have satisfactory psychometric properties. A subsample ($n = 293$) completed self-report measures of intrusive thoughts, thought suppression and transliminality. Intrusive thoughts and the conscious desire to undertake thought suppression both correlated with levels of auditory, but not visual or felt-presence H&H hallucinations. Transliminality correlated with all DHQ subscales, but significantly more strongly with felt-presence than visual H&H experiences. Implications of these findings are considered, and recommendations for future research made.

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Chapter 3: Hypnagogia

Introduction

Hallucinatory experiences on the borders of sleep affect the majority of individuals (Ohayon, 2000). Hypnagogic (experienced in the wake-sleep transition) and hypnopompic (experienced in the sleep-wake transition) hallucinations may occur across the spectrum of sensory modalities, including the visual, tactile, and olfactory. They may also involve feelings of a presence that cannot be heard, felt, seen or smelt. Following Mavromatis’ (1987) argument that no clear phenomenological or physiological differences exist between hypnagogic and hypnopompic (henceforth H&H) hallucinations, we treat them here as belonging to the same group of phenomena. In 1925 Leaning noted that H&H states were a “little cultivated area” (p. 290), and today we still know relatively little about the correlates of H&H experiences (Watson, 2001).

One factor limiting research into such experiences is the absence of a brief, psychometrically reliable and valid self-report measure. Cheyne and colleagues (e.g., Cheyne & Girard, 2007) have developed a valuable tool to assess H&H hallucinations (the Waterloo Unusual Sleep Experiences Survey), which incorporates both qualitative and quantitative responses. This instrument was not designed, however, to have internally reliable subscales assessing the presence of H&H experiences in each modality, and is also time-consuming for participants to complete. The first aim of the present study was hence to establish a brief, valid and reliable measure of auditory, visual, and felt-presence H&H experiences. The development of such a tool, suitable for inclusion with a battery of other psychometric assessments, should help enable specific hypotheses in this area to be tested, such as whether levels of H&H experiences prospectively predict psychosis or near-death experiences (Jones & Fernyhough, in press).
Our second aim was to investigate factors potentially associated with H&H experiences. Many theories of H&H hallucinations model these as intrusions from REM sleep (Hori, Hayashi, & Morikawa, 1994). Such theories can be seen to share some commonalities with so-called ‘seepage’ models of hallucinations, in which hallucinations occur due to material crossing a boundary from the unconscious to the conscious (e.g., West, 1962). We proposed that general susceptibility to intrusions would relate to H&H experiences. A number of ways of assessing and conceptualizing individual differences in susceptibility to such incursions exist. One important such concept is that of transliminality (Thalbourne & Delin, 1994). Transliminality has been defined as the “extent to which the contents of some preconscious (or “unconscious” or “subliminal”) region of the mind are able to cross the threshold into consciousness” (Thalbourne & Delin, 1994, p.3). We hence firstly hypothesized that transliminality would relate positively to individuals’ susceptibility to H&H experiences.

Another way to conceptualize intrusions into consciousness is through the paradigm of intrusive thoughts. The intrusive occurrence of thoughts has been linked to auditory hallucinations in the waking state in clinical and non-clinical populations (Morrison & Baker, 2000; Jones & Fernyhough, 2006). However, less research has considered how intrusive thoughts relate to hallucinations in other modalities, or proposed a theoretical basis for such a relation. Our second hypothesis was hence that the tendency to experience intrusive thoughts in the waking state would be positively associated with higher levels of auditory, but not visual or felt-presence H&H experiences.

If, as hypothesized, intrusive thoughts are associated with auditory H&H experiences, cognitive strategies that encourage the occurrence of intrusive thoughts
Chapter 3: Hypnagogia

should also be linked to auditory H&H phenomena. One such strategy is likely to be thought suppression, defined as “the intentional conscious removal of a thought from subsequent conscious attention” (Wegner, 1992, p. 194). Due to its potential to create intrusive thoughts, thought suppression has been linked with auditory hallucinations in healthy populations in clear consciousness (Jones & Fernyhough, 2006). Suppression of a thought also makes it more likely to occur in an H&H hallucination (Schmidt & Gendolla, 2008). It therefore seems plausible that trait levels of thought suppression should be linked to the occurrence of auditory H&H phenomena, through its encouragement of cognitive intrusions. Our third hypothesis was thus that levels of thought suppression would be associated with auditory, but not visual or felt-presence, H&H experiences.

In summary, the present study set out to develop a brief, valid, and reliable self-report questionnaire for assessing H&H experiences. We hypothesized firstly that levels of transliminality would be positively associated with all forms of H&H experiences. Our second and third hypotheses were that auditory, but not visual or felt-presence, H&H experiences would be associated with the self-reported tendencies both to experience intrusive thoughts and to undertake thought suppression.

Method

Participants

A first sample of students (N = 399, 215 women) at a United Kingdom university, with a mean age of 19.5 years (SD = 1.1, range = 18-24) completed a 25-item, on-line H&H experiences questionnaire. Participants were recruited through e-mail invitation. There was no financial incentive to participate. Answers were given anonymously, with only age and gender being requested.
A second, separate sample of students ($N = 365, 236$ women) with a mean age of $21.1 \text{ years } (SD = 2.8, \text{ range } 18-29)$ completed a revised $14$-item version of the original H&H questionnaire. This $14$-item questionnaire was named the *Durham Hypnagogic and Hypnopompic Hallucinations Questionnaire* (DHQ). Issues around recruitment, lack of financial incentive and anonymity of responses were the same as for the first sample. Of this sample, $293 \ (195$ women) with a mean age of $21.1 \text{ years } (SD = 2.8, \text{ range } 18-29)$ consented to go on to complete the questionnaire measures detailed below.

Response rates were approximately $20\%$, and comparable to previous studies employing on-line questionnaires of similar length (e.g., Kaplowitz, Hadlock & Levine, 2004). On-line questionnaires have been shown to be a reliable method of data collection (e.g., Jones, Fernyhough, de-Wit, & Meins, 2008; Appendix A)

**Measures**

Measures employed included those described below. The development of the DHQ is detailed in Section 3.1.1.

*White Bear Suppression Inventory* (WBSI; Wegner & Zanakos, 1994) is a $15$-item self-report measure of tendency to suppress thoughts. Each item is scored on a five-point Likert scale ranging from “strongly agree” ($5$) to “strongly disagree” ($1$). Muris, Merckelbach, and Horselenberg (1996) argued that the WBSI taps intrusive thoughts as well as thought suppression, and devised a “corrected WBSI” (p. 505) which removed all items relating to intrusion (items $2, 3, 4, 5, \text{ and } 9$). The resultant scale ($WBSI_{\text{sup}}$) was found to have satisfactory internal reliability and test-retest reliability, and was used in the present study as a measure of self-reported thought suppression (Chapter 1). Following Muris et al. (1996), numerous factor analyses have confirmed that the WBSI measures both thought suppression and intrusive
thoughts. A range of studies (see Jones & Fernyhough, 2006) all identified subtly
different “unwanted intrusive thoughts” factors of the WBSI, all finding the core
items Muris et al. (1996) identified as the “intrusion items” on the WBSI (items 2, 3,
4, 5 and 9) to load onto this factor. Thus, we used these five items as a separate
measure of self-reported intrusiveness of unwanted thoughts (WBSIintru).

*Hallucination-proneness.* This was assessed using the revised Launay-Slade
Hallucination Scale (Bentall & Slade, 1985), a 12-item instrument designed to
measure predisposition to hallucination-like experiences. Each item is scored on a
five-point Likert scale ranging from “certainly applies to me” (0) to “certainly does
not apply to me” (4). Higher scores indicate a greater predisposition to hallucination-
like experiences. This tool has been found to have satisfactory psychometric
properties (ibid.). In the current administration it was stated at the start that
participants should only endorse items if they had experienced them in clear
consciousness, and not in the H&H state.

*Transliminality.* This was assessed using the 17-item scoring scheme (Lange
et al., 2000) for the 29-item Transliminality Scale (Thalbourne, 1998). Scores can
range from 13.7 to 37.3, with higher scores representing higher levels of
transliminality. Response options are “true” or “false”. Examples of items are “I have
felt that I have received special wisdom, to be communicated to the rest of humanity”
and “I think I really know what some people mean when they talk about mystical
experiences”. The validity and reliability of this scale has previously been
demonstrated (ibid).
Development of the DHQ. A preliminary 25-item questionnaire assessing the presence of auditory, visual, and felt-presence H&H hallucinations was developed through face-to-face discussions with students, an Internet search for accounts of such experiences, and integration of features of the categories used by Cheyne & Girard (2007). Preliminary instructions make it clear to participants that items should only be endorsed if they have been experienced in the H&H state. These instructions read: “In the drowsy state when you are about to fall asleep, or have just woken up, healthy people can experience hallucination-like experiences. We are interested in whether you have any experiences like this. There are a number of statements below about such experiences, please indicate if you have had such experiences or not. To be clear, all the statements refer to experiences you may have had on the border of falling asleep or waking up, and which could not have had a source in the ‘real world’. For example, the item ‘I have seen a blurry figure in the room’ refers to seeing a blurry figure when you were about to fall asleep or wake up, which you later realised was not really there, although at the time it seemed real”. Response options (and scoring) were on a 6-point Likert scale: “never” (0), “very rarely” (1), “rarely” (2), “occasionally” (3), “frequently” (4), and “very frequently” (5).

This questionnaire was administered to a first set of participants (N = 399). Initial exploratory factor analysis was then performed using principal components analysis (PCA) with oblique rotation (Direct Oblimin) on the basis that factors were likely to correlate with each other. The Kaiser-Meyer-Olkin measure of sampling adequacy was .89, and Bartlett’s test of sphericity was significant (p < .001). Items were discarded if over 85% of participants indicated they had never had the
experience, the item communality was < .4, or if feedback indicated problems with
the wording. This led to eleven items being discarded, including "I've heard animal
noises", and "I've seen someone I know to be dead". A further PCA was then
performed on the remaining fourteen items.

Scree-plot inspection, Kaiser's rule, and parallel analysis using a Monte Carlo
analysis with 1000 repetitions all suggested the extraction of three factors, with
eigenvalues of 5.67, 1.69, and 1.21, accounting for 61.23% of the observed variance.
Each item loaded (> .5) onto a single factor. The three factors clearly related to
auditory H&H hallucinations, visual H&H hallucinations, and the experience of a felt
presence.

The 14 items identified above, modified after feedback from participants, were
administered to a new sample (N = 365), and constituted the final version of the DHQ
(Table 3.1). Confirmatory factor analysis was performed on the data using AMOS
6.0, in order to examine if a three-factor solution, consisting of auditory (DHQaud:
items 2, 5, 6, 8, & 12), visual (DHQvis: items 3, 7, 9, 11, 13) and felt-presence
(DHQpres: items 1, 4, 10, 14) H&H factors, was a good fit to the data. Previous
psychometric research has demonstrated that in order to generate a well-fitting model,
it may be necessary to allow for correlated errors (Byrne, Shavelson, & Muthon,
1989). Byrne et al., (1989) have noted that “such parameter specifications are justified
because, typically, they
### Table 3.1

Endorsement rates for DHQ items ($N = 365$)

<table>
<thead>
<tr>
<th>Item</th>
<th>Mean (SD)</th>
<th>Percentage (%) of people endorsing</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Never</td>
</tr>
<tr>
<td>1. I've felt an evil presence in the room, but could not see, hear,</td>
<td>.88 (1.24)</td>
<td>58</td>
</tr>
<tr>
<td>touch or smell anyone there</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. I've heard someone calling my name</td>
<td>1.11 (1.35)</td>
<td>51</td>
</tr>
<tr>
<td>3. I've seen a blurry human figure in the room</td>
<td>.84 (1.24)</td>
<td>60</td>
</tr>
<tr>
<td>4. I've had the sense of an invisible presence watching me</td>
<td>1.16 (1.38)</td>
<td>48</td>
</tr>
<tr>
<td>5. I've heard the voice of a person I could not identify</td>
<td>.65 (1.13)</td>
<td>69</td>
</tr>
<tr>
<td>6. I've heard human speech which spoke in a garbled, unclear way</td>
<td>.56 (1.11)</td>
<td>74</td>
</tr>
<tr>
<td>7. I've seen the image of a face</td>
<td>.71 (1.19)</td>
<td>67</td>
</tr>
</tbody>
</table>
8. I've heard a voice of a person familiar to me  .99 (1.34)  56  26  12  6
9. I've seen things or figures floating in my room  .49 (1.05)  77  15  4  4
10. I've felt the presence of an intruder in my bedroom, though I didn't actually see, hear, touch, or smell anyone .94 (1.30)  55  28  12  6
11. I've clearly seen people in my room  .33 (.87)  84  9  4  2
12. I've heard non-speech sounds, such as laughter, music, or other noises .78 (1.18)  62  26  8  4
13. I've seen things in my room other than people .56 (1.11)  73  18  5  4
14. I have had the feeling of a presence in the room which I felt was aware of me too, but I couldn't actually see, hear, touch or smell them .79 (1.18)  60  28  9  3
Chapter 3: Hypnagogia

represent non-random measurement error due to method effects such as item format associated with subscales of the same measuring instrument (p. 460). We hence hypothesized that it may be necessary to allow errors of some items on the same DHQ subscale to correlate in order to achieve a satisfactory fit.

As the data was non-normal, initial analysis was performed using the asymptotically distribution free (ADF) method. However, this performs poorly for samples of the size employed here (Bentler & Yuan, 1999). We hence adjudged fit using Bentler and Yuan’s (1999) $F$ statistic ($T_F$), a modification of the ADF statistic which performs well with non-normal data in sample sizes as low as 90. Other goodness of fit indices (such as GFI, CFI, and RMSEA) depend on the choice of estimation method. As AMOS 6.0 is unable to recalculate such goodness of fit statistics for use of the $T_F$ estimation method, we were only able to report such statistics for the ADF method. As shown below, the more appropriate $T_F$ statistic often suggested better model fit than the ADF statistic, and hence the GFI, CFI, and RMSEA fit statistics reported below, based on the ADF statistic, are likely to be underestimates.

The standard minimum fit chi-squared was a poor fit to the data, $\chi^2_{ADF} (91) = 536.64, p < .001$. Similarly, a one-factor solution was found to differ significantly from the data, $T_F (77, 288) = 2.28, p < .001$ [$\chi^2_{ADF} (77) = 222.80, p < .001$, GFI = .79, CFI = .67, RMSEA = .07 (90% CI = .06-.08)]. The proposed three-factor solution also differed significantly from the data, $T_F (74, 291) = 1.51, p < .01$ [$\chi^2_{ADF} (77) = 794.40, p < .001$, GFI = .87, CFI = .82, RMSEA = .05 (90% CI = .04-.06)]. As hypothesized, modification indices indicated that the 3-factor solution would be improved by allowing errors for a number of similar items from within the same subscale (specifically DHQ items 3 & 13, 2 & 6, 5 & 6, and 2 & 8) to correlate. The resultant
model did not differ significantly from the data, $T_F(70, 295) = 1.24$, n.s. [$\chi^2_{ADF}(77) = 107.53, p < .001, GFI = .90, CFI = .92, RMSEA = .04 (90\% CI = .04-.06)$]. In this model standardized regression weights ranged from .69 to .88, and correlations between the factors ranged from .65 to .71.

Finally, Rasch analysis\(^4\) (one-parameter item response theory) was performed to determine whether each DHQ subscale was unidimensional. Rasch analysis places questionnaire response data for each individual and each question on the same spectrum of person severity and item severity. The Rasch model (Rasch, 1960) assumes that the probability that a particular individual will respond in a certain way to a particular item is a function of the relative distance between the item and person severity and only a function of this. Analyses were conducted using the Rasch Unidimensional Measurement Model (RUMM2020; Andrich, Lyne, Sheridan & Luo, 2003) software. The adequacy of DHQ scale fit to the Rasch model was evaluated using the item-trait interaction $\chi^2$ fit statistic, and item fit was evaluated through individual item $\chi^2$ fit statistics. Significant statistics ($p < .01$ given the large number of tests completed) indicate misfit to the Rasch model. Item fit residuals were also examined and a final test conducted within the RUMM framework to confirm the absence of multidimensionality in each scale. Differential Item Functioning (DIF; Holland & Wainer, 1993) by gender was also examined to determine whether responses to DHQ items were significantly influenced by the gender of the respondent.

Each of the final DHQ subscales exhibited fit to the Rasch model ($\chi^2 p > .01$) with no individual items exhibiting misfit ($\chi^2 p > .01$) or excessive residuals ($< +/-2.5$). In addition no item was subject to DIF by gender (ANOVA $p < .01$). The tests

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\(^4\) The Rasch analysis reported here was performed by David Meads of the Galen Institute, Manchester, UK.
for multidimensionality indicated that the three DHQ scales are in fact unidimensional. Although the combined 14 items of the DHQ fit the Rasch model these additional tests indicated that they could not be considered to capture a unidimensional construct.

**Pattern of responses to the DHQ.** Mean (SD, range) DHQ scores were \( \text{DHQ}_{\text{aud}} = .83 \) (.99, 0-4.6), \( \text{DHQ}_{\text{vis}} = .58 \) (.81, 0-4.2), and \( \text{DHQ}_{\text{pres}} = .95 \) (1.09, 0-4.5). Paired t-tests (with a Bonferroni corrected alpha of \( \alpha = .02 \)) showed that both mean \( \text{DHQ}_{\text{pres}} \) and \( \text{DHQ}_{\text{aud}} \) subscale scores were higher than mean \( \text{DHQ}_{\text{vis}} \) subscale scores, \( t(364) = 7.78, p < .001 \), and \( t(364) = 5.49, p < .001 \), respectively. Mean \( \text{DHQ}_{\text{aud}} \) and \( \text{DHQ}_{\text{pres}} \) scores did not differ.

85% of participants reported experiencing at least one of the items on the DHQ. 67% of participants endorsed at least one item on the \( \text{DHQ}_{\text{pres}} \), with the equivalent rates being 65% and 58% for the \( \text{DHQ}_{\text{aud}} \) and \( \text{DHQ}_{\text{vis}} \) respectively. Typically, around 5% of participants experienced frequent or very frequent H&H experiences. The most common items in each modality were hearing one’s name called, seeing a blurry figure in the room, and the feeling of an invisible presence watching. There were no gender differences in scores on any of the DHQ subscales, or at item level when Bonferroni corrections were employed.

**Reliability and validity of the DHQ.** Cronbach’s alphas for all DHQ subscales were satisfactory (Table 3.2). Face validity of the DHQ was maximized by developing the original questionnaire items from accounts of such experiences. Convergent validity of the DHQ was evaluated by its correlation with the Revised Launay-Slade Hallucination Scale. Given that (regardless of frequency of occurrence) approximately 50% of people who report daytime hallucinations also experience H&H hallucinations (Ohayon, 2000), we expected that hallucinatory experiences in
the H&H state would be related to the more general tendency to experience hallucinatory experiences. In line with this, a positive correlation was found between the LSHS-R and all subscales of the DHQ (Table 3.3).

Table 3.2

Descriptive statistics (n = 293)

<table>
<thead>
<tr>
<th></th>
<th>Mean (SD, range)</th>
<th>Cronbach’s alpha</th>
</tr>
</thead>
<tbody>
<tr>
<td>DHQaud</td>
<td>0.82 (0.99, 0-4.6)</td>
<td>0.86</td>
</tr>
<tr>
<td>DHQvis</td>
<td>0.59 (0.83, 0-4.2)</td>
<td>0.82</td>
</tr>
<tr>
<td>DHQpres</td>
<td>0.93 (1.08, 0-4.5)</td>
<td>0.87</td>
</tr>
<tr>
<td>WBSIintru</td>
<td>18.32 (4.14, 6-25)</td>
<td>0.80</td>
</tr>
<tr>
<td>WBSIsup</td>
<td>34.26 (8.43, 10-50)</td>
<td>0.87</td>
</tr>
<tr>
<td>Transliminality</td>
<td>22.21 (3.66, 13.7-35.0)</td>
<td>0.75</td>
</tr>
<tr>
<td>LSHS-R</td>
<td>18.21 (9.24, 0-42)</td>
<td>0.85</td>
</tr>
</tbody>
</table>

Intrusion-proneness and H&H experiences

Descriptive statistics for the participants (n = 293) who went on to complete further questionnaires are given in Table 3.2. LSHS-R, WBSI_{sup} and WBSI_{intru} scores were in line with previous studies assessing these variables in student populations (e.g., Jones & Fernyhough, 2006). Bivariate correlational analyses are presented in Table 3.3. Bonferroni-corrected significance levels were employed and alpha set at $p = .001$ (i.e., $\sim .05/28$). Transliminality correlated significantly with all DHQ subscales. DHQ_{pres} correlated more strongly with transliminality than DHQ_{vis}, $\chi^2 = 4.94$, $p < .05$, but not significantly differently to DHQ_{aud}. Intrusive thoughts and thought
suppression both correlated significantly with DHQ<sub>aud</sub>, but not with any other DHQ subscales.

Table 3.3
Bivariate correlational analysis

<table>
<thead>
<tr>
<th></th>
<th>DHQ&lt;sub&gt;aud&lt;/sub&gt;</th>
<th>DHQ&lt;sub&gt;vis&lt;/sub&gt;</th>
<th>DHQ&lt;sub&gt;pres&lt;/sub&gt;</th>
<th>WBSI&lt;sub&gt;intru&lt;/sub&gt;</th>
<th>WBSI&lt;sub&gt;sup&lt;/sub&gt;</th>
<th>Transliminality</th>
<th>LSHS-R</th>
</tr>
</thead>
<tbody>
<tr>
<td>DHQ&lt;sub&gt;aud&lt;/sub&gt;</td>
<td>1</td>
<td>.56*</td>
<td>.46*</td>
<td>.27*</td>
<td>.23*</td>
<td>.35*</td>
<td>.50*</td>
</tr>
<tr>
<td>DHQ&lt;sub&gt;vis&lt;/sub&gt;</td>
<td>-</td>
<td>1</td>
<td>.58*</td>
<td>.16</td>
<td>.11</td>
<td>.28*</td>
<td>.43*</td>
</tr>
<tr>
<td>DHQ&lt;sub&gt;pres&lt;/sub&gt;</td>
<td>-</td>
<td>-</td>
<td>1</td>
<td>.11</td>
<td>.14</td>
<td>.44*</td>
<td>.38*</td>
</tr>
<tr>
<td>WBSI&lt;sub&gt;intru&lt;/sub&gt;</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>1</td>
<td>.65*</td>
<td>.35*</td>
<td>.54*</td>
</tr>
<tr>
<td>WBSI&lt;sub&gt;sup&lt;/sub&gt;</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>1</td>
<td>.28*</td>
<td>.35*</td>
</tr>
<tr>
<td>Transliminality</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>1</td>
<td>.50*</td>
</tr>
<tr>
<td>LSHS-R</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>1</td>
</tr>
</tbody>
</table>

* p < .001

Discussion

The research reported here represents the first attempt to produce a brief, psychometrically reliable and valid self-report measure of H&H phenomena. Exploratory and confirmatory factor analysis indicated that the resulting instrument, the DHQ, had a three-factor structure, indexing auditory H&H hallucinations, visual H&H hallucinations, and the experience of a felt presence. Rasch analysis confirmed that each of these scales was unidimensional and therefore capable of generating summed total scores. The reliability and validity of this tool were satisfactory, with the DHQ subscales being found to be internally reliable and to have both face and convergent validity.
Each subscale of the DHQ had at least one item endorsed by approximately two-thirds of participants, with only 15% of participants reporting never having experienced any of the items on the DHQ. The resulting prevalence rate of H&H phenomena of 85% is significantly higher than the 31% rate (in a 15-44 year age group) of hypnagogic hallucinations reported by Ohayon (2000) but consistent with the rates of hypnagogic (79%) and hypnopompic (72%) imagery (in a 20-29 year old student sample) reported by Richardson, Mavromatis, Mindel and Owens (1981). One possible reason for this disparity in prevalence is that Ohayon’s methodology, which involved collecting data via telephone interviews whilst also asking about mental illnesses, may have led to reduced disclosure. Another possible reason is that the frequency of H&H experiences is positively related to levels of drug use (Ohayon, 2000), and drug use is higher in student populations than in the general population (Hope, Dring, & Dring, 2005). Finally, Ohayon does not report the question(s) used to assess the presence of H&H experiences. If Ohayon only utilized a single question, the multiple-item DHQ could likely have generated higher endorsement rates due to its provision of specific examples of the relevant experiences.

Auditory and felt-presence H&H experiences were found to be more frequent than visual experiences. This is in contrast to the findings of Ohayon (2000) who found visual experiences to be more common than auditory or felt-presence experiences. No clear gender differences in DHQ responses were found, in contrast to previous findings that such experiences are more common in women than men (Ohayon, 2000; Richardson et al., 1981). We did however note that a number of items not included in our final version of the DHQ, such as “Have you ever felt a benevolent, protecting figure?” were significantly more often endorsed by women.
than men. Future studies may wish to consider whether gender differences are only found in relation to specific H&H content.

Our study also aimed to examine the relation between cognitive intrusions and H&H experiences. In line with our first hypothesis, transliminality was positively related to all subscales of the DHQ. We found support for our second and third hypotheses that auditory (but not visual or felt-presence) H&H experiences would be associated with the self-reported susceptibility to intrusive thoughts and tendency to undertake thought suppression. Although a weak correlation, this could be taken to suggest that different explanatory models are needed for each modality of H&H phenomena.

A number of limitations of the present study need to be acknowledged. Firstly, the forgetting or elaboration of hypnagogic experiences may affect responses on the DHQ. Secondly, in order to achieve satisfactory internal consistency, the visual H&H factor of the DHQ is weighted towards experiences involving people, with less emphasis on experiences such as seeing lights, landscapes, and animals. Thirdly, the DHQ does not distinguish between experiences in the hypnagogic and hypnopompic states. In order to address this, the DHQ could be administered to a sample of participants twice, with instructions relating to hypnagogic and hypnopompic experiences in each instance. Another potential limitation is that the DHQ does not make a distinction between H&H hallucinations associated with sleep paralysis and those not. Future studies may also wish to establish the discriminant validity of the DHQ, as well as its test-retest reliability.

In addition to the broad categories assessed here (auditory, visual, and felt-presence H&H experiences), future researchers may also wish to consider a multidimensional conception of H&H phenomena. It would be of particular interest to
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examine what factors affect the content of the experiences, the distress caused by
them, and the individual’s conviction about their reality. Furthermore, it may be asked
what factors differentiate individuals who experience a primarily benevolent felt
presence from those whose experience is primarily of an evil presence. Trauma may
be one candidate. Future research may also consider the phenomenological relation of
H&H hallucinations to those that occur in clear consciousness. Additionally, the DHQ
could be used in samples at high risk for schizophrenia to examine if it predicts onset
of this disorder. It is hoped that the present study will be seen as a step towards
allowing greater understanding of the causes, correlates and consequences of this
intriguing phenomenon.
References


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University of Chicago Press.

differences in hypnagogic and hypnopompic imagery. *Journal of Mental
Imagery, 5,* 91-96.

Schmidt, R. E., & Gendolla, G. H. E. (2008). Dreaming of white bears: The return of
the suppressed at sleep onset. *Consciousness and Cognition, 17,* 714-724.

*Journal of the American Society for Psychical Research, 92,* 402-419.

paranormal, creative personality, mystical experience and psychopathology.
*Journal of Parapsychology, 58,* 3-38.

Watson, D. (2001). Dissociations of the Night: Individual Differences in Sleep-
Related Experiences and Their Relation to Dissociation and Schizotypy.
*Journal of Abnormal Psychology, 110,* 526-535.

suppression of unwanted thoughts. In M. Zanna (Ed.), *Advances in
experimental social psychology,* (Vol. 25, pp. 193-225). San Diego, CA:
Academic Press.

Personality, 62,* 615-640.

Chapter 4

Rumination, reflection, intrusive thoughts, and hallucination-proneness: towards a new model

ABSTRACT

Although rumination has been proposed to play an important role in the creation of hallucinations, direct empirical tests of this proposal have not yet been performed. Employing a distinction between ruminative and reflective self-consciousness, we set out to test a new model of the relations among rumination, reflection, intrusive thoughts, thought suppression, social anxiety, and hallucination-proneness. This model proposed that rumination would be related to hallucination-proneness through the mediating variable of intrusive thoughts, but that reflection would not be related to hallucination-proneness. The model was tested in a student population (N = 296) using path analyses. A modified version of the model was found to be a good fit to the data, once a direct path from reflection to hallucination-proneness had been added. As hypothesized, rumination was related to hallucination-proneness only indirectly, through the mediating variable of intrusive thoughts. Implications for interventions and future directions for research are considered.

Chapter 4: Rumination and hallucination-proneness

Introduction

The cognitive mechanisms underlying auditory verbal hallucinations (AVHs) are still not well understood. Many contemporary accounts of AVHs propose that such experiences have their roots in the voice-hearer's own inner speech (e.g., Leudar, Thomas, McNally, & Glinski, 1997). One problem for such theories arises from the observation that, in both clinical and non-clinical samples, AVHs are typically perceived as the voice of another person (Nayani & David, 1996). One approach to this problem is to ask why voice-hearers would be generating other people’s voices in their inner speech (Jones & Fernyhough, 2007b). We have previously argued, employing a Vygotskian approach, that inner speech should naturally take a dialogic form, i.e., be in the form of a dialogue rather than a first person monologue (Jones & Fernyhough, 2007a, 2007b; Appendix B). If dialogic inner speech is the norm, then it raises the question of what factors may be relevant to the experiencing of such internalized voices as AVHs (Fernyhough, 2004).

Fowler et al. (2006) have proposed that inner speech may be experienced as alien if it involves the recreation of a voice associated with a stressful event in one’s past. Based on observations that early trauma, such as sexual and physical abuse, often precede onset of AVHs (e.g., Read, van Os, Morrison, & Ross, 2005), they suggest that rumination or inner dialogues about interactions with the people in these situations could form the raw material of some AVHs. For example, “rumination or inner dialogue about self in relationship to what a shaming and insulting abuser might say about one’s current actions” (Fowler et al., 2006, p. 113) may explain why AVHs commonly take the form of commentaries on present actions. This proposal is also in line with the finding that a significant number of AVHs have similar themes and content to earlier abuse experiences (Hardy et al., 2005).
Chapter 4: Rumination and hallucination-proneness

Although the relation between rumination and hallucinations has not yet been studied, the relation between hallucinations and a construct related to rumination, the metacognitive trait of cognitive self-consciousness, defined by Cartwright-Hatton & Wells (1997) as "the tendency to focus attention on thought processes" (p. 387), has been examined. For example, Baker and Morrison (1998) found that levels of cognitive self-consciousness were higher in patients with schizophrenia with auditory hallucinations than in patients with schizophrenia with delusions but no auditory hallucinations.

Rumination may also be related to hallucinations in other modalities, such as the visual. Allen, Coyne, and Console’s (1997) argument that stressful or traumatic events may leave individuals “vulnerable to ... [their] inner world” (p. 332) allows for an inner experience consisting of reconstructed visual images as well as auditory experiences. Indeed, indirect evidence suggests that rumination will be associated with hallucinations in general, rather than specifically with hallucinations of the auditory-verbal type. For example, cognitive self-consciousness has been found to be positively related to a general measure of hallucination-proneness, the Launay–Slade Hallucination Scale (Launay & Slade, 1981), in student populations (Morrison, Wells, & Northard, 2000; Sterling, Barkus, & Lewis, 2007). This led Sterling et al. (2007) to conclude that hallucinations are related to a “relatively stable trait-like form of ‘ruminative thinking’” (p. 1407).

Nevertheless, as the studies discussed above utilized a general measure of cognitive self-consciousness rather than dedicated measures of rumination, there is as yet no evidence for a specific relation between rumination and hallucinatory experiences. Trapnell and Campbell (1999) have argued that self-consciousness of one’s thoughts should be divided into two distinct forms, based on the individual’s
motivations. They note that one reason why individuals may pay attention to their thoughts is because they are “motivated by curiosity or epistemic interest in the self” (p. 297). This form of self-consciousness is termed reflection, and is proposed to be associated with adaptive self-knowledge and generally positive effects. In contrast, individuals may also focus on their own thoughts for reasons of negative affect and anxiety. Trapnell and Campbell term this rumination, or “self-attentiveness motivated by perceived threats, losses, or injustices to the self” (p. 297).

Given Fowler et al.’s (2006) contention that it is recreating imagined interactions with significant individuals associated with negative affect or anxiety that form the raw material of some hallucinations, rumination as conceptualized by Trapnell and Campbell (1999) would appear to be likely to relate strongly to hallucinations. The first aim of the present study was hence to investigate the relations among rumination, reflection and hallucination-proneness. We hypothesized that rumination, but not reflection, would be related to hallucination-proneness.

We proposed that this relation would occur because of rumination’s tendency to cause cognitive intrusions. Cognitive intrusions have been linked to hallucinations in both clinical and non-clinical populations. Morrison, Haddock, and Tarrier (1995) proposed that intrusive thoughts may form the raw material for auditory hallucinations, with intrusive imagery being the raw material of visual hallucinations. This has received support from studies showing that patients with schizophrenia with auditory hallucinations report more cognitive intrusions than psychiatric and healthy controls (Morrison & Baker, 2000). In a student population Jones and Fernyhough (2006b) have also found a relation between intrusive thoughts and hallucination-proneness. As rumination has been found to increase both the number and intensity of cognitive intrusions (e.g., Guastella & Moulds, 2007), we proposed that rumination
would relate to hallucination-proneness through its tendency to promote cognitive intrusions. As there is no evidence that the other element of self-consciousness identified by Trapnell and Campbell (1999), reflection, is related to intrusions, we predicted no relation between and hallucination-proneness.

We were also interested in examining other variables potentially related to rumination, cognitive intrusions and hallucinations. Thought suppression, “the intentional conscious removal of a thought from subsequent conscious attention” (Wegner, 1992, p. 194), is known to encourage both intrusive thoughts and rumination. Attempting thought suppression has been found to cause suppressed thoughts to intrude into consciousness in an unexpected manner (Wegner, 1992). Furthermore, like rumination, thought suppression has been found to increase the intensity of intrusive memories (Yoshizumi & Murase, 2007). Indeed, there is evidence that thought suppression may be related to hallucinations due to its tendency to encourage intrusive thoughts (Jones & Fernyhough, 2006b). Rumination is also associated with greater levels of thought suppression (Erskine, Kvalilashvili, & Kornbrot, 2007). Thus, we proposed that rumination may be linked to hallucination-proneness through its direct and indirect (via thought suppression) encouragement of cognitive intrusions.

The final factor of interest was social anxiety. Trapnell and Campbell (1999) proposed that rumination, but not reflection, should be related to anxiety. In support of this proposal, social anxiety has been found to be associated with higher levels of ruminative thought (Kocovski & Rector, 2007). Individuals with high levels of social anxiety have also been shown to attempt more thought suppression (Magee & Zinbarg, 2007), and to do so less successfully, experiencing more intrusions and unwanted thoughts than controls (Fehm & Margraf, 2002; Magee & Zinbarg, 2007).
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This led us to propose that social anxiety may relate to hallucinations through its tendency to promote cognitive intrusions via the mediating variables of rumination and thought suppression.

In summary, the findings and arguments set out above led us to propose a model of relations between hallucination-proneness, rumination, reflection, intrusive thoughts, thought suppression, and social anxiety, as shown in Figure 4.1. The key points of this model are as follows. Rumination is understood as having an indirect effect on hallucination-proneness through acting as the generative force behind the thoughts that intrude into consciousness. Reflection does not relate to hallucination-proneness in the model, either directly or indirectly.

It is now well established both that hallucinations exist on a continuum with normal experiences, and stretch into the general population (Johns & van Os, 2001). It has also been argued that the mechanisms underlying sub-clinical (i.e., experienced by non-patients in the general populace) and clinical hallucinations may be the same. For example, based on the finding that risk factors for hallucinations are the same for those with sub-clinical and clinical hallucinations, Aleman and Larøi (2008) have argued for “a developmental mechanism in nonclinical participants that is similar to the one reported in schizophrenia” (p. 82). We hence proposed to perform an initial test of this model, using path analyses, in an analogue study using data obtained from a sample drawn from a student population.
Figure 4.1

Model of hallucinations to be tested

- Reflection
- Intrusive thoughts
- Rumination
- Hallucination-proneness
- Social anxiety
- Thought suppression
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Method

Participants

Undergraduate and postgraduate students \((N = 296, 198 \text{ women})\) at a UK university, with a mean age of 21.3 years \((SD = 2.83, \text{ range } = 18-29)\), completed online questionnaires. Participants were recruited through e-mail invitation and there was no financial incentive to participate; the likelihood of repeated participation was thus considered negligible. Response rates were approximately 20%, and comparable to previous studies employing online questionnaires of similar length (e.g., Kaplowitz, Hadlock & Levine, 2004). Answers were given anonymously, with only the age and gender of the participants being recorded. Thought suppression and hallucination-proneness data reported here overlaps with that reported in Jones, Fernyhough, & Meads (2009).

Measures

*White Bear Suppression Inventory* (WBSI; Wegner & Zanakos, 1994) is a 15-item self-report measure of tendency to suppress thoughts. Each item is scored on a five-point Likert scale ranging from “strongly agree” (5) to “strongly disagree” (1). Higher scores indicate a greater tendency to suppress thoughts. Muris, Merckelbach, and Horselenberg (1996) have argued that the WBSI taps intrusive thoughts as well as thought suppression, and devised a “corrected WBSI” (p. 505) which removed all items relating to intrusion (items 2, 3, 4, 5, and 9). The resultant scale, which we term WBSI\textsubscript{sup}, was found to have satisfactory internal reliability and test-retest reliability, and was used in the present study as a measure of self-reported thought suppression (Chapter 1; Chapter 2). Following Muris et al. (1996), numerous factor analyses have confirmed that the full version of the WBSI measures both thought suppression and intrusive thoughts (e.g., Rassin, 2003) and all such studies have found the core items
Muris et al. (1996) identified as the ‘intrusion items’ on the WBSI (items 2, 3, 4, 5 and 9) to load onto this factor. Thus, we used items 2, 3, 4, 5, and 9 of the WBSI as a separate measure of self-reported intrusiveness of unwanted thoughts ($WBSI_{intru}$).

**Hallucination-proneness.** This was assessed using the revised Launay-Slade Hallucination Scale (LSHS-R; Bentall & Slade, 1985), a 12-item instrument designed to measure predisposition to hallucination-like experiences. Items are scored on a five-point Likert scale ranging from “certainly does not apply to me” (0) to “certainly applies to me” (4). Examples of items include “In the past, I have often had the experience of hearing a person’s voice and then found that no-one was there”, and “I have been troubled by hearing voices in my head”. This scale also contains one item directly related to unwanted intrusive thoughts (“No matter how hard I try to concentrate, unrelated thoughts always creep into my mind”). We hence removed this item from the scale in order to avoid inflating the correlation between the LSHS-R and our measure of intrusive thoughts ($WBSI_{intru}$). The resulting 11-item scale had a total score that could range from 0 to 44. Higher scores indicate a greater predisposition to hallucination-like experiences (Chapter 3).

**Rumination and Reflection.** This was assessed using the Rumination and Reflection Questionnaire (RRQ; Trapnell & Campbell, 1999), a 24-item self-report measure of individuals’ dispositions to engage in ruminative and reflective private self-consciousness. The scale is scored as two separate 12-item ruminative and reflective subscales, derived by factor analysis. Examples of ruminative and reflective items include “I always seem to be ‘re-hashing’ in my mind recent things I've said or done” and “I'm very self-inquisitive by nature” respectively. Items are scored on a 5-point Likert scale ranging from “strongly agree” (5) to “strongly disagree” (1). Total scores on each subscale are divided by twelve to give a mean item score, hence
subscale scores may range from 1-5. This instrument has been shown to have good psychometric properties (ibid).

Social Anxiety: This was assessed by the Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987), a 24-item self-report measure in which participants first rate the amount of fear a social situation causes them. This is scored on a four-point Likert scale ranging from “none” (0) to “severe” (3). Participants then rate how much they avoid such situations, and respond on a 4-point Likert scale ranging from “never” (0) to “usually” (3). An overall total score is calculated by summing the total fear and total avoidance scores. The LSAS has been shown to be a valid and reliable tool (Baker, Heinrichs, Kim, & Hofmann, 2002).

Results

Descriptive statistics are presented in Table 4.1. Rumination and reflection scores on the RRQ were in line with those found by Trapnell and Campbell (1999) in the general population. WBSI_{sup} and WBSI_{intra} scores were in line with previous studies assessing these variables in student populations (e.g., Jones & Fernyhough, 2006b). Scores on our 11-item version of the LSHS-R were in line with pro-rated scores of students taken from previously published data on the 12-item version of this scale (e.g., Waters, Badcock, & Maybery, 2003). Cronbach’s alphas for all scales employed were satisfactory. Partial correlations (controlling for age and gender) between the variables are presented in Table 4.2. Due to the number of correlations performed (15), a Bonferroni correction was employed and alpha set at $\alpha = .05/15 = .003$. 

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Table 4.1

Descriptive statistics for variables under investigation (N = 296)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean (SD, range)</th>
<th>Cronbach's alpha</th>
</tr>
</thead>
<tbody>
<tr>
<td>LSHS-R</td>
<td>15.11 (8.77, 0-38)</td>
<td>.84</td>
</tr>
<tr>
<td>Rumination</td>
<td>3.85 (.70, 1.17-5.00)</td>
<td>.91</td>
</tr>
<tr>
<td>Reflection</td>
<td>3.33 (.77, 1.25-5.00)</td>
<td>.85</td>
</tr>
<tr>
<td>WBSI\text{intru}</td>
<td>18.34 (4.12, 6-25)</td>
<td>.80</td>
</tr>
<tr>
<td>WBSI\text{sup}</td>
<td>34.29 (8.42, 10-50)</td>
<td>.87</td>
</tr>
<tr>
<td>SocAnx</td>
<td>45.64 (27.61, 2-144)</td>
<td>.94</td>
</tr>
</tbody>
</table>

Note. LSHS-R = Revised Launay Slade Hallucination Scale, WBSI\text{intru} = Intrusive thoughts subscale of the White Bear Suppression Inventory, WBSI\text{sup} = Thought suppression subscale of the White Bear Suppression Inventory, SocAnx = Liebowitz Social Anxiety Scale.

Table 4.2

Partial correlations between variables (controlling for age and gender)

<table>
<thead>
<tr>
<th></th>
<th>LSHS-R</th>
<th>Rumination</th>
<th>Reflection</th>
<th>WBSI\text{intru}</th>
<th>WBSI\text{sup}</th>
<th>SocAnx</th>
</tr>
</thead>
<tbody>
<tr>
<td>LSHS-R</td>
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<td>.39*</td>
<td>.30*</td>
<td>.46*</td>
<td>.31*</td>
<td>.19*</td>
</tr>
<tr>
<td>Rumination</td>
<td>1</td>
<td>.24*</td>
<td>.57*</td>
<td>.42*</td>
<td>.33*</td>
<td></td>
</tr>
<tr>
<td>Reflection</td>
<td>1</td>
<td>.15</td>
<td>.04</td>
<td>.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>WBSI\text{intru}</td>
<td>1</td>
<td></td>
<td>.64*</td>
<td>.27*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>WBSI\text{sup}</td>
<td>1</td>
<td></td>
<td></td>
<td>.32*</td>
<td></td>
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<tr>
<td>SocAnx</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

* p < .003
The model shown in Figure 4.1 was subjected to path analysis using AMOS 6.0. The sample size was deemed appropriate for reliable performance of such analysis as it met the criterion of $N > 200$ proposed by Barrett (2007). Due to data non-normality, analysis was performed using Bentler and Yuan's (1999) $T_F$ statistic, which has been shown to be reliable with non-normal data (ibid). The model in Figure 4.1 was found to differ significantly to the data, $T_F (8, 288) = 3.39, p < .01, \text{GFI} = .97, \text{AGFI} = .92, \text{CFI} = .96, \text{RMSEA} = .09$. Modification indices indicated that the model fit could only be significantly improved by addition of a direct path from Reflection to LSHS-R. This model was found to be an excellent fit to the data, $T_F (7, 289) = 1.48, \text{n.s., GFI} = .99, \text{AGFI} = .97, \text{CFI} = .99, \text{RMSEA} = .04$. Standardized estimates of the strength of the relations between the variables for this revised model are shown in Figure 4.2. Modification indices indicated that adding further additional paths between variables (such as a direct path between Rumination and LSHS-R) would not improve the fit of the model.
Figure 4.2

Standardized estimates of path strengths

Note. $p < .001$ for all standardized co-efficients
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Discussion

The present study aimed to provide a test of the model of hallucination-proneness set out in Figure 4.1. Path analyses indicated that the model was a poor fit to the data. After examination of modification indices, our original model was revised to include a direct path from reflection to hallucination-proneness. This amended model (Figure 4.2) was found to be an excellent fit.

A number of features of this model are worthy of discussion. Firstly, although correlation analyses indicated a strong relation between rumination and hallucination-proneness, the path analysis, as predicted, demonstrated that this relation was not direct, but rather mediated by intrusive thoughts. Furthermore, as predicted, rumination was found to be associated with intrusive thoughts directly, as well as indirectly through the mediating variable of thought suppression. This is consistent with a model of hallucination-proneness that sees intrusive thoughts playing a key role, with levels of intrusive thoughts being influenced by ruminative and thought suppression processes.

As noted above, reflection was found, contrary to our prediction, to be directly related to levels of hallucination-proneness. It is possible that this relation may have obtained due to the increased tendency of reflective individuals to notice their thoughts, and hence to notice dissonance between their meta-cognitive beliefs and the actual occurrence of thoughts. Morrison et al. (1995) have proposed that such dissonance may result in individuals making attributions of such thoughts to another, resulting in hallucination formation. Finally, in line with previous research, social anxiety was found to relate to rumination and thought suppression, but these relations, although statistically significant, were weak. This suggests that, although social
anxiety may influence rumination and suppression, it may not be a key causal factor in the creation of hallucinations.

One possible objection to the argument that intrusive thoughts are the raw material of hallucinations is that those with hallucinations should report fewer, rather than more, intrusive thoughts, as any such intrusions would be experienced as hallucinations. It is not clear, however, that all intrusive thoughts will automatically be experienced in this way. For example, in the case of AVHs, the factors affecting whether a verbal intrusion is experienced as a hallucination include individual differences in source monitoring abilities (Bentall, 1990). It has been proposed that source monitoring deficits may be particularly associated with verbal imagery of non-self speakers rather than one’s ordinary inner speech (Hoffman, Varanko, Gilmore, & Mishara, 2008). Based on a review of neuroimaging studies in this area, we too have advocated the position that the raw material for AVHs is inner speech involving non-self speakers (Jones & Fernyhough, 2007).

A number of limitations of our study must be acknowledged. Firstly, it is not able to establish the direction of causation among the variables under investigation. It is possible that a more complicated pattern of relations could exist. For example, rumination and thought suppression could be seen as maladaptive strategies to cope with intrusive thoughts (rather than agents acting in the genesis of them) that work in a feedback circuit to amplify the effects of cognitive intrusions (Ehlers & Steil, 1995).

Secondly, given that the LSHS-R is heavily weighted towards AVHs, and that the theoretical and empirical arguments for a relation between the variables discussed here are strongest for AVHs, it may be that our model is more appropriate for AVHs than visual hallucinations. Future studies may wish to examine whether this model holds equally well for AVHs and visual hallucinations. Thirdly, the present study
lacked a sufficient sample size (Barrett, 2007) to examine whether the model held equally for males and females. It is hoped that future studies with larger samples may address this issue. Fourthly, metacognitive beliefs not examined here, such as beliefs about the uncontrollability and danger of thoughts, are also related to hallucination-proneness (Morrison et al., 1995; Jones & Fernyhough, 2006b). Future work may wish to investigate the relations of such factors to the present variables. A fifth limitation was the self-report nature of the study, which future research might overcome through the employment of behavioural measures. For example, cognitive intrusions or the tendency to ruminate could be assessed by experience sampling techniques such as have been used effectively in research into delusions (Thewissen, Bentall, Lecomte, van Os, & Myin-Germeys, 2008). Finally, another limitation of the present study was its failure to control for levels of depression in participants, which is associated with rumination (Spasojevic & Alloy, 2001).

Looking forward, it is worth considering how our model may be extended to account for the causes of ruminative and reflective thinking, factors causing intrusions to be experienced as not authored by the self, and whether it is likely to be applicable to hallucinations in populations such as patients with schizophrenia. Firstly, in terms of accounting for the causes of ruminative thinking, traumatic stress may play a key role in generating such ruminative thinking (Gold & Wegner, 1995). Secondly, our model needs extending to include other factors such as individuals’ appraisals of, and responses to, cognitive intrusions (Morrison, Frame, & Larkin, 2003), as well as other factors that may be involved in causing self-generated cognitions to feel non-self generated (Chapter 5; Chapter 6). These may include disrupted source monitoring abilities (Bentall, 1990), and corollary discharge mechanisms (Jones & Fernyhough, 2006; see Chapter 5). Such factors are termed ‘Disrupted agency mechanisms’ in our
Chapter 4: Rumination and hallucination-proneness

extended hypothesized model, presented in Figure 4.3. As we have suggested our model may be most appropriate to AVHs, we have focused on these experiences in this model.

In terms of the applicability of this model to clinically relevant AVHs, such as in patients with schizophrenia, a number of arguments are suggestive that this model may be valid, although this must ultimately be tested by future research. Firstly, as we noted in the Introduction, common mechanisms may indeed underlie hallucination-proneness and clinical hallucinations. For example, abuse and trauma have been found to be associated with hallucinations (especially AVHs) in both clinical (Read et al., 2005) and non-clinical (Morrison & Petersen, 2003) populations. Similarly, it has been argued that it is the content and distress associated with clinical AVHs, such as the distressing abusive content found particularly in clinical populations (Nayani & David, 1996), and not their form or pragmatics, that separates AVHs in psychiatric and non-psychiatric populations. We would hence call for this model to be tested, in both clinical and non-clinical samples, by replicating the present study whilst also taking appropriate measures of exposure to trauma and stressful events. Such studies may also wish to use more detailed measures of the tendency to experience intrusive thoughts, such as the Intrusive Thoughts Questionnaire (Reynolds & Salkovskis, 1992). Similarly, when assessing the tendency to ruminate, existing measures could be adapted to assess the extent to which imagined responses from other individuals are created in inner speech or produced in visual imagery.

Finally, if the model can be validated in future studies, it may inform interventions with those distressed by hallucinatory experiences, or those at high risk for such experiences. Firstly, strategies that can help reduce the frequency of cognitive intrusions may be beneficial. One such strategy may be for individuals to be
encouraged to openly express stressful or traumatic experiences. As Sparrow and Wegner (2006) have found that intrusive thoughts may be deactivated by merely expressing them, such a strategy may profitably impact upon the frequency of hallucinations. Furthermore, interventions that attempt to directly reduce rumination on and around traumatic events may also be likely to prove beneficial. For example, self-affirmation has been found to be successful at reducing rumination on negative events (Koole, Smeets, Van Knippenberg, & Dijksterhuis, 1999). That said, more work requires to be performed to validate the model presented here before clear recommendations on cognitive-behavioural interventions can be made.
Figure 4.3

Extended proposed model of cognitive mechanisms behind hallucinations

- Stressful / traumatic event
- Reflection
  - Ruminative inner dialogue involving auditory verbal imagery of non-self speakers.
- Depression / anxiety
- Cognitive intrusions
- Disrupted agency mechanisms
- Thought suppression
- AVHs
References


Chapter 4: Rumination and hallucination


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Chapter 4: Rumination and hallucination


HALLUCINATIONS AND AGENCY
Chapter 4 highlighted that, in addition to the presence of factors such as rumination, thought suppression, intrusive thoughts and anxiety, there is the need to understand how self-generated thoughts come to be experienced as non-self produced. Mechanisms involved in this process were termed disrupted agency mechanisms. Chapter 5 offers a detailed consideration of how agency mechanisms may typically work and how these may come to be disrupted in those who experience AVHs. This draws on the key concepts of the forward model and the illusion of conscious will. Chapter 5 also offers insights into how a Vygotskian conception of inner speech may prove valuable in enhancing our understanding of AVHs. Chapter 6 then turns to an empirical experimental test of whether agency mechanisms are disrupted in those prone to hallucinations.
Chapter 5

Thought as Action: Inner Speech, Self-monitoring, and Auditory Verbal Hallucinations

ABSTRACT

Passivity experiences in schizophrenia are thought to be due to a failure in a neurocognitive action self-monitoring system (NASS). Drawing on the assumption that inner speech is a form of action, a recent model of auditory verbal hallucinations (AVHs) has proposed that AVHs can be explained by a failure in the NASS. In this article, we offer an alternative application of the NASS to AVHs, with separate mechanisms creating the emotion of self-as-agent and other-as-agent. We defend the assumption that inner speech can be considered as a form of action, and show how a number of previous criticisms of applying the NASS to AVHs can be refuted. This is achieved in part through taking a Vygotskian developmental perspective on inner speech. It is suggested that more research into the nature and development of inner speech is needed to further our understanding of AVHs.

Chapter 5: Thought as action

Introduction

The phenomenon of auditory verbal hallucinations (AVHs), where individuals report hearing speech in the absence of any external stimulation, continues to puzzle psychiatrists and psychologists. Schneider (1959) classified AVHs as a first-rank symptom of schizophrenia, reflecting the approximately 60 to 74% of those with schizophrenia who report experiencing them (Wing, Cooper, & Sartorius, 1974; Slade & Bentall, 1988). However a movement has developed away from understanding AVHs as necessarily signifying pathology, and towards an acceptance that voice-hearing can be a part of normal experience (Johns & van Os, 2001; General Introduction). Furthermore, there do not seem to be radical differences in the structure and functions of AVHs between voice-hearers with a diagnosis of schizophrenia and those without (Leudar, Thomas, McNally, & Glinski, 1997). Whether in a clinical or non-clinical sample, one of the fundamental characteristics of AVHs is their alien quality. In this article, we take a new look at the question of how it is possible that a self-generated cognition may come to be experienced as produced and performed by an agent other than the self.

Explaining Agency: Is It Me?

Frith and colleagues (e.g., Frith, Blakemore, & Wolpert, 2000) have developed an elegant model of the passivity experiences, such as delusions of control, found in schizophrenia. This model attributes such experiences to deficits in a postulated neurocognitive action self-monitoring system (NASS), and has had its predictions supported by empirical research (e.g., Blakemore, Wolpert, & Frith, 1998). The NASS model is based on Miall and Wolpert’s (1995) forward model which was developed to model systems in which, due to temporal constraints, it makes sense to base decisions on the predicted consequences of actions. Frith and colleagues’ utilization of these ideas may
be summarized as follows (adapted from Frith et al., 2000, and Blakemore, 2003; see Figure 5.1).

Firstly a representation is created of what motor command is needed to achieve a particular goal, based on the estimated current state of the system and the desired end-state. The motor command needed to achieve this goal is then issued. In parallel to this an efference copy of the motor command is also issued. The efference copy is used by the brain, in conjunction with knowledge of the current state of the system, to create a prediction of what will happen if this motor plan is executed. It is proposed that if the actual sensory feedback matches the predicted state then awareness of initiation of movement will remain based on the predicted state. In this scenario awareness of performing a motor action is hence based on the predicted state, which is available before the movement is actually performed. This results in individuals being aware of the occurrence of their motor action around 50 to 100ms before they have actually moved (Libet, Gleason, Wright, & Pearl, 1983; Haggard, Newman, & Magno, 1999). Thus in Figure 5.1, awareness of motor actions (although not self-authorship) occurs at the time of predicted state generation, which temporally precedes actual performance of the action. If the action is self-produced then predicted sensory feedback should be cancelled out by reafference from the actual sensory feedback. If this occurs then there is perceptual sensory attenuation of the motor act, meaning that one does not feel or pay as much attention to the movement. If the actual movement does not match the predicted movement, due for example to a defective predicted state mechanism, then the predicted sensory feedback and actual sensory feedback signals will not cancel each other out.
Figure 5.1.
Forward model of motor control (adapted from Frith et al., 2000).
Frith et al. (2000) use this postulated mechanism to explain why an action may be actively experienced as performed by the self or passively experienced as performed by an alien ‘other’. Blakemore (2003) has detailed the mechanism through which we come to experience an action as authored by another. She claims that it is the predicted sensory feedback not matching and consequently not cancelling out the actual feedback, leading to greater activity in the parietal cortex, which makes the movement feel “externally controlled” (p. 651). Support for this assertion comes from the work of Spence, Brooks, Hirsch, Liddle, Meehan, and Grasby (1997), who suggest that over-activity of the parietal cortex may contribute to the feeling that willed actions are externally controlled in patients suffering from delusions of control.

The mechanism through which we come to experience an action as authored by ourselves has been detailed by Frith (2002). Frith claims that we get our awareness of authoring movements before the comparison has been made between the predicted and actual feedback. To argue for this, Frith draws on Wegner and Wheatley’s (1999) work showing that the temporal contiguity of a thought of what is about to happen (e.g., hearing the word ‘swan’) followed by it actually happening (e.g., finding that your hand has just moved a pointer to a swan) causes the emotion of self-authorship. Wegner (2002) calls this “apparent mental causation” (p. 64). Frith applies this to the forward model by suggesting that the “emotion” (Wegner, 2002, p. 325) of self-authorship is created when awareness of an action about to occur, based on the predicted state (available 50–100ms before we move as discussed above), is promptly followed by the actual action.

The forward model can be applied to deviations from the normal processes of action authorship in the following way. If the predicted state mechanism is malfunctioning, either through efference copy information not reaching it or through some other impairment, then
firstly Wegner’s mechanism of apparent mental causation cannot work, meaning that the actor does not feel the authorship emotion, even though the action is self-initiated. Secondly, the high level of parietal cortex activation (due to non-cancellation of predicted and actual feedback) is the same as if the movement were passive (i.e., caused by someone else). This gives the event the same ‘feel’ as a passive or externally-caused action, and the actor hence feels as though someone else caused the action.

The Application of the Forward Model to AVHs

Somewhat surprisingly Frith and colleagues have only applied their forward model to abnormalities involving overt actions such as delusions of control and the alien hand sign (e.g., Blakemore & Frith, 2003; Frith, 2002; Frith et al., 2000). Frith and colleagues have not attempted to use the forward model to explain phenomena not involving overt movements, such as AVHs. One reason for this may be criticisms (e.g., Gallagher, 2004; Stephens & Graham, 2000) of Frith’s previous attempt to apply his prior model to cognitions (Frith, 1992). We address these criticisms, and offer some possible responses to them, in the second part of this article.

Frith et al.’s (2000) model has, however, recently been applied to AVHs by Seal, Aleman, and McGuire (2004). Seal et al. begin by noting that any neurocognitive model of AVHs needs to account for how self-generated thought, misperceived as speech, is experienced as unintended. The highlighting of unintendedness as a defining feature of AVHs leads Seal et al. to apply the forward model to AVHs in a particular manner. Their application of the forward model to AVHs is founded on the assumption that inner speech is the primary material of AVHs. The involvement of inner speech in AVHs is accepted by many psychologists (e.g., Bentall, 2003; Fernyhough, 2004), and supported by empirical research such as Gould’s (1950) study showing that when patients hallucinated,
subvocalisations occurred which could be picked up with a throat microphone. That these
subvocalisations were causally responsible for the inner speech perceived in AVHs, and not
just echoing it, was suggested by the work of Bick and Kinsbourne (1987), who
demonstrated that if people experiencing hallucinations opened their mouths wide, stopping
vocalizations, then the majority of AVHs stopped.

Having made the assumption that inner speech is the primary material of AVHs,
Seal et al. next propose that, “once some trigger event brings about the generation of the
AVHs motor commands are issued and inner speech is produced” (p. 65). This makes the
(unexamined) assumption that inner speech may be conceptualized as an action, and that it
is therefore valid to apply the forward model to inner speech. Once this assumption has
been made, Seal et al. are then able to apply Frith et al.’s (2000) forward model to the
phenomenon of inner speech, postulating that the experience of unintendedness,
characteristic of AVHs, is due to the predicted sensory consequences of inner speech
initiation being distorted or absent. This distortion or absence of the predicted state means
that, when the actual sensory consequences of the inner speech command occur, there is no
predicted state to accurately attenuate it. As Seal et al. (2004) phrase it, “The experience of
unintendedness can be accounted for by failure of feed forward information” (p. 64). This
concept of the unintendedness of inner speech is part of Seal et al.’s proposal that the
failure of the predicted state leads to inner speech whose origins (self or other) is
experienced, “in various states of ambiguity rather than two separate states of awareness
(self/other)” (p. 65). The proposal is then made that top-down factors, such as attributional
biases, lead to the unintended inner speech being experienced as other-generated. This
mechanism is illustrated in Figure 5.2.
Figure 5.2
Seal et al.'s (2004) application of the forward model to AVHs.
We suggest, however, that this application of the forward model to inner speech is problematic. Contrary to Seal et al.’s assertions, the forward model outlined by Frith et al. does not state that the absence (or distortion) of the predicted state causes the experience of unintendedness, which is then resolved into a feeling of self or other authorship by preconscious attributions. Instead, as discussed above, the forward model has a two-part mechanism, in which the emotions of self-authorship or other-authorship of an action are determined by the Frith/Wegner and Blakemore mechanisms respectively.

Our alternative interpretation of how to apply the forward model to inner speech is shown in Figure 5.3. Firstly, as in Seal et al.’s model, the brain either produces a degraded predicted state or fails to produce a predicted state at all. The reasons for this are still poorly understood, but it seems likely that it will involve a particular neurological deficit, potentially modulated by stress. The consequences of failing to generate a coherent predicted state from the initial inner speech motor command are likely to be that awareness of performing inner speech cannot occur, as Libet et al.’s (1983) work suggests it would, 50–100ms before inner speech actually occurs. Consequently, Wegner’s (2002) mechanism of apparent mental causation (responsible for generating the emotion of self-authorship) is unable to operate. Instead, Blakemore’s (2003) mechanism of non-cancellation of predicted and actual feedback, leading to increased parietal cortex activation and hence the feeling of external control, would be operative, leading the event to be attributed to an external cause.

To summarise, Seal et al.’s application of the forward model (shown in Figure 5.2) proposes that a mismatch between predicted and actual state leads to an experience of unintended inner speech which is then resolved into self/other authorship by preconscious attributions. Instead we argue (see Figure 5.3) that the failure of the predicted state means that the emotion of self-authorship is not felt, and that instead the mismatch of predicted
Chapter 5: Thought as action

Figure 5.3

Revised application of the forward model to AVHs.
Chapter 5: Thought as action

and actual state leads to the experience of other-authorship of inner speech. This model, *contra* Seal et al., does not require that there be a feeling of unintendedness, the ambiguity of which must then be resolved into self/other authorship by preconscious attributions.

*Inner Speech as a Kind of Action*

One potentially problematic aspect of Seal et al.’s (2004) account is the unexamined assumption that it is possible to transpose Frith et al.’s (2000) model from overt actions to the process of thinking in inner speech. Gallagher (2004) has made a strong case that it is incorrect simply to transpose Frith’s forward model from its successful explanation of passivity experiences, involving overt actions, to cognitive phenomena such as AVHs where there is no overt behaviour. Gallagher suggests that any such arguments are based on the erroneous assumption that “thinking is a kind of action” (p. 6).

Gallagher’s suggestion is that Frith and colleagues’ use of the forward model makes sense for overt actions, as there are at least two clear reasons why a predicted state is needed for overt actions. Firstly, we need to know whether our actions are internally caused (i.e., if I lift my arm) or externally caused (i.e., if someone else lifts my arm). Secondly, if our action is not going to achieve our goal we need to know this in advance so we can make the necessary adjustments. Gallagher goes on to point out that if the forward model is applied to purely internal events such as thoughts, then these two reasons for having the predicted state no longer come into play. Firstly, why would we need to know whether a thought has been caused by us or by an external agent? As Frith and Done (1988) point out, if all our thoughts are internally generated, there is no possibility of our having thoughts other than our own. Stephens and Graham (2000) expand on this by pointing out that whereas we have to distinguish our actions from the actions of others, “we are never
confronted with the... problem of having to sort out our thoughts out from other people's thoughts” (p. 138).

However, the issue of how we judge the ownership of our thoughts makes more sense when inner speech and verbal thought are considered within their developmental context. A key figure in this respect is the developmental psychologist, L. S. Vygotsky (e.g., 1934/1987). He suggested that inner speech constitutes a form of verbal self-regulation that is derived from semiotically mediated exchanges with interlocutors in the social world. Dialogue that originally exists on the interpsychological plane, as an exchange between individuals, is reconstructed on the intrapsychological plane as inner speech or verbal thought. Generally speaking, Vygotsky's hypotheses about the development of inner speech (and its semi-covert precursor, private speech), have been supported by empirical research (Berk, 1992; Winsler, 2004).

The implications of these ideas about inner speech for the present discussion are, firstly, that thought has a social origin and, secondly, that the challenges of determining the ownership of one's utterances continue when those utterances are transferred to the intrapsychological plane. Indeed, a major cognitive challenge for children is to distinguish self-as-speaker from other-as-speaker (Femyhough & Russell, 1997). Femyhough (2004) has noted that a Vygotskian approach to inner speech, whereby the experience of alien voices is explained in terms of atypical patterns of internalization of external dialogue, can help to resolve the paradox that AVHs are both experienced as alien and at the same time acknowledged to be of the self (Leudar & Thomas, 2000).

Taking seriously the idea of ownership of thoughts would thus militate against Stephens and Graham's (2000) conclusion that a system that assigns ownership of thoughts is just an “ad hoc” (p. 138) creation devised solely to help scientists explain delusions of
control. Rather, a Vygotskian perspective on inner speech entails an assumption, similar to that made in other recent discussions of the 'extended mind' (Clark, 1997; 1998; Clark & Chalmers, 1998), that mental activity is not necessarily co-extensive with the boundaries of the biological organism. For present purposes, the implication of Vygotsky's ideas about the social origins of thought is that inner speech retains the dialogic quality of the external exchanges from which it derives (Fernyhough, 1996), and that thinking is therefore naturally permeated by other voices (Fernyhough, 2004).

A related question posed by Gallagher (2004) is whether the process of planning and executing an utterance in inner speech can be mistaken in the same way that it can for the generation of overt utterances. In other words, is there an internal, purely cognitive equivalent to the process of making subtle adjustments to an action plan? Although the relevant data for inner speech are necessarily lacking, due to the unobservability of such utterances, some interesting findings from the study of children's private speech (seen by Vygotsky to represent a way-station between external dialogue and verbal thought) are relevant to this question. Manfra, Tyler, Shiflett, and Winsler (2003) reported evidence that preschool children apply the same correction for speech errors and dysfluencies to their private speech as they do to their social utterances. If private speech is seen as continuous with verbal thought, as the Vygotskian approach entails, it suggests that similar corrections to inner speech might be plausible. In other words, it seems plausible that individuals can indeed act as though an utterance in inner speech is wrong, and needs adjusting.

A further objection raised by Gallagher's (2004) critique concerns the problem of unbidden thoughts. He notes that thoughts can strike us 'out of the blue' without us having a sense of agency, and yet we do not attribute these thoughts to someone else (as a person with psychosis may do). However, there is no evidence that it is these out-of-the-blue
thoughts that those diagnosed with psychosis experience as AVHs. Indeed, as Fernyhough (2004) noted, we know very little about the normal processes of inner speech and verbal thought in disorders such as schizophrenia. It may be, conversely, that these out-of-the-blue thoughts are responsible for the AVH-like experiences found in non-clinical populations, but not full-blown AVHs. In the model outlined above, thoughts that occur to us in this way should not be attributed to someone else, as a predicted state is still generated. Asking voice-hearers (both from the non-clinical and patient populations) whether, and under what conditions, they experience such unbidden thoughts will presumably shed further light on the role of predicted state formation in determining the ownership of utterances in inner speech.

One of the benefits of applying Frith’s forward model to inner speech, which Seal et al. do not note, is that this approach has the benefit of being immune to Akins and Dennett’s (1986) criticism of Hoffman’s (1986) influential account of AVHs. Hoffman (1986) proposed that AVHs were due to inner speech that was experienced as unintended, and tried to explain this unintendedness as due to problems with discourse planning. This led to Akins and Dennett’s objection that, if inner speech is intelligently planned by means of discourse plans, then there must be another intelligent entity planning the discourse plans, and so on, leading to an infinite regress. Frith’s forward model sidesteps this problem by not assigning any crucial role in the model to judgments about the unintendedness (or otherwise) of actions. Rather, the model (when applied to AVHs, as outlined here) proposes a direct causal mechanism leading from the malfunction of the predicted state to the experience of inner speech as being of alien origin, in the absence of any competing self-authorship emotion (see Figure 5.3). Failure of the predicted state leads to neurological activity associated with passivity experiences, which may indeed lead to inner speech being
experienced as unintended. However the unintendedness of the inner speech is a result of the failure of the predicted state and follows from the passivity experience itself. Thus unintendedness is a consequence, not a cause, of the AVH and hence does not play the problematic theoretical role that it does in Hoffman's (1986) model.

A number of further challenges for the present model need to be addressed by future research. One important issue is to pinpoint precise brain areas or networks involved in the generation of (and, in cases of damage, the failure to generate) the predicted state. Leube, Knoblich, Erb, Grodd, Bartels, and Kircher (2003) have suggested that the neurological instantiation of a deficit in the efference copy mechanism may be located not in specific brain areas, but rather may arise from defective interactions between perceptual and motor areas. These areas are likely to involve the cortical network that de Vignemont and Fourneret (2004) note as being involved in action attribution, which include the prefrontal and the parietal cortex, the supplementary motor area, and the cerebellum. However, current research in this vein has tended to focus on the role of the cerebellum in signalling the sensory discrepancy between the predicted and actual sensory consequences of movements (e.g., Blakemore, Frith & Wolpert, 2001) and not on the neural correlates of the predicted state itself. Another route of entry into understanding the neural underpinnings of the predicted state mechanism is via neuropsychological syndromes, such as the alien hand syndrome, which are associated with lesions in known brain regions. In the case of the alien hand syndrome, patients make involuntary yet seemingly purposeful limb movements (Giovannetti, Buxbaum, Biran & Chatterjee, 2005), suggesting a breakdown in the forward model, but without the feeling that their actions are caused by another. As this syndrome is associated with medial frontal and callosal lesions (Della Sala, Marchetti, & Spinnler,
1991), this identifies these as regions to be potentially incorporated into any neural instantiation of the forward model.

A further challenge, noted by Gallagher (2004), is the selectivity problem, which recognises the episodic nature of positive symptoms. That is, if the predicted state is malfunctioning, why do all thoughts not seem alien? The first point to note is that the predicted state may only intermittently be malfunctioning. This raises the further question of what is it that causes the predicted state to fail. A possible answer is that it could be the same factors that Bentall (1990) has described as affecting our judgements about whether an event is public or private: namely, factors such as stress-induced arousal, our ability to use cues, perceptual attenuation, or effects of suggestion. These remain areas for future empirical research. The second point relates to the fact, noted above, that we do not have sufficient understanding of normal inner speech in individuals who experience AVHs. Only a greater understanding of the phenomenology of inner speech in affected individuals will allow us to specify how voice-hearing may be determined by the breakdown of the predicted state mechanism.
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Chapter 6

A new spin on the Wheel of Fortune:

*Gender effects in priming of action-authorship judgments*

**ABSTRACT**

The proposal that there is an illusion of conscious will has been supported by findings that priming of stimulus location in a task requiring judgments of action-authorship can enhance participants’ experience of agency. We attempted to replicate findings from the ‘Wheel of Fortune’ task [Aarts, H., Custers, R., & Wegner, D. M. (2005). On the inference of personal authorship: enhancing experienced agency by priming effect information. *Consciousness and Cognition*, 14, 439-458.] and to investigate whether the effect of priming on judgments of action-authorship was of equivalent strength in men and women. We also examined participants’ performance on this task in relation to self-reported passivity experiences and hallucination-proneness. There was a significant effect of priming, with primes being found to increase the experience of agency. An interaction between gender and priming was also found, with further analysis indicating that priming enhanced feelings of agency in women but not in men. There were no significant correlations between levels of self-reported passivity experiences or hallucination-proneness and participants’ susceptibility to the priming effect on ratings of agency. Implications of these findings are discussed with regard to a prominent model of passivity experiences. Possible explanations of the observed gender effect are also considered.

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Chapter 6: The Wheel of Fortune

Introduction

The feeling of having consciously willed something to happen is a fundamental human experience. Wegner (2002, 2004) has proposed that this experience of action-authorship could be a result of the same mental processes we use in the perception of causality more generally. As Hume (1739) noted, the constant conjunction of $x$ followed by $y$ leads us to infer that $x$ caused $y$. Wegner (2004) translates this argument, typically used to explain causality for mechanistic events (the paradigmatic example being the collision of billiard balls), into the realm of mental phenomena. In essence Wegner proposes that, just as we experience a moving billiard ball as causing the movement of a stationary ball with which it collides, we experience ourselves as causal when an action is preceded by a relevant forethought about the action. This leads us to experience ourselves as causal agents when “we have experienced relevant thoughts about the act at an appropriate interval in advance, and so can infer that our own mental processes have set the act in motion” (p. 654). Wegner terms this the theory of apparent mental causation: “people experience conscious will when they interpret their own thought as the cause of their action” (Wegner & Wheatley, 1999, p. 480). Wegner describes three aspects of thought–action contiguity that determine the extent to which mental causation is felt. These are the priority, consistency, and exclusivity of the thought about the action (Wegner & Wheatley 1999). For the perception of apparent mental causation, the thought should occur before the action, be consistent with the action, and not be accompanied by other potential causes.

An important implication of this theory is that people may experience the feeling of doing something when there is actually no causal connection between their thoughts and their actions. Indeed, as Wegner notes, there may be a third factor causing both the thought
and the action, with no causal linkage from the thought to the action. It is this that has led Wegner (2002, 2004) to talk of the "illusion" of conscious will.

Priming and the artificial enhancement of conscious will

The potential for our feeling of conscious will to be illusory, and show no relation to the actual mechanisms by which our actions have been generated, has been empirically demonstrated by Wegner and colleagues (e.g., Wegner & Wheatley, 1999; Aarts, Custers, & Wegner, 2005). At the heart of such experiments is the idea that briefly priming an effect of an action (by priming a thought/representation of an action's consequences just before it occurs) increases the probability that one will experience that effect as due to one's own volition.

One methodology created by Wegner and colleagues to investigate this phenomenon has been dubbed the 'Wheel of Fortune' task (Aarts et al., 2005). In this task, participants are told that they control the motion of a grey square rapidly traversing a rectangular grid whilst a computer independently moves another square in the opposite direction at the same speed. At a certain point participants are instructed to press a key to stop the motion of their square. They are then shown a new grid with one square highlighted and they must judge whether their key-press caused their square to land there, or whether that was where the computer had caused its square to stop. The high velocity at which the squares traverse the grid result in this decision being ambiguous. This ambiguity regarding who caused the square to land on the final highlighted position (i.e., whether it was the participant's button press that stopped their square there, or whether it was where the computer's square finished) allows the participant's experience of authorship over the observed stops to be experimentally manipulated. In primed trials, the position of the final
Chapter 6: The Wheel of Fortune

square is briefly presented to participants (for 34ms) before they see the final square’s position, which is in fact predetermined. This is termed an ‘effect-prime’, as it primes the judgment about the apparent effect of the participant’s button press (Aarts et al., 2005). Wegner’s theory of apparent mental causation leads to the prediction that, in effect-primed trials, the occurrence of the thought/representation of the square’s position, followed by the perception of it being there, will increase participants’ feeling of conscious volition of stopping the square, compared to a control condition where no prime is present.

Aarts and colleagues have published three papers demonstrating that the provision of an effect-prime does increase the experience of conscious will (Aarts, 2007; Aarts et al., 2005; Aarts, Wegner, & Dijksterhuis, 2006). However, this methodology has yet to be replicated by other research groups. The first aim of the current study was therefore to provide an independent replication of Aarts et al.’s findings. We were also interested in examining whether the magnitude of this effect was similar in both genders as, unusually, Aarts and colleagues have neither reported the gender compositions of their samples, nor analyzed the effect of effect-primes by gender. Given that Wegner (2004) implies that the mechanism that generates the “illusion of conscious will” (p. 649) is likely to be a universal, non-gender-specific, human phenomenon, foregoing an analysis by gender is understandable.

There are nonetheless reasons to believe that a gender effect on the susceptibility to effect-primes may emerge through appropriate analyses. Firstly, as Wegner and Wheatley (1999) themselves note, the experience of conscious will is an expression of our tendency to take what Dennett (1987) terms the ‘intentional stance’. This, as Wegner and Wheatley state, involves viewing “psychological causation not in terms of causal mechanism but rather in terms of agents who have beliefs and desires that cause their acts. Conscious will
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is part of taking an intentional stance towards oneself” (p. 490). However, Baron-Cohen (2002, 2003, 2005) has argued that the “drive to identify another’s mental states” (2005, p. 820), which he terms of the skill of empathizing, is greater in women than men. This argument has received substantial empirical support (e.g., Davis, 1994; Hall, 1978; Happé, 1995; Tannen, 1990). It hence seems plausible that the greater propensity in women to take the intentional stance towards others may lead to an equivalent propensity to overrate the role of thoughts in causing their own actions. In terms of the Wheel of Fortune task, this led us to predict that, whereas both men and women were likely to have greater self-authorship ratings on effect-primed (as compared to non-primed) trials, the magnitude of this effect would be greater in women due to their propensity to overrate the role of thoughts induced by effect-primes in causing the observed event.

Applying the Wheel of Fortune task to questions of psychopathology

In addition to explaining typical judgments of action-authorship, Wegner and colleagues’ theory of apparent mental causation has been used to account for disturbances in action-authorship judgments associated with various forms of psychopathology (e.g. Frith, 2002; Jones & Fernyhough, 2007a). For example, patients with schizophrenia often suffer from what are termed passivity experiences. In such experiences patients feel as if their actions have been caused, or are under the control of, another agency. For example, a patient reported by Mellor (1970) describes how his “fingers pick up the pen, but I don’t control them. What they do is nothing to do with me” (p. 18). Frith and colleagues (Blakemore, 2003; Frith, 2002; Frith, Blakemore, & Wolpert, 2000) have attempted to explain such experiences as resulting from a breakdown in the mechanism responsible for

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8 This idea was suggested to me by Lee de-Wit.
Chapter 6: The Wheel of Fortune

generating the emotion of authorship (see Chapter 5). In their model, the issuing of a motor command is accompanied by the production of a parallel efference copy of the motor command which is then used by a predictor mechanism to create a prediction (the 'predicted state') of what the movement will result in. Awareness of starting a movement appears to be based on this predicted state and not on sensory feedback from movements themselves. In experiments investigating the time at which a self-generated movement is perceived as starting, it is found that individuals experience their movement as starting ~50–80ms before their actual movement begins (Haggard, Newman, & Magno, 1999).

Frith (2002) draws on Wegner's (2002) concept of apparent mental causation to explain how the emotion of self-authorship is created when awareness that an action is about to occur (based on the predicted state, which is available 50–80ms before we move), is promptly followed by the actual action (see Jones & Fernyhough, 2007a). If the predicted state mechanism malfunctions, then Wegner's mechanism of apparent mental causation cannot work, meaning that the actor does not feel the authorship emotion. It is this malfunction of the predicted state that has been proposed to be the source of passivity experiences (Frith, Blakemore, & Wolpert, 2000). In addition to its involvement in the generation of passivity experiences, a malfunction of the predicted state has been proposed to play a causal role in the genesis of auditory verbal hallucinations (AVHs). In such models, a failure in the predicted state has been proposed to cause self-produced speech to be experienced as not produced by the self, resulting in an AVH (e.g., Jones & Fernyhough, 2007a; Seal, Aleman, & McGuire, 2004).

Although Frith and colleagues are not clear on this point, there are at least two potential ways in which the predicted state mechanism could lead to a breakdown in the feeling of authorship. Firstly, there may be a problem upstream of the predicted state,
resulting in a complete or partial failure to generate a predicted state. Secondly, there may be a problem downstream of the predicted state. In this latter case, the creation of the predicted state may be normal, but the individual may be unable to use this information to generate the feeling of agency. The Wheel of Fortune procedure offers a potential method of distinguishing which of the above possibilities is the best explanation of passivity experiences and AVHs. We propose that effect-primes may be seen as proxy predicted states, in that they give a preview of what is about to occur. If there were indeed a problem downstream of the predicted state, meaning that individuals could generate it but not use it, then individuals with passivity experiences or AVHs should be impaired in their ability to use the effect-primes on the Wheel of Fortune task to increase their feeling of authorship. This would result in a negative correlation between susceptibility to both passivity experiences and AVHs and the increase in the feeling of authorship on effect-primed trials. A failure to find a correlation would be in line with the prediction of the upstream explanation, namely that such experiences result from a lack/breakdown of the predicted state, and the ability to use the predicted state (prime) should hence not be related to levels of such experiences.

One obvious way of testing these potential explanations would be to employ the Wheel of Fortune procedure in examining action-authorship judgments in patients with schizophrenia who have passivity experiences, or in patients who experience AVHs. Another strategy, which we adopt here, is to use measures of susceptibility to passivity experiences and AVHs in a non-clinical sample. Measures of general delusionality in the healthy population often include items relating to passivity experiences. For example, the Peters et al. Delusions Inventory (PDI-21; Peters, Joseph, Day, & Garety, 2004) includes items relating to passivity experiences such as "Do you ever feel as if you are a robot or
zombie with no will of your own?" (item #21). This item was found to be endorsed by 6.3% of a student sample by Jones and Fernyhough (2007b). This provides evidence that, just as hallucinations and persecutory delusions have been found to exist on a continuum stretching into the healthy population (Johns & van Os, 2001; General Introduction), passivity experiences may also exist on a continuum. In order to test the specificity of any observed relation, we also set out to examine associations between Wheel of Fortune performance and susceptibility to another form of delusional ideation, not linked to problems with the predicted state and feelings of authorship, namely persecutory delusions.

In summary, this study set out to investigate the following hypotheses. Firstly, we hypothesized that authorship ratings on the Wheel of Fortune task would be higher on effect-primed trials than non-effect-primed trials. We also made the second hypothesis that although the effect of priming would be significant in both men and women, the magnitude of this effect would be greater in women, resulting in an interaction between gender and priming. Thirdly, we hypothesized that self-reported susceptibility to both passivity experiences and AVHs would correlate negatively with increases in the feeling of action-authorship resulting from exposure to effect-primes. Our final hypothesis was that self-reported persecutory delusion-like beliefs would not correlate with increases in the feeling of action-authorship resulting from exposure to effect-primes.

Method

Participants and design

A convenience sample of 144 students (62 men, 82 women) with mean age of 21.5yrs ($SD = 3.91$) participated in the experiment. Participants were tested in two groups in order to assess whether there was an effect of order of testing. In Group 1, 92 participants (40 men, 52 women) first completed the three questionnaires described above,
recorded their basic demographic data, including their gender, and then undertook the Wheel of Fortune task, using laptop computers in a quiet room. Participants were not paid for their participation. In a within-participants design, participants experienced trials on which they were primed with effect information (the final location of the square) and trials in which they were not primed. In Group 2, the remaining 52 participants (22 men, 30 women) firstly undertook the Wheel of Fortune task, using desktop computers in isolated cubicles, and then completed the questionnaire portion of the study and recorded their demographic details including gender. Again, in a within-participants design, participants experienced trials on which they were primed with effect information (the final location of the square) and trials in which they were not primed.

Measures

Passivity experiences. A new 5-item questionnaire was developed for the purpose of assessing levels of passivity experiences in a healthy population sample (see Appendix C), as there is no existing tool that attempts to measure these specific experiences in non-clinical samples. This was termed the Scale for Assessment of Passivity Experiences in the General Population (SAPE-GP). The items constituting this were taken in part from the Peters et al. Delusions Inventory (Peters et al., 2004) and in part adapted from the Scale for the Assessment of Positive Symptoms (Andreasen, 1984). Items were scored on a 5-point Likert scale with potential responses being: Always (4), Very Often (3), Fairly many times (2), Occasionally (1) and Never (0). Total scores could hence range from 0 to 20, with higher scores indicating more passivity experiences.

LSHS-R: The revised Launay-Slade Hallucination Scale (Launay & Slade, 1981, as modified by Bentall & Slade, 1985) is a 12-item instrument designed to measure predisposition to hallucination-like experiences. Each item is scored on a five-point Likert
scale consisting of: “certainly applies to me” (4), “possibly applies to me” (3), “unsure (2),
“possibly does not apply to me” (1), “certain does not apply to me” (0). Total scores can
range from 0 to 48. Higher scores indicate a greater predisposition to hallucination-like
experiences (Chapter 3; Chapter 4).

Persecutory delusion-like beliefs. These were assessed using the Persecutory
Ideation Questionnaire (PIQ; McKay et al., 2006), a 10-item questionnaire designed to
measure persecutory ideation in both clinical and non-clinical samples. Items are rated on a
5-point Likert scale ranging from Very True (4) to Very Untrue (0), with higher scores
meaning that individuals experienced greater levels of persecutory ideation. This measure
has been shown to have good reliability and validity (McKay et al., 2006; see Chapter 1).

Experimental task and procedure

The Wheel of Fortune task was programmed in C+ using the Borland builder
environment. Precise control of timing was ensured with the use of Direct X.
Administration and scoring of the task was exactly as reported in Aarts et al. (2005).
Participants were informed that the study was designed to examine feelings of control. The
participants had to press and hold the S-key on the keyboard to cause a grey square rapidly
to traverse a rectangular path, consisting of eight white squares, in a counter-clockwise
direction. At the same time, another grey square (under the computer’s control) moved
along the same path but in a clockwise direction. At a random point in time (between 8 and
10 laps of the screen) the grid went blank and participants had to stop the motion of the
squares, which they were told continued to rotate invisibly, by pressing the Enter-key. This
resulted in one of the white squares of the grid turning black, which was said to represent
the position of either their own square, or the computer’s, at the time they pressed stop.
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After each stop, participants were asked to indicate whether they had caused the square to stop on that position or whether the computer had. The authorship judgment was measured on a 10-point Likert scale, running from ‘not me at all’ (1) to ‘absolutely me’ (10). The stop location was presented twice on each of the eight tiles of the path. The experimental task thus consisted of 16 trials. The order of primed and unprimed trails was randomly specified for each participant. Before the experimental phase proper, participants were given two practice trials to familiarise themselves with the procedure. These data were not used in the analyses reported here.

Events in a trial. Each trial began with a warning signal (the word ‘Warning’ appeared on the screen). Next the message ‘Start’ appeared in the centre of the grid until the participant pressed (and held) the S-key. The participant’s and the computer’s squares then began to move along the path in alternate steps (the squares were presented one after the other). Squares were displayed for 67ms on each position. It hence took 1072ms for one complete lap to occur (67ms x 8 positions x 2 [participant’s and computer’s] squares; Aarts et al., 2005). The number of laps that occurred in a trial was programmed to vary randomly between 8 and 10 laps. When the message ‘Stop’ appeared in the centre of the grid, only the eight white empty tiles were shown, until the participant pressed the Enter-key. At the press of this button a black square was presented after 101ms. The placement of this square was always four positions farther than that of the last position of the participant’s square before the stop message had appeared. Thus, participants did not have actual control of where the black square landed.

Effect-priming. In 8 of the 16 trials the black square that was to be displayed was flashed on the screen before the message ‘Stop’ appeared. The primed location was always the same as the subsequently presented position of the black square. The prime occurred
33ms after the last presentation of the participant's square. Primes were presented for 33ms and were followed 51ms later by the message 'Stop' (the total priming period hence being 117ms). In the no-priming condition, the position of the black square was not flashed (the position was presented in white for 33ms). The priming event was employed for every possible location on the 8 square grid, resulting in eight trials of the priming condition, and 8 of the no-priming condition.

Measurement of potential control. Participants' potential control over producing the effects was also measured. The computer measured participants' reaction times (in ms) between the word 'Stop' appearing on screen, and their press of the Enter-key. As Aarts (2007) notes, if the participants press the Enter-key 330ms (equivalent to 369ms using our timings) after the presentation of the stop message, then they will have pressed it at the same time that their square was actually in the position indicated by the black square. A measure of potential control was hence calculated as the absolute difference between the participant's reaction time (time of Enter-key press less time of presentation of the word 'Stop') and 369ms. Most participants on most trials stopped the motion of the squares before one lap of the squares could be completed. For slower responses the response times first had the time of the completed laps (1072ms for each lap) subtracted from it, then the absolute difference between this figure and 369ms was calculated (Aarts et al., 2005). The smaller the absolute difference in these numbers, the more likely it was that participants could actually have caused the square to land on that position. If there are no differences in this figure between primed and non-primed trials, it rules out the possibility that participants may have rated their authorship higher on primed trials due to them actually landing their square closer to the final position of the black square on such trials.
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A number of individual trials resulted in participants showing high reaction times. This was interpreted as the participant losing concentration on the task or hitting the wrong button to stop the motion of the squares. As such, any trials on which participants’ reaction times were greater than 2144ms (two laps of the invisible squares) were removed from the data. This eventually led to the removal of a total of 104 individual trials (Group 1: 90 removed, Group 2: 14 removed) representing 5% (Group 1: 6%, Group 2: 2%) of the total trials performed by participants.

Debriefing. At the end of the session participants were debriefed. The debriefing indicated that none of participants realised the true nature of the study. Previous studies utilising this task (e.g., Aarts, 2007) with a 34ms prime duration have noted that none of their participants reported having seen the primes. In our replication, one participant confidently reported having seen a black square flash on around half of the trials and further noted that the position of this square was identical to the location of the final square. This participant’s data were excluded from the analysis, as their experience of the task was clearly not comparable to the other participants. A minority of participants (<10%) reported having seen a flash on the screen on a few trials, but they were unaware that the position of this square matched the position of the final black square.

Results

Authorship ratings and effect-priming

Descriptive statistics are presented in Table 6.1. The average ratings of experienced authorship across the 16 trials were analysed using a 2 (Prime: present, absent) x 2 (Order of testing: Group 1, Group 2) x 2 (Gender: male, female) mixed-design ANOVA. A Kolmogorov-Smirnov test indicated that the ratings of experienced authorship on both
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Table 6.1
Descriptive statistics for variables under investigation

<table>
<thead>
<tr>
<th></th>
<th>Overall (N = 143)</th>
<th>Men (n = 61)</th>
<th>Women (n = 82)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Authorship rating (effect-prime)</td>
<td>5.35 (1.52)</td>
<td>4.92 (1.41)**</td>
<td>5.67 (1.52)**</td>
</tr>
<tr>
<td>Authorship rating (no effect-prime)</td>
<td>4.74 (1.53)</td>
<td>5.17 (1.67)**</td>
<td>4.42 (1.33)**</td>
</tr>
<tr>
<td>Passivity experiences</td>
<td>2.00 (2.74)</td>
<td>2.60 (3.78)*</td>
<td>1.61 (1.68)*</td>
</tr>
<tr>
<td>Hallucination-proneness</td>
<td>18.09 (7.39)</td>
<td>16.90 (7.04)</td>
<td>18.86 (7.56)</td>
</tr>
<tr>
<td>Persecutory Ideation Questionnaire</td>
<td>7.85 (5.70)</td>
<td>7.54 (4.57)</td>
<td>8.05 (6.36)</td>
</tr>
</tbody>
</table>

*p < .05, **p < .01

Note. Passivity experience and persecutory ideation questionnaire data was unavailable for eleven participants.

effect-primed and non effect-primed trials did not deviate significantly from a normal distribution, \( D (143) = .05, p > .05 \) for both. The main effect of priming was significant, \( F (1, 139) = 7.44, p < .01, \eta^2 = .05 \). This effect size is small to medium by Cohen's (1988) criteria, and is comparable to the effect size for effect-priming of \( \eta^2 = .09 \) found by Aarts (2006). The interaction between priming and gender was also found to be significant, \( F (1, 139) = 16.96, p < .001, \eta^2 = .11 \). By Cohen's (1988) criteria, this is a medium to large effect size. There was no interaction between priming and group, \( F (1, 139) = .49, p = .49 \), nor between priming, group, and gender, \( F (1, 139) = 1.01, p = .32 \). Finally, there was no main effect of gender, \( F (1, 139) = .03, p = .87 \), or group, \( F (1, 139) = .01, p = .94 \).

To investigate the interaction between priming and gender, paired t-tests were performed separately for each gender on the authorship ratings for primed and non-primed...
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trials. For men, authorship ratings for the primed and non-primed trials did not differ significantly, \( t(61) = -0.85, p = 0.40 \). For women, authorship ratings were significantly higher for the primed than the non-primed trials, \( t(82) = 5.37, p < .001 \), Cohen’s \( d = .59 \). This is a large effect size as defined by Cohen’s (1988) criteria for paired t-test effect sizes.

Finally, as shown in Table 6.1, there was a significant difference in authorship ratings on both primed trials and non-primed trials between men and women, with men scoring significantly higher than women on non-primed trials, and significantly lower on primed trials.

Potential control

The mean absolute differences between the response time after the Stop message and the time required to land exactly halfway through the presentation of the black square (see Section 2.5) were compared for the 8 primed trials and the 8 non-primed trials using a 2 (Prime: present, absent) x 2 (Order of testing: Group 1, Group 2) x 2 (Gender: male, female) mixed-design ANOVA. It was found that there was no effect of priming, \( F(1, 139) = .08, p = .77 \), indicating that priming did not affect participants’ potential control over causing the effects. Furthermore, there was also no interaction between either gender, \( F(1, 139) = .13, p = .72 \), or group, \( F(1, 139) = .26, p = .61 \), with priming, and no three-way interaction between priming, group, and gender, \( F(1, 139) = .17, p = .68 \). There was no main effect of gender, \( F(1, 139) = 1.73, p = .19 \), although there was a main effect of group, \( F(1, 139) = 7.49, p < .01, \eta^2 = .05 \), with reaction times significantly quicker in Group 2. Overall the mean absolute difference score was 94ms (\( SD = 83 \)).
Passivity experiences, persecutory ideation, and effect-priming

 Cronbach’s alpha for the SAPE-GP was 0.73, indicating satisfactory internal reliability. Levels of passivity experiences reported were exceedingly low. Only thirteen participants scored five or over on the SAPE-GP. As shown in Table 6.1, men had significantly higher levels of passivity experiences than women. Correlations among passivity experiences, hallucination-proneness, persecutory delusion-like beliefs, and the difference in authorship between primed and non-primed trials (average primed authorship rating less average non-primed authorship ratings for each participant) are reported in Table 6.2. There were no significant correlations among the variables on either the entire data set or when analysed by gender. There was a trend towards females (but not males) showing a significant correlation between hallucination-proneness and effect-priming ($p = .09$). Greater hallucination-proneness was associated with lesser susceptibility to effect-primes in women. However, due to the number of correlations performed, it appears likely that to conclude there is a relation between hallucination-proneness and effect-priming in women would be to make a Type I error.
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Table 6.2

Correlations between delusions and effects on authorship of priming

<table>
<thead>
<tr>
<th></th>
<th>Passivity experiences</th>
<th>Hallucination-proneness</th>
<th>Persecutory ideation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall effect of priming (N = 132)</td>
<td>-.02</td>
<td>-.09</td>
<td>.10</td>
</tr>
<tr>
<td>Effect of priming in men (n = 52)</td>
<td>.09</td>
<td>-.08</td>
<td>.01</td>
</tr>
<tr>
<td>Effect of priming in women (n = 80)</td>
<td>-.04</td>
<td>-.19*</td>
<td>.13</td>
</tr>
</tbody>
</table>

Note. Passivity experience and persecutory ideation questionnaire data were unavailable for eleven participants. * p = .09

Discussion

This study aimed to replicate the Wheel of Fortune methodology as devised by Aarts et al. (2005). In concordance with our first hypothesis we found a significant main effect of effect-priming on this task. Participants reported greater feelings of authorship on primed compared to non-primed trials. Our second hypothesis, that there would be a significant interaction between gender and priming, was also supported. Further analysis showed that the direction of our second hypothesis, namely that women would show greater effects of priming on authorship ratings than men, was also supported. However, our second hypothesis also predicted that although the magnitude of the effect in women would be greater than in men, both would still show a significant effect of priming. This was not supported, as it was found that only women showed an effect of priming on authorship ratings, with authorship ratings higher on primed than non-primed trials.

It was also found that men reported significantly higher authorship ratings than women on non-primed trials, yet lower authorship ratings than women on primed trials.
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The higher authorship ratings by men on non-primed trials may potentially be explained by the greater perceived self-efficacy on computer-based tasks experienced by men (Busch, 1995; Durndell, Haag, & Laithwaite, 2000). On primed trials this greater male perceived self-efficacy may have been offset by women’s (but not men’s) greater susceptibility to priming, resulting in lower authorship ratings for men than women.

We had also hypothesised that levels of passivity experiences and AVH-proneness would correlate with participants’ susceptibility to effect-primes. There was no significant correlation between participants’ (of either gender) susceptibility to effect-primes and either levels of self-reported passivity experiences or AVH-proneness. As predicted, no correlation was found between participants’ susceptibility to effect-primes and their levels of persecutory delusion-like beliefs.

Effect-priming and gender effects

Our finding of an effect of priming in women but not in men is thus an intriguing finding which requires some discussion. One possibility is that we found an effect of priming in women but not in men due to the smaller number of men than women used in the present study, and the subsequent lower power of the study when examining men. However, if the effect size of priming in men had been equivalent to that found in women in the present study (d = .59), then the sample size of men would have resulted in a power in excess of β = .80, ample to detect an effect.

If this finding is not due to a lack of power, it raises the question as to whether our data can be reconciled with the previous Wheel of Fortune experiments by Aarts and colleagues. All of these previous studies which reported a main effect of effect-primes on the Wheel of Fortune task (Aarts, 2007; Aarts et al., 2005, 2006) failed to report splits of
their data by gender or include gender in their ANOVAs as a between-subjects variable. It is possible that these studies would have found a gender effect had they analysed their data in this way. Furthermore, we may speculate that, as the series of studies by Aarts and colleagues have all used student populations, these potentially consisted of a large number of psychology students. Psychology students are predominantly women: for example, 81% of students accepted onto a Psychology degree in the United Kingdom in 2006 were women (UCAS, 2007), and similar gender compositions are seen in studies that report gender splits on samples drawn from psychology departments in other European countries (e.g., Bohne et al., 2002). Since Aarts (2007) drew his Wheel of Fortune sample from Dutch undergraduates, it is probable that there was a preponderance of women in this sample. Thus it may be the case that the overall effect of effect-priming Aarts et al. reported is due to the effect of priming in women offsetting the lack of a priming effect in men. This would be consistent with our findings.

Although we have argued that our findings can be explained in terms of the greater propensity to take the intentional stance in women compared to men, and that it is not an artifice resulting from a lack of power, other alternative explanations are also possible. It could be argued that gender differences in perceptual abilities could be the cause of the present findings. For example, in an experiment examining the impact of odour priming on the ability to recognize a target word, only women (and not men) were found to show an effect of priming (Hermans, Baeyens, & Eelen, 2004). The authors of the study ascribed this gender effect to the superior olfactory perceptual abilities of women (Doty, 1991). Given that the Wheel of Fortune task involves a significant spatial component, and a prime which varies in its spatial location, it could be argued that a difference between men and women in susceptibility to spatial primes could have caused the present pattern of results.
However, the findings of a study by Koshino, Boese, and Ferraro (2000) on the efficacy of spatial primes suggests that such a differential susceptibility is not likely to exist. In this study the letter $O$ was presented on a screen in one of four possible locations. Once it appeared on screen participants had to identify its location as quickly as possible. Brief priming of the target’s location decreased participants’ reaction times for identifying its position, but with no gender difference in the magnitude of the priming effects.

Notwithstanding the above arguments, at the present time we are unable to speculate in any detail as to the precise causal mechanism that may lead to gender differences in the propensity to take the intentional stance to result in the effect found in this study. We would hence call for our findings to be replicated by other studies using the Wheel of Fortune methodology, perhaps including independent measures of propensity to take the intentional stance (de-Wit, Fernyhough, & Jones, 2006). Although we have argued that differential sensitivity to spatial primes is unlikely to be a cause of the current findings, it would also prove interesting to investigate the artificial enhancement of conscious will using experimental methods that do not use spatial primes. One potential method for this is the I-Spy methodology of Wegner and Wheatley (1999), which involves auditory rather than spatial priming. If the gender differences reported here were also obtained on the I-Spy paradigm, it would be a clue that our findings are due to gender differences in general susceptibility to primes in action-authorship paradigms, rather than to any specific susceptibility to spatial primes. However, the only existing study utilising the I-Spy paradigm (Wegner & Wheatley, 1999) has not reported on gender effects within the data. It would hence be desirable for any replication of the I-Spy experiment to analyse the data by gender to investigate possible interactions between gender and priming.
Chapter 6: The Wheel of Fortune

We also note here that, whereas Aarts and colleagues (e.g. Aarts, 2007; Aarts et al., 2005) reported none of their participants consciously saw their 34ms prime, approximately 10% of our participants reported being aware at some level of seeing our comparable 33ms prime. However, we do not anticipate this will affect the nature of our conclusions because Aarts et al. (2005: Experiment 2) have previously demonstrated that priming is effective whether it is induced by brief (34ms) primes which participants do not report being aware of, or longer (68ms) primes (visible to 44% of participants).

Passivity experiences, AVH-proneness, and effect-priming

The finding that susceptibility to priming on this task was unrelated to self-reported passivity experiences provides preliminary evidence that it is not an inability to use the predicted state that leads to passivity experiences, but rather its absence altogether. One alternative explanation might be that the passivity experiences scale devised for this study was not sensitive or reliable enough to detect relevant differences in a non-clinical sample, and we would call for further work in developing such scales. Another alternative might be that passivity experiences are not sufficiently common and/or intense in such a sample for any such relations to obtain. Given the low endorsement rates of items on the SAPE-GP, this appears a likely explanation. Only the replication of the present findings with a clinical sample, and clinically proven assessments of passivity experiences, would allow any firm conclusions on this issue to be drawn.

Similar arguments apply to the finding that susceptibility to priming was unrelated to AVH-proneness. As items measuring hallucination-proneness were endorsed to a wider extent than those assessing passivity experiences, this offers firmer evidence for an upstream impairment in the predicted state than the passivity data. However, again we
would call for the replication of these findings with both clinical and non-clinical samples in order to allow any firm conclusions to be drawn.

Conclusions and future directions

In summary the present study firstly points to the need for further examination of potential gender effects in the artificial enhancement of the experience of action-authorship. The initial findings reported here suggest that previously documented gender differences in the tendency to take the intentional stance may extend to the generation of our feeling of volition. This also supports the more general idea that the generation of our own feeling of conscious will involves taking the intentional stance towards our own minds (de-Wit et al., 2006; Frith, 2002). We have also suggested that it is possible to use brief effect-primes to mimic the predicted state proposed as part of Frith et al.'s (2000) forward model. Such a methodology should potentially allow a range of future experiments to explore further the mechanisms behind passivity experiences and hallucination in clinical populations.
Chapter 6: The Wheel of Fortune

References


Chapter 6: The Wheel of Fortune


HALLUCINATIONS AND INNER SPEECH
Chapter 5 highlighted the potential for inner speech to be the raw material of auditory verbal hallucinations (AVHs), and noted how a Vygotskian approach to inner speech can help inform our understanding of the role inner speech may play in AVH formation. The next two chapters involve a detailed consideration of the role that inner speech may play in AVHs.

Firstly, as noted in detail in Appendix B, it has been proposed that electrophysiological methods can be used to assess whether a disconnection between inner speech production and reception areas in the brain (termed a corollary discharge mechanism) is faulty in those who hear voices. Chapter 7 reports an electrophysiological study of event-related potentials in response to sounds in healthy participants to assess whether such arguments are likely to be valid.

Chapter 8 then considers in detail the plausibility of a role for inner speech in AVHs. Although numerous neurological and electrophysiological studies of those with AVHs have been performed (see Appendix B for a review), what I term an argument-from-phenomenology has been sidelined. Specifically, the chapter addresses whether the phenomenology of inner speech is consistent with the phenomenology of all, some, or no AVHs. Based on this idea, Chapter 8 considers whether inner speech- and memory-based models are a good fit to the phenomenology of the experience, and explores implications for the future study of AVHs.
Chapter 7: Electrophysiological study of inner speech

Chapter 7

The N1 event-related potential in response to sound during inner speech: Disentangling attentional demands from evidence of corollary discharge mechanisms

ABSTRACT

Auditory verbal hallucinations (AVHs) have been proposed to be caused by a failure of the corollary discharge system involved in inner speech production. Evidence that patients with schizophrenia with AVHs show reduced dampening of the N1 event-related potential component in response to external sounds when performing inner speech has been taken to be indicative of malfunctioning of this corollary discharge system. This article reports an investigation into the N1 component in response to sounds during a range of inner speech tasks, as well as a non-verbal control task (mental rotation), in healthy individuals (N =10). It was found that the N1 response to sound was dampened during subvocal rehearsal, dialogic inner speech, auditory verbal imagery, and mental rotation, as compared to a silent baseline. The dampening of the N1 found during the mental rotation control task is taken as evidence that dampening during inner speech tasks is likely due to the attentional demands of the task. The dampening of the N1 during inner speech tasks may therefore not be an indication of the activation of a corollary discharge system. Implications for inner-speech models of AVHs are explored.
Chapter 7: Electrophysiological study of inner speech

Introduction

Many prominent models of auditory verbal hallucinations (AVHs) propose that the raw material for such experiences is the voice-hearer's own inner speech. It has been proposed that self-produced inner speech may be experienced as alien due to a breakdown in a postulated neurocognitive action self-monitoring system (NASS; Blakemore, Wolpert, & Frith, 1998; Jones & Fernyhough, 2007; see Chapter 5). In brief, this model proposes that a failure to predict the consequences of inner speech production leads to a discrepancy between the predicted and actual neural antecedents of inner speech, resulting in inner speech being experienced as alien (Jones & Fernyhough, 2007; see Chapter 5). In a related formulation, Ford and colleagues (Ford et al., 2001a, 2001b; Ford & Mathalon, 2005) have proposed that the reason that such inner speech may come to be experienced as alien is due to a breakdown in a postulated corollary discharge system. This system is proposed to work through neural speech production areas sending efference signals to speech reception areas to dampen their activity, resulting in such speech being experienced as self-produced. Ford et al. (2001a) propose that a disconnection between these areas, and a resultant breakdown in the corollary discharge system, may result in self-produced inner speech being experienced as alien, resulting in an AVH.

Ford and colleagues (2001a, 2001b) have attempted to test the hypothesis that a deficit in the corollary discharge mechanism is a cause of AVHs through a specific electrophysiological technique involving event-related potentials. Electrophysiological methods take advantage of the fact that neighbouring neurons in the brain frequently fire together, creating potentials which can be detected by electrodes placed on the scalp. Event-related potentials (ERPs) are changes in voltage resulting from neural activity which occurs at a particular time after a specific event, such as an external stimulus (Luck, 2005).
As ERPs are relatively small (1–30μV) compared to background cortical activity, participants must be repeatedly exposed to a procedure, so that an average ERP can be calculated. The resultant ERP waveform consists of a number of well-documented components including positive and negative voltage fluctuations at specific latencies post-stimulus (Luck, 2005).

Ford and colleagues have attempted to experimentally test the proposal that a corollary discharge breakdown occurs in those with AVHs by examining auditory cortical responsiveness to auditory stimuli during inner speech, as compared to a silent control condition. Ford et al. (2001a) suggested that the N1 component of the ERP “elicited by unattended, irrelevant acoustic events” (p. 1914) would be an appropriate measure of auditory cortical responsiveness. The N1 component is the first long-latency negative component on the ERP waveform, seen approximately 100ms post-stimulus onset, and is of greatest amplitude in response to auditory stimuli at the Cz electrode site, located at the vertex of the scalp (Katz, 2001). In healthy controls, Ford et al. found that the N1 response to acoustic stimuli (taken as the average of Cz, and two adjoining sites, namely Pz and Cza) was reduced when participants were reciting sentences in inner speech, compared to when they were silent. This was taken as evidence of a corollary discharge mechanism in action. Ford and Mathalon (2005) refer to this as “our N1 measure of corollary discharge dysfunction” (p. 187). When Ford et al. (2001a) studied patients diagnosed with schizophrenia, they found that their N1 response to auditory stimuli was significantly less dampened during inner speech, as compared to a silent condition. This was interpreted to mean that such patients had a less effective corollary discharge system. However, Ford and Mathalon (2005) later failed to find that this effect was linked to the severity of AVHs in
such patients. Specifically, there was no relation between the extent of dampening of the N1 response and the severity of auditory hallucinations.

There were, however, a number of limitations to Ford et al.'s (2001a) methodology. Firstly, as Adkin, Campbell, Chua, and Carpenter (2008) have noted, "[i]ncreased attentional demand has... been shown to affect cortical response amplitude through attention-related facilitation of cognitive activity" (p. 123). The modulation of the N1 wave by attention is a well-documented effect, with increased attention to auditory probes resulting in greater N1 wave amplitude (Naatanen & Picton, 1987). Thus, the differences found by Ford et al. (2001a) between the amplitude of the N1 response to auditory stimuli in the silent and inner speech conditions may be solely due to less attention being paid to the auditory stimuli in the latter condition. The first aim of the present study was to examine whether the dampening of the N1 response during inner speech (compared to silent baseline) could be accounted for solely by attentional factors. This was done by adding a control condition to Ford et al.'s methodology, specifically one which placed demands on the attention of the participants, but which did not involve inner speech. The control task chosen was mental rotation of objects (Shepard & Metzler, 1971), which can be seen as unlikely to involve a significant inner speech component. If participants showed a dampening of the N1 response in this task (compared to silent baseline) it would suggest that dampening in inner speech conditions is due to attentional demands, and not the operation of a corollary discharge system. Conversely, if no dampening of the N1 response to auditory stimuli was shown during the mental rotation task, this would suggest that the dampening may be specific to inner speech tasks. This would provide support for the claim that the dampening of the N1 response is a valid proxy measure of a corollary discharge system that results in self-produced speech being experienced as self-produced.
A second limitation of Ford et al.'s (2001a) methodology is its reliance on unexamined assumptions about the nature and elicitation of inner speech. Ford et al. asked patients and controls to generate inner speech in the form of simple statements said to themselves in their own voice (e.g., "That was stupid"). It has previously been argued that such simple, subvocal rehearsal is not an ecologically valid method of eliciting inner speech (Jones & Fernyhough, 2007a, 2007b; see Chapter 5 and Appendix B). Although a subvocal rehearsal conception of inner speech has a clear historical foundation in Baddeley and Hitch's (1974) model of working memory, other conceptions of inner speech have been proposed. One valuable, alternative account has been developed by Vygotsky (1934/1987). In this model, supported by a body of empirical work (e.g., Winsler, 2004), inner speech represents the endpoint of a developmental process in which external discourse gradually becomes internalized to form verbal thought. One result of this internalization is that verbal thought takes a dialogic form (Fernyhough, 1996, 2004; see Appendix B for further discussion). For this reason, the study reported here set out to investigate whether Ford et al.'s (2001a) original finding of dampening of the N1 response in healthy participants during subvocal rehearsal also obtained when participants were asked to generate dialogic inner speech, argued to be a more ecologically valid form of the phenomenon (Jones & Fernyhough, 2007; see Appendix B). In the dialogic inner speech condition, participants were given an outline of a scenario within which they were free to create their own spontaneous dialogue.

Alongside these concerns about the ecological validity of inner speech conditions, the present study set out to build on previous research in this area by considering a specific type of inner speech that has been proposed to be key in the creation of AVHs (Jones & Fernyhough, 2007; see Chapter 4 and Appendix B). Neuroimaging studies (e.g., Shergill,
Bullmore, Simmons, Murray, & McGuire, 2000) have found that when patients diagnosed with schizophrenia with AVHs produce statements in inner speech such as "That was stupid" (i.e., similar to the Ford et al., 2001, paradigm), no differences in neural activation exist in relation to controls. However, differences to controls have been found when patients imagine the voices of other people talking to them in their inner speech, termed 'auditory verbal imagery' (AVI; Shergill et al., 2000). It has been proposed that this is due to patients with AVHs having specific difficulties with tasks requiring high levels of verbal self-monitoring, the cognitive capacity responsible for monitoring inner speech (Shergill et al., 2003). Greater levels of verbal self-monitoring are thought to be involved in AVI due to the necessity to assess whether the voice has the prosody, tone, pitch, and rhythms of the voice it is intended to be (McGuire et al., 1996). The present study accordingly investigated dampening of the N1 response, compared to silent baseline, when participants performed AVI.

In summary, the present study set out to examine the following four hypotheses. Firstly, it was hypothesised that a dampened N1 response during subvocal rehearsal, as compared to silent baseline, would be found. Secondly, it was hypothesised that this dampening would be found in a comparison between baseline and dialogic inner speech. Thirdly, it was predicted that this dampening would also be found during AVI. Finally, it was hypothesised that no dampening of the N1 response would be found during a control task (mental rotation) designed to involve minimal levels of concurrent inner speech.

Method

Participants

An e-mail was circulated to students and staff in the Psychology Department at a United Kingdom University with an invitation to participate in the study. Those who
indicated that they would potentially be interested were sent a Participant Information Sheet (Appendix D) with details of the study. All participants gave written informed consent after the procedures had again been fully described. Although a total of 16 individuals took part in the study, clean data sets were only obtained for 10 participants (4 men, 6 women) with a mean age of 32.4 years ($SD = 12.0$). Participants were paid £7 to participate in the study, which lasted between 60 and 90 minutes.

**Procedure**

*Experimental stimuli.* Stimuli were programmed and presented using the STIM software package. Stimuli were presented to subjects continuously during each experimental condition, and consisted of a series of three equiprobable items as employed by Ford et al. (2001a). These were i) a speech sound (a voice saying 'no') presented via headphones, ii) a pure 1000Hz tone presented via headphones, and iii) a square chequerboard presented visually on the screen. Each had a duration of 250ms. These were all presented with three equiprobable, random stimulus onset asynchronies of 0.8, 1.0, and 1.2s (i.e., time of onset of the next stimulus was randomly selected as being 0.8, 1.0, or 1.2s after the previous stimulus). The volume of the tones was 90dB. These aspects of the methodology were thus identical to the procedures used by Ford et al. (2001a).

*Experimental conditions*

*Baseline.* In the baseline condition participants sat upright, wearing foam ear-cuff headphones and facing a 15-inch computer monitor. They were asked to keep their eyes focused on a fixation point on the screen throughout the experiment, when the chequerboard was not present. There was no other task associated with this condition.

*Subvocal rehearsal.* A sentence printed on an A4 sheet was presented to the participant. They were asked to memorise this sentence before the experimental portion of
the study began. When the participant was happy that they knew the sentence, the A4 sheet was removed and the experimental portion of the study began. Participants repeated the given sentence continuously to themselves silently in their head for 30s. After the completion of one sentence, the procedure was stopped, the participant was given an A4 sheet with the second sentence on, and the procedure was repeated. A total of seven sentences were utilized: “Go to the kitchen”, “That was stupid”, “Put it down”, “Tie your laces”, “Put on your jumper”, “That was very smart” and “You’re an idiot”. These were designed to be similar to the sentences used by Ford et al. (2001a) which in turn had been designed to be similar to typical AVHs.

*Auditory verbal imagery (AVI).* This condition was the same as the subvocal rehearsal condition except that participants were asked to imagine hearing a significant person in their life saying the sentences to them. Participants were asked to imagine hearing the same person for each sentence.

*Dialogic inner speech.* In this condition participants were first given a scenario. Five scenarios were employed in total. These were “imagine you are back at your old school and that you have just met one of your teachers”, “imagine you are back at home talking to your mother or father”, “imagine you are helping a small child do a jigsaw puzzle”, “imagine you have just met an old school friend whom you have not seen for years”, and “imagine someone has come to this country from a relatively deprived country. Try to imagine explaining to them one of your hobbies or pastimes which they are likely to be unfamiliar with. For example, this could be a sport you play”. Once the participant was familiar with the scenario, they were given the following instructions: “Imagine, silently in your head, how this conversation might go. For example, imagine them asking you questions, and you answering them”. Each scenario was used as the basis for generating
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dialogic inner speech for a period of 45s, at which point the trial was terminated and the
next trial began.

*Mental rotation.* In this condition participants were presented with a picture
of a 3D object, on an A4 sheet of paper, taken from the object set designed by Shepard and
Metzler (1971). These objects are presented in Appendix E. Participants were asked to keep
this object in mind during the task that followed. The picture of the object was then
removed and for the next 30s participants were asked to rotate this object in their mind’s
eye in a defined direction. This was done for each of the objects shown in Appendix E.

**ERP Procedure**

Electroencephalogram (EEG) was recorded from 10 specific sites (Fp1, Fp2, AFz, 
Fz, FCz, Cz, Pz, O1, O2, and Iz) using a 32 channel EasyCap EC40, amplified by a 
SynAmps amplifier and recorded using SCAN 4.3.1 software. Only 10 sites were enabled
in order to ensure the experimental procedure lasted an acceptable length of time for the
participants, and to hence reduce the impact of fatigue on participants’ EEG. Due to poor
signal quality from many of the frontal sites, only ERPs collected from the Cz electrode
(located at the vertex of the scalp) are reported here. ERPs in response to auditory probes
have previously been found to be largest at the Cz site (Katz, 2001). Vertical
electrooculogram (VEOG) was recorded from electrodes placed above and below the right
eye, and horizontal electrooculogram (HEOG) from electrodes placed at the outer rim of
each eye. EEG and EOG were sampled every 4ms. The continuous EEG was first filtered
using a low pass (30Hz) filter. It was then epoched into trials which began 200 ms before
the stimulus and ended 500ms after it. These trials were manually examined for indications
of eye blinks and eye movements. Any significant fluctuations of the VEOG and HEOG on
a trial led to the trial being excluded from the data set. Approximately 10% of trials were
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rejected due to indications of such eye blinks and movements. An average ERP amplitude for auditory trials was then calculated. To increase the power of the study the speech stimuli and pure tone stimuli were summed into one overall auditory ERP. Following standard practice, N1 was identified as the most negative peak between 75 and 200 ms. Iz was used as the reference electrode due to the low level of N1 activity seen at this site compared to Cz (Katz, 2001).

Results

Descriptive statistics for peak N1 amplitudes in each condition are presented in Table 7.1. The latencies of the peak N1 amplitudes are presented in Table 7.2. Figures 7.1 to 7.5 illustrate the averaged electrophysiological responses of the individuals over the epoched period from 200ms pre-stimulus to 500ms post-stimulus for the different conditions. The N1 component is clearly identifiable in all these figures.

Peak N1 amplitudes were compared for the target comparisons using paired sample t-tests. As all hypotheses were directional, one-tailed \( p \) values are reported. As Table 7.3 shows, peak N1 responses to auditory stimuli were significantly lower than baseline in all experimental conditions. The significant differences between baseline and subvocal rehearsal, AVI, and mental rotation were all classed as being large effect sizes (Cohen, 1988). The difference between baseline and dialogic inner speech was classed as a medium effect size (Table 7.3). The latencies of the N1 peaks (i.e., the time, post-stimulus, at which N1 peaks occurred) were also compared for each of these across-condition comparisons (Table 7.4). If the latencies were to be significantly different for participants across the experimental conditions this could be indicative that values of peak N1 amplitudes across conditions may be unreliable (Luck, 2005). As indicated in Table 7.4, latencies of the N1 peaks did not differ significantly across the experimental conditions.

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Table 7.1

<table>
<thead>
<tr>
<th>Participant #</th>
<th>Baseline (silent)</th>
<th>Subvocal rehearsal</th>
<th>Dialogic inner speech</th>
<th>Auditory verbal imagery</th>
<th>Mental rotation</th>
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<tr>
<td>1</td>
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<td>10</td>
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<td>-3.07</td>
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<td>Mean</td>
<td>-8.30</td>
<td>-6.42</td>
<td>-6.00</td>
<td>-4.67</td>
<td>-4.71</td>
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<td>(3.20)</td>
<td>(2.71)</td>
<td>(1.87)</td>
<td>(1.66)</td>
<td>(1.47)</td>
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**Note:** *= Data was too noisy to clearly identify the N1 peak.
<table>
<thead>
<tr>
<th>Participant #</th>
<th>Baseline (silent)</th>
<th>Subvocal rehearsal</th>
<th>Dialogic inner speech</th>
<th>Auditory verbal imagery</th>
<th>Mental rotation</th>
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<td>160</td>
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<td>174</td>
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<tr>
<td>Mean (SD)</td>
<td>146 (16)</td>
<td>150 (14)</td>
<td>150 (9)</td>
<td>147 (19)</td>
<td>149 (17)</td>
</tr>
</tbody>
</table>

Note. * = Data was too noisy to clearly identify the N1 peak.
Chapter 7: Electrophysiological study of inner speech

Figure 7.1

Averaged electrophysiological response in baseline condition (silent listening, N =10)
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Figure 7.2

Averaged electrophysiological response in subvocal rehearsal condition (N = 10)
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Figure 7.3

Averaged electrophysiological response in dialogic inner speech condition (N = 8)
Figure 7.4

Averaged electrophysiological response in auditory verbal imagery condition (N = 9)
Figure 7.5

Averaged electrophysiological response in mental rotation condition (N = 8)
## Table 7.3
Comparisons between peak N1 amplitudes between experimental conditions

<table>
<thead>
<tr>
<th>Conditions compared</th>
<th>t, p</th>
<th>Cohen's d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline—Subvocal rehearsal</td>
<td>t(9) = 2.85, p &lt; .01</td>
<td>.90</td>
</tr>
<tr>
<td>Baseline—Dialogic inner speech</td>
<td>t(7) = 1.96, p &lt; .05</td>
<td>.55</td>
</tr>
<tr>
<td>Baseline—AVI</td>
<td>t(8) = 4.95, p &lt; .001</td>
<td>1.48</td>
</tr>
<tr>
<td>Baseline—Mental rotation</td>
<td>t(7) = 3.84, p &lt; .01</td>
<td>1.03</td>
</tr>
</tbody>
</table>

## Table 7.4
Comparisons between peak N1 latencies between experimental conditions

<table>
<thead>
<tr>
<th>Conditions compared</th>
<th>t, p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline—Subvocal rehearsal</td>
<td>t(9) = 1.04, n.s.</td>
</tr>
<tr>
<td>Baseline—Dialogic inner speech</td>
<td>t(7) = 1.40, n.s.</td>
</tr>
<tr>
<td>Baseline—AVI</td>
<td>t(8) = .30, n.s.</td>
</tr>
<tr>
<td>Baseline—Mental rotation</td>
<td>t(7) = .18, n.s.</td>
</tr>
</tbody>
</table>
This study firstly aimed to replicate the findings of Ford et al. (2001a) showing dampening of the N1 response to auditory stimuli during subvocal rehearsal, as compared to a silent baseline task. As hypothesised, this finding was replicated. Secondly, as predicted, this dampening effect was also found during dialogic inner speech. Thirdly, again as hypothesised, a dampened N1 response was found during auditory verbal imagery as compared to silent baseline. Fourthly, the hypothesis that a dampened N1 response would not be found in the mental rotation task was not supported. There were no differences between the peak latencies of the N1 response between the baseline and experimental conditions, supporting the conclusion that the N1 peak amplitude data was reliable.

The finding of a dampened N1 response during mental rotation suggests that the dampening seen in response to inner speech tasks is not specific to the concurrent performance of inner speech, but is rather due to a reduction in attention to the auditory stimuli. The implications of this are that we should be cautious about interpreting Ford et al.’s (2001a) findings as evidence of a corollary discharge mechanism being defective in voice-hearing patients with schizophrenia. Furthermore, the lack of dampening of the N1 response during inner speech by such patients may indicate attentional differences in this group. It may be that the patients were unable to focus entirely on the inner speech task, and found their attention returning to the acoustic probes instead, resulting in their N1 responses during inner speech being more similar to that during baseline.

This interpretation of Ford et al.’s (2001a) findings as resulting from an attentional artefact is supported by a substantial literature on deficits in attention in schizophrenia (e.g., Harris, Minassian, & Perry, 2007). Indeed, previous ERP studies have found that “the
ability to selectively attend to a target in one modality while ignoring the other is compromised in patients with schizophrenia" (Wood, Potts, Hall, Ulanday, & Netsiri, 2006, p. 67) and that "executive control of auditory attention cannot be sustained in schizophrenia" (Mathalon, Heinks, & Ford, 2004, p. 872). Were the present experimental procedure to be performed with patients with schizophrenia with AVHs, it would be expected that they would show less of a difference in their N1 response between baseline and subvocal rehearsal, dialogic inner speech, and AVI conditions, than healthy controls would, due to attentional impairments in the patients. However, it would also be predicted that they would also show a dampening of the N1 response to auditory stimuli during the mental rotation task (although probably less dampening than healthy controls). If these predictions were supported, it would offer further evidence that the dampening of the N1 is an attention-related phenomenon, and not related to corollary discharge mechanisms. Such a view would also entail the prediction that no differences in N1 response in the various conditions examined here would be found between patients with AVHs and those without AVHs. Indeed, such a prediction is consistent with Ford and Mathalon’s (2005) finding that patients with schizophrenia with AVHs, and patients with schizophrenia with no AVHs, do not differ in the magnitude of their N1 dampening during subvocal rehearsal (as compared to silent baseline).

A number of limitations of the present study need to be acknowledged. Firstly, this was a relatively small sample and it would be beneficial to replicate these findings using a larger number of participants. However, it is worth noting that the sample sizes employed were comparable to those employed by Ford et al (2001a, N = 15, 2001b, N = 7). Secondly, the present study used the Iz electrode as the reference electrode, as opposed to the traditional mastoid reference electrode. It could be claimed that, by referencing to the Iz
electrode rather than the mastoid, smaller Cz ERPs would result (due to greater N1 ERPs being recorded at Iz than at the mastoid), leading to a loss in power of the study. However, firstly, it has previously been shown that the N1 ERP at the Iz site is low, and comparable to that recorded at the mastoids (Katz, 2001). Secondly, the present study was able to detect significant differences in all its N1 peak amplitude comparisons, and hence it is unlikely that use of the Iz site as the reference electrode impacted significantly on the present findings due to power reductions.

A third possible objection is that participants were not correctly performing the tasks they were required to. For example, participants’ thoughts may have drifted away from the set task onto other, non-task-related topics. Although this is possible, it was noted that, even though participants indicated that some of the tasks were harder than the others (performing AVI was noted to be difficult by some participants), they all reported being able continuously to attempt the tasks. Finally, it should be noted that the silent baseline condition was assumed to have no inner speech in it. This is likely to be a false assumption (Jones & Fernyhough, 2007; see Appendix B) as some spontaneous inner speech was reported by most participants in this condition upon questioning. That said, volumes of inner speech were still likely to be greater in the inner speech experimental conditions (subvocal rehearsal, dialogic inner speech, and AVI), where inner speech was directly elicited, as compared to the silent baseline. For these reasons, it is arguable that the presence of spontaneous inner speech in the silent baseline condition is unlikely significantly to have contaminated the results of this study.

In terms of future avenues for research, it has been argued here that the N1 ERP response to acoustic stimuli during inner speech is unlikely to be a good proxy measure of corollary discharge. Therefore alternative techniques for examining any such postulated
corollary discharge mechanisms are likely to be required. One such alternative is diffusion
tensor imaging (DTI), as used by Hubl et al. (2004). In their DTI study Hubl et al., found
greater dysfunction in white matter signalling tracts in patients with schizophrenia with
AVHs, as compared to patients without AVHs. This lead them to propose that dysfunctions
in such white matter tracts could lead to inner speech activating regions involved in the
processing of external stimuli, resulting in AVHs. Taking a structural anatomical approach
to identifying abnormalities in white matter signalling between frontal and temporal regions
may hence prove more valuable in charting the mechanisms of inner speech production
than electrophysiological methods.
Chapter 7: Electrophysiological study of inner speech

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Chapter 7: Electrophysiological study of inner speech


Chapter 8

Do we need multiple models of auditory verbal hallucinations?

Examining the phenomenological fit of cognitive and neurological models

ABSTRACT

The causes of auditory verbal hallucinations (AVHs) are still unclear. The evidence for two prominent cognitive models of AVHs, one based on inner speech, the other on intrusions from memory, is briefly reviewed. The fit of these models, as well as neurological models, to the phenomenology of AVHs is then critically examined. It is argued that only a minority of AVHs, such as those with content clearly relating to verbalizations experienced surrounding previous trauma, are consistent with cognitive AVHs-as-memories models. Similarly, it is argued that current neurological models are only phenomenologically consistent with a limited subset of AVHs. In contrast, the phenomenology of the majority of AVHs, which involve voices attempting to regulate the ongoing actions of the voice-hearer, is argued to be more consistent with inner speech-based models. It is concluded that subcategorisations of AVHs may be necessary, with each underpinned by different neurocognitive mechanisms. The need to study what is termed the dynamic developmental progression of AVHs is also highlighted. Future empirical research is suggested in this area.

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Chapter 8: Multiple models of AVHs?

Introduction

The cognitive and neurological mechanisms involved in auditory verbal hallucinations (AVHs) remain poorly understood. With approximately two-thirds of patients with schizophrenia, and 4% of the general population (Johns & van Os, 2001), reporting such experiences (Slade & Bentall, 1988; Wing, Cooper, & Sartorius, 1974), there is a clear need to understand this frequently distressing experience (see General Introduction). The term AVH encapsulates a diverse phenomenological experience, which may involve single and/or multiple voices, who may be known and/or unknown, speaking sequentially and/or simultaneously, in the first-, second-, and/or third person, which may give commands, comments, insults, or encouragement. Given the prima facie heterogeneity of AVHs it is surprising that only recently has the suggestion been made (David, 2004) that "perhaps we now have to consider further subcategorisations of [auditory] hallucinations" (p. 118) and that this is likely to have important clinical, theoretical and empirical implications (Laroi, 2006; Laroi & Woodward, 2007). This paper aims to consider how well different cognitive and neurological models of AVHs accord with the phenomenology of the experience, and what implications this has for potential subcategorisation of the experience.

AVHs and inner speech

One prominent type of model proposes AVHs to result from self-monitoring deficits leading to inner speech not being recognised as self-produced, and instead being perceived as an autonomous, non-self voice (Allen, Aleman, & McGuire, 2007; Frith & Done, 1989; Jones & Fernyhough, 2007; Seal, Aleman, & McGuire, 2004) Inner speech has been defined in a variety of ways, ranging from the "subjective phenomenon of talking to oneself, of developing an auditory-articulatory image of speech without uttering a sound"
Chapter 8: Multiple models of AVHs?

(Levine, Calvanio, & Popovics, 1982, p. 391) to “the overlapping region of thought and speech” (Jones & Fernyhough, 2007, p. 148), or simply “thinking in words” (McGuire et al., 1995, p. 596). Allen and colleagues (Allen et al., 2007) have recently reviewed the behavioural and neuroimaging evidence for an involvement of inner speech in AVHs. They concluded the behavioral evidence for impaired monitoring of inner speech relating to AVHs is “limited” (p. 416), and that the specificity of such a deficit to AVHs is “questionable” (p. 412) as impaired self-monitoring is also found in patients with delusions. However, studies of neural activation associated with AVHs, inner speech, and source monitoring were found to offer “more convincing evidence for the defective monitoring of inner speech in patients with hallucination” (p. 415). Such studies were argued to favor a disconnections model, in which speech production areas are unable to modulate the activity of the auditory cortex (i.e., is disconnected from it) to signal that inner speech is self-generated, resulting in it being experienced as alien. Overall, Allen and colleagues concluded the existing neuroimaging evidence for an involvement of inner speech is stronger than the behavioral evidence, which is equivocal.

One limitation of Allen et al.’s (2007) review is that it did not attempt to evaluate inner speech-based models of AVHs on the basis of what can be termed an argument-from-phenomenology. Specifically, are AVHs phenomenologically consistent with inner speech? Recently, it has been claimed that an alternative cognitive model of AVHs, which conceives of such experiences as reactivated memories, concords well with the phenomenology of the experience (Waters, Badcock, Michie, & Maybery, 2006). This model will be briefly reviewed before evaluating it, as well as inner speech-based models of AVHs, by the argument-from-phenomenology.
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AVHs as a sin of memory

West’s (1962) perceptual release theory proposed AVHs to be “previously recorded information: percepts, engrams, templates, neural traces, etc” (p. 281). In a cognitive reprise of elements of this model, Waters and colleagues (Waters et al., 2006) have argued that auditory hallucinations (AHs), including AVHs, are a result of the “unintentional activation of memories” (p. 65) or “the failure to inhibit memories of prior events” (Badcock, Waters, Maybery, & Michie, 2005, p. 132).

Waters and colleagues (Waters et al., 2006) propose that AHs result from two specific deficits. The first is a failure in intentional inhibition: the ability to deliberately suppress thoughts of an item after deciding it is irrelevant, a process which is effortful and available to conscious reflection (Nigg, 2000). This has been shown to result in intrusive thoughts, which occur more often in patients with schizophrenia with AVHs than healthy and psychiatric controls (Morrison & Baker, 2000). AVHs in the healthy population have also been linked to the presence of intrusive thoughts, potentially caused by failing thought suppression (Jones & Fernyhough, 2006). A number of studies have found failures of intentional inhibition in patients with schizophrenia with AVHs (Badcock et al., 2005; Waters, Badcock, Maybery, & Michie, 2003). However, as some patients without AHs have also been found to have such deficits, Waters and colleagues (Waters et al., 2006) suggest that a second deficit is necessary for the occurrence of AHs, namely a context memory deficit.

Waters and colleagues (Waters et al., 2006) note that episodic memory research differentiates between an event’s content and its context (the source or temporal characteristics surrounding the encoding of the event), and propose that in AHs the content of a memory is unintentionally activated, yet the context is not. This leads to these
representations being confused with ongoing reality. Consistent with this, impaired
processing of contextual information has been found in patients with schizophrenia with
AHs (Bazin, Perruchet, Hardy-Bayle, & Feline, 2000). More specifically, Waters and
colleagues (Waters, Maybery, Badcock, & Michie, 2004) found that nearly all such patients
tested had a context memory deficit. Patients were asked to watch or perform pairings of
two different sets of 24 common objects over two sessions 30 min apart. In a recognition
test performed five minutes later patients were tested on their episodic memory (‘did you
see this pair?’) and two forms of context memory: source context (‘did you put the pair
together or did the experimenter?’) and temporal context (‘was the pair in the first or
second set?’). A deficit in source context memory was found to be specific to patients with
AHs, whereas temporal context memory was found to be impaired in both hallucinating and
non-hallucinating patients, compared to healthy controls. However, later studies have found
temporal context memory deficits specific to those with AVHs (Brebion, David, Jones,
Ohlsen, & Pilowsky, 2007).

Waters and colleagues (Waters et al., 2006) also provide evidence that the
combination of context memory deficits and intentional inhibition is associated with AHs.
When deficit was defined as performance one standard deviation worse than controls, 90%
of patients with current AHs were found to have deficits in context memory and intentional
inhibition. Contrastingly, only 33% of patients (significantly less) with remitted AHs
showed deficits in both context memory and intentional inhibition. In mapping these
deficits to specific aspects of AHs, it has been argued that the failure of intentional
inhibition leads to the intrusive cognition, with the failure of context memory resulting in it
being not experienced as a product of one’s own mind (Badcock, Waters, & Maybery,
2007). Overall, although weakened by the fact that some hallucinating patients did not
show both deficits, and some non-hallucinating patients did show both deficits, intentional inhibition and context memory appear to be associated with AVHs (Aleman & Larøi, 2008). However, whether these deficits play a causal role, or derive from some other factor which causes AVHs, is as yet unknown.

AVHs as memories: a phenomenological fit to the experience?

In evaluating their model, Waters and colleagues (Waters et al., 2006) claim “the proposal of auditory hallucinations as memories” is consistent with the phenomenology of the experience and can explain why “entire dialogues from a conversation may be recalled” and “why voices often refer to the patient’s personal details” (p.76). However, this argument-from-phenomenology is based on only a limited number of forms of AVHs, without any comment by the authors on whether these forms are typical of the experience. A more detailed evaluation of this model based on the argument-from-phenomenology is hence required.

AVHs consistent with memory-based models

The phenomenology of some AVHs do indeed appear to be consistent with the AVHs-as-memories account, particularly where the content of the AVH can be linked to memories of previous traumatic/abuse experiences. It seems likely such AVHs are related to decontextualised intrusions of this material from memory. In accord with the perceptual quality of many AVHs, intrusive trauma memories tend to be sensory fragments of the traumatic experience (Ehlers, Hackmann, & Michael, 2004). There is substantial evidence demonstrating that hallucinations in general (Read, Agar, Argyle, & Aderhold, 2003) AVHs specifically (Morrison & Peterson, 2003; Offen, Waller, & Thomas, 2003) and
particularly AVHs which take the form of commands to hurt the self or others (Hammersley et al., 2003), are associated with earlier experiences of physical and sexual abuse. However, there has been significantly less research into the concordance between the actual content of the AVH and the auditory experiences undergone during and surrounding such abuse. If actual memories associated with the trauma (e.g., what an abuser said) are the basis of some AVHs, then concordance between the content of AVHs and trauma memories would be expected.

Direct evidence for this comes from findings showing that the content of AVHs can be linked to traumatic experiences. Read and Argyle (1999) found, in three of seven instances in which content of the AVHs of patients with schizophrenia was recorded, that the content could be linked to physical or sexual abuse. For example, command hallucinations to self-harm were found to be in the voice of the abuser. Similarly, Fowler (1997) reported a history of trauma in 14 of 24 patients with psychosis who experienced hallucinations, and in 4 of these 14 patients the voice heard was appraised as being that of the abuser. In addition, some content of the voices matched utterances heard at the time of the abuse. Clinical experience also suggests that the content of AVHs in those who have experienced abuse/trauma can be related to what was said during, and surrounding, these events (e.g., if you tell anyone I’ll kill you). This has led Read and colleagues (Read, Van Os, Morrison, & Ross, 2005) to claim that “some psychotic hallucinations appear to be nothing more or less than memories of traumatic events” (p. 341).

AVHs not consistent with memory-based models

Despite the suggestive evidence above, further consideration shows the AVHs-as-memories account to be in phenomenological accordance with only a (relatively small)
subset of AVHs. Firstly, in the study of Fowler (1997) cited above, only in 4 of the total 24 voice-hearers studied could the content of voices be seen as “sometimes” being suggestive that these were memories. In the remainder of the sample who had a history of trauma, meaningful connections could be made between the trauma and the voices. However, such connections were thematic (e.g., both the voices and trauma involved humiliation) rather than involving a direct relation between the content of the voices and what was said during and surrounding the trauma. Similarly, another study (Hardy et al., 2005) found only 7% of individuals with AVHs were rated as demonstrating clear concordance between the theme and content of the trauma and the themes and content of the voices. This study also found that 42% of people with hallucinations, who reported having current problems with past trauma, had no association between the content of their hallucinations and the past trauma.

Instead, Fowler and colleagues (Fowler et al., 2006) have noted that, in those who have experienced trauma, AVHs typically involve critical comments or comments about the person’s day-to-day experiences. This observation is consistent with the conclusion of a study of AVH phenomenology in patients with schizophrenia, and in those without any psychiatric diagnoses, by Leudar and colleagues (Leudar, Thomas, McNally, & Glinski, 1997). This study concluded that AVHs are “focused on the regulation of everyday activities” (p. 896). Similarly, Nayani and David (1996) note that 46% of their sample of patients with schizophrenia said their AVHs had come to replace their “voice of conscience” (p. 185) and that a proportion relied on their AVH for making decisions. Furthermore, AVHs were typically “minutely engaged in the apprehension of objective reality” (p. 185). It is hard to understand how intrusions from memory of past verbalisations could function in such a role. It is also worth noting that Nayani and David observe that AVHs tend to evolve over time with the voices “fashioning increasingly
detailed dialogues with or about the patient” (p. 187). Again, it is hard to see how verbal intrusions from memory could create such an interactive dialogue.

That some AVHs are not simply an intrusive memory of verbalisations experienced in and surrounding trauma/abuse, or more generally, but more of a dynamic creation, is suggested by the technique of voice-dialogue (Romme & Escher, 1993). In this process a practitioner speaks to the voice-hearer’s AVH through the voice hearer. For example, the questioner may ask “what do you want” (addressing the voice-hearer’s AVH) and the voice-hearer is asked to listen to their AVH’s response to the therapist and report it verbatim (e.g., “I want her to die”). The ability of some individuals with AVHs to engage in such dialogues (and no empirical work yet exists clarifying what percentage of those with AVHs can engage in such a process) suggests that they are not simply experiencing static intrusions from memory during this process, but that the process is more dynamic and creative. Furthermore, when voice-hearers themselves engage in a dialogue with their voices the number of voices may multiply (Davies, Thomas, & Leudar, 1999), again pointing towards a more dynamic relation than simply static intrusions from memory.

Furthermore, other types of AVHs are hard to explain by this cognitive memory-based account. For example, AVHs may take the form of crowds of mumbling voices (Nayani & David, 1996). This seems an unusual form for an intrusive memory to take. Whilst it is well documented that trauma leads to intrusive recollections of the experience and surrounding events (Ehlers et al., 2004), it is unclear why anyone should experience an intrusive memory of mumbling voices. The same argument applies to non-verbal AHs. As Bleuler (1952) noted, in addition to verbal AHs, “blowing, rustling, humming, rattling, shooting, thundering, music, crying and laughing” (p. 96) may also be heard. Although Waters and colleagues’ (Waters et al., 2006) argue that their model can explain such types
of AHs, it would appear that these experiences, with seemingly random content, are instead more parsimoniously accounted for by an bottom-up ictal-based neurological model (see below) (Nayani & David, 1996).

In conclusion, what is to be made of the claim (Badcock et al., 2005) that a model which views AVHs as “the failure to inhibit memories of prior events” (p. 132), and sees “auditory hallucinations as memories” (Waters et al., 2006, p. 76), is consistent with the phenomenology of the experience? It appears from the above that such AVHs-as-memories models can only account for the phenomenology of a minority of types of AVH. For example, this may be applicable to the ~10-20% (Fowler, 1997; Hardy et al., 2005) of individuals whose voices include content which can be linked directly back to memories of trauma. However, the majority of AVHs, which phenomenological surveys have shown to be related to the ongoing patterns of activity in the voice-hearers life, seem to be inconsistent with this account. Such observations, and the conclusion that AVHs cannot be satisfactorily understood as simply a “direct intrusive [auditory] image of the trauma event” (p.113) have led Fowler and colleagues (Fowler et al., 2006) to argue instead for an inner speech-based model.

AVHs as inner-speech based: a phenomenological fit to the experience?

Based on their findings (Leudar et al., 1997) that AVHs are typically “focused on the regulation of everyday activities...and are characterized by the same dialogical structures one finds in ordinary speech” (p. 896) Leudar and colleagues have argued AVHs are phenomenologically consistent with inner speech. It has also been argued elsewhere, building on a consideration of the form, function, and development of private and inner speech, that the phenomenology of many types of AVHs is consistent with a basis in inner
speech (Jones & Fernyhough, 2007). For example, the high frequency of command AVHs, such as ‘get the milk’ or ‘go to the hospital’ (reported by 84% of voice-hearers in Nayani and David’s phenomenological survey; Nayani & David, 1996) is consistent with inner speech’s developmental linkage with the control of action (Vygotsky, 1987). If some AVHs do indeed have a basis in inner speech, it should not be surprising that they frequently have a similar regulatory quality.

However, inner speech conceived of as simply speaking silently in one’s own voice is hard to reconcile with the phenomenology of AVHs which are typically experienced as hearing the voice of another person. For example, Nayani and David found 71% of patients with schizophrenia reported that their AVH’s accent differed to their own (Nayani & David, 1996). Indeed, to argue that inner speech, conceived in this way, is the raw material of AVHs leads to a number of predictions that have been experimentally falsified. For example, if AVHs occur because such inner speech is misidentified, individuals with AVHs should presumably report less frequent inner speech. However, a recent study found little difference between patients with schizophrenia with AVHs and healthy controls in terms of the frequency and form of their inner speech (Langdon, Jones, Connaughton, & Fernyhough, in press). Furthermore, when the form and content of inner speech (again conceived of as speaking silently in one’s own voice) of patients with schizophrenia with AVHs are compared to controls, no differences are found, and the pragmatics of such patients’ inner speech are not related to the pragmatics of their AVHs (Langdon et al., in press). Additionally, neuroimaging research has found that when patients with schizophrenia with AVHs simply speak silently in their own voice in inner speech, saying sentences such as “I like x”, patterns of neural activation do not differ to controls (Shergill et al., 2001).
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In response to this, a number of recent inner speech-based models have attempted to explain why inner speech/verbal thought may sometimes involve not speaking in our own voice, but instead taking another individual’s perspective on our experiences (Jones & Fernyhough, 2007). A Vygotskian approach to inner speech has proposed, in brief, that our thinking takes the form of a dialogue which is literally shot through with other voices (Fernyhough, 2004). Similarly, Dialogical Self Theory proposes inner speech can involve multiple sets of characters, with autonomous thought centres, that interact in verbal and non-verbal dialogical forms in an imaginal space (Hermans, 2001; Hermans & Dimaggio, 2004). Another approach comes from Fowler and colleagues (Fowler et al., 2006), who have proposed a basis for AVHs in inner speech, but inner speech conceived of as rumination and inner dialogue. Specifically, due to the association between abuse and AVHs, Fowler and colleagues propose that rumination or inner dialogue about interactions with an abuser may be the raw material for many AVHs (Fowler et al., 2006). This would predict the content of AVHs to be likely to “reflect patterns of rumination or internal dialogue about self in relationship to what a shaming and insulting abuser might say about one’s current actions” (p. 113). Such an account is hence consistent with the phenomenology of many AVHs which, as noted above, are not the same as what was said during/surrounding earlier trauma, yet related to it.

Other similar inner speech-based accounts have also been developed. Such accounts have proposed that individual differences in the tendency to ruminate and perform imaginative verbal dialogues involving others may act as a risk factor for AVHs (Jones & Fernyhough, 2008; see Chapter 4), or that “vivid and perceptual imagination”, in combination with source monitoring deficits and particular metacognitive beliefs, may be the main constituent of AVHs (Moritz & Larøi, 2008). Although the ability to produce
imagined speech of others does itself not appear to be impaired in patients with AVHs (Evans, McGuire, & David, 2000), I have argued elsewhere (Jones & Fernyhough, 2007) that neuroimaging studies of patients with schizophrenia with AVHs showing atypical neural activation associated with the ability to silently imagine another’s voice speaking to oneself (Shergill et al., 2001) are consistent with involvement for this type of inner speech in AVHs. Hoffman and colleagues (Hoffman, Varanko, Gilmore, & Mishara, 2008) have also recently noted this latter point, namely that “source monitoring mislabeling may selectively attach to verbal imagery of non-self speakers rather than ordinary inner speech” (p. 1172).

Inner speech-based theories are still left with the problem that AVHs are typically reported as having the phenomenological quality of being heard. For example, in a study by Leudar and colleagues (Leudar et al., 1997) all patients with schizophrenia who heard voices reported that it was “very much like hearing other people speak” (p. 889). One approach to reconciling this to the phenomenology of inner speech has been to that suggest inner speech has more acoustical properties in voice-hearers. For example, a recent study found that approximately 40% of patients with schizophrenia with AVHs rated their own thoughts as having some acoustical properties (as opposed to being absolutely silent) (Moritz & Laroi, 2008). This led the authors to argue that AVHs may be associated with abnormalities with sensory inner perception “which apparently arise already at the stage of thoughts” (p. 105). An alternative approach is to question the degree to which an experience being labelled as a voice is to do with its acoustical properties. Stephens and Graham (Stephens & Graham, 2000) have argued that “something can count as a voice without being experienced as audition-like or mistaken for sensory perception of another’s speech” (p. 114). In line with this, not all AVHs have the phenomenal qualities of a heard
voice. Bleuler (1952) noted that some “patients are not always sure that they are actually hearing the voices or whether they are only compelled to think them. There are such ‘vivid thoughts’ which are called voices by the patients” (p. 110). More recent studies have found that 38% of patients with schizophrenia with AVHs said their voices were not very real (Moritz & Laroï, 2008), 44% said their AVHs were more like ideas than external sensations (Nayani & David, 1996), and only 20% of patients said their voices were indistinguishable from auditory perceptions (Miller, 1996).

Interestingly, inner speech models can predict the phenomenology of one of the more unusual forms of AVHs. Vygotsky (1987), Fernyhough (2004) has noted, proposed that inner speech typically becomes syntactically and semantically condensed and abbreviated, losing most of its structural and acoustic qualities, becoming a process of “thinking in pure meanings”. If inner speech forms the raw material for AVHs then, in addition to fully formed words or sentences being experienced as AVHs, it can be predicted that some would also have this quality of “pure meaning”. Frith (1992) has described this as the experience of receiving information without any sensory component. Such types of AVHs have indeed been documented. Bleuler (1952) termed these “soundless voices” (p. 110). In such AVHs a message or meaning is communicated although it is not actually heard. For example, a patient of Bleuler’s who threw himself into the Rhine reported afterwards that “It was as if someone pointed his finger at me and said “Go and drown yourself” (p. 111, italics added). Janet also noted this phenomenon, giving the example of a patient who reported that “it is not a voice, I do not hear anything, I sense that I am spoken to” (Leudar & Thomas, 2000).

In conclusion, the phenomenology of inner speech, including its regulatory nature, its linkage to ongoing events, its ability to involve the voices and perspectives of others, its
ability to take the form of “thinking in pure meanings” (Fernyhough, 2004, p.56), and its creative nature, are consistent with the phenomenological properties of a large number of AVHs. However, inner speech-based models have a number of limitations when compared to the phenomenology of AVHs. Firstly, they do not seem appropriate for the ~10-20% of individuals, as noted above, whose voices have content which can be linked directly back to memories of trauma. These instead appear better modelled as verbatim intrusions from memory. Secondly, as Waters et al. (2006) have argued, such models cannot explain other types of AVH, such as the voices of crowds, or other AHs, such as environmental noise and music. Indeed, Nayani and David (1996) found the latter to be quite frequent, with 36% of patients with schizophrenia with AVHs also reporting musical hallucinations, and 16% reporting elemental sounds such as clicks and bangs. Thirdly, it may be worth considering Hurlburt and Schwitzgebel’s differentiation between inner speech and inner hearing (Hurlburt & Schwitzgebel, 2007). These authors note that whilst inner speech is experienced as “going away”, “produced by”, and “under the control of” the individual, and is “just like speaking aloud except no sound”, in contrast inner hearing is the experience of a sound which is “coming toward”, “experienced by”, and “listened to” by the individual (p. 257). In these terms many AVHs are more phenomenologically consistent with inner hearing than inner speech. Despite these limitations, and in need of future empirical testing, inner speech-based models have the starting advantage of being in accordance with the phenomenology of a significant number of AVHs.

Overall, the arguments presented above suggest that the AVHs-as-memories model, and inner-speech based models, are both in accordance with the phenomenology of separate subsets of AVHs. However, neither model is consistent with the phenomenology of all AVHs. One possible conclusion is that both these models are correct, albeit each for a
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separate subset of AVHs. However, it is also worth considering an alternative view, namely that neither of these models is correct, with the appropriate explanation of AVHs lying at the neurological level rather than in cognition.

Neurological models and the phenomenology of AVHs

It is possible that the mechanisms underlying AVHs do not fit neatly into any cognitive model. For example, complex visual hallucinations in epilepsy and Charles Bonnet syndrome have been explained by bottom-up neurological models involving deafferentation and neural circuitry disinhibition (Hoffman, 2007), and are not readily accounted for by any cognitive mechanistic model. It has been proposed that an analogical model may be the appropriate level of explanation for AVHs (Hoffman, 2007). The following section attempts to evaluate this, and other bottom-up neurological models, in terms of their ability to predict the observed phenomenology of AVHs.

Bottom-up neurological models of AVHs tend to reason from analogy from Penfield and Perot’s (1963) studies involving direct external electrical stimulation of the temporal cortex (David, 2004). In these studies of Penfield and Perot (1963), participants reported a range of AHs. Firstly, reports of musical hallucinations were common, such as “I hear music” (p. 620) and “I hear singing... it is White Christmas” (p. 618). Other forms of environmental sounds were also reported, such as “a toilet flushing or a dog barking” (p. 628). Secondly, voices were heard. These were typically overheard conversations, not directed at the individual, including “I heard someone speaking, my mother telling one of my aunts to come up tonight” (p. 617) and voices that “sounded like a bunch of women talking together, just a lot of women chattering” (p. 622). Participants heard multiple simultaneous voices, including “something like a crowd” (p. 640) and “a lot of people
shouting at me” (p. 630). Reports of speech directed at the person were rarer, although still present. For example, one participant reported that “Someone is telling me to stay still” (p. 628).

Such AHs have commonality with the musical hallucinations, elemental sounds, and crowds of simultaneous voices found to co-occur with more complex AVHs in schizophrenia (Nayani & David, 1996). The phenomenology of these AHs can be seen to be consistent with what has been termed (Nayani & David, 1996) a “random and quasi-ictal explanation” (p. 187).

However, the ability of most bottom-up neurological models to account for the more typical phenomenological aspects of AVHs, such as voices directed at the hearer and focused on everyday activities, is less successful. For example, David and colleagues have proposed that AVHs result from language input processes in the temporal lobe functioning hyperactively, leading to a strong perceptual representation of auditory images (Brebion et al., 2007). Such a model can account well for some phenomenological properties of AVHs, such as their involuntariness, which many patients with AVHs highlight as important in differentiating their AVHs from their everyday thoughts (Hoffman et al., 2008), their intermittent occurrence, and their perceptual qualities. This model can also be seen to account for AVHs involving verbatim traumatic memories. Furthermore, such a model also predicts patterns of activation during AVHs found in many neuroimaging studies (Copolov et al., 2003). However, it gives no clear reason why many AVHs tend to be focused on everyday activities, and directed at the voice-hearer. Furthermore, such a model would also appear to predict that content of AVHs should be highly repetitive. This is the case in some, but by no means all AVHs (Nayani & David, 1996). However, a bottom-up neurological
model that makes more specific predictions about the phenomenology of AVHs has recently been developed.

Hoffman (2007) proposes that a lack of social contact may result in deafferentation-like reorganisation in regions of association cortex leading to AVHs, in a way analogous to the complex visual hallucinations in Charles Bonnet Syndrome which result from deafferentation due to vision loss. Specifically, Hoffman argues that as a result of social isolation, language detection systems' ability to detect complex verbal meaning is increased in response to the deprivation of normal conversational interaction, resulting in the production of spurious auditory experiences. This functions "in the service of filling in the 'blank slate' due to withdrawal from the world" (Hoffman et al., 2008, p. 1172). Such a model is claimed to predict the personal relations many individual have with their voices. Hoffman (2008) also claims such a model would predict AVHs to involve spurious social meaning in the form of complex, emotionally compelling voices of other persons or agents. This account also appears to predict that the emotional valence of the voice, and its pragmatics, would be in accordance with the way the individual relates to people within their social environment. This proposal appears to be empirically supported (Hayward, 2003). However, such a model still appears to fail to explain the tendency for AVHs to be associated with action regulation, and is hard pressed to explain the phenomenological findings of Moritz and Laroi (2008), discussed above, that highlight the similarity between thoughts and some AVHs.

Separate phenomenologies, separate causes?

From the above argument-from-phenomenology it appears that each of the cognitive models reviewed is only able at present to account for a subset of AVHs. One possible
conclusion is that neither of these models is correct, and that the true mechanism underlying AVHs is not readily captured by any cognitive model because at a neurological level the mechanism cuts across many cognitive, metacognitive, linguistic and/or perceptual capacities. An alternative conclusion is that both are correct, and that different types of AVHs have different underlying neurocognitive mechanisms. Such a proposal would entail subcategorisations of AVHs. The first type of AVH would be that with content directly linked to intrusive memories of early abuse/trauma, which could be understood via a neurocognitive model that sees AVHs as a failure to inhibit memories. A second type would involve seemingly random auditions, such as those involving crowds, noises, and music, and could be seen as best explained at the neurological level, using an ictal, bottom-up neurological model. Finally, a class of AVHs typified by novel statements that attempt to regulate the actions of the voice-hearer, linked to their ongoing activities, and which cannot be linked directly to a verbatim memory, can be delineated. Such AVHs appear to be phenomenologically best accounted for by a neurocognitive inner speech-based model.

The proposal that subcategorisations of AVHs are necessary, each involving different underlying neurocognitive mechanisms, leads to a number of falsifiable hypotheses. Firstly, different patterns of neural activation would be likely to underlie these different forms of AVHs. For example, a patient with an AVH identifiable as a verbatim memory of abuse would be hypothesised to show a different pattern of neural activation to a patient with a more novel AVH commenting on on-going actions. The first type of AVH may show activation more similar to that seen during traumatic autobiographical memory recall. As Broca’s area activation decreases during recall of traumatic (as compared to neutral) memories in patients with PTSD (Rauch et al., 1996), and increases during inner speech production (Shergill et al., 2001), less activation should be seen in this area during
AVHs involving intrusive memories of abuse, than during AVHs involving novel comments on ongoing actions. This proposal may explain the inconsistent findings of studies of activation in Broca’s area during AVHs (Jones & Fernyhough, 2007). Such inconsistency may be due to such studies involving differing ratios of patients with AVHs associated with intrusive memories of trauma/abuse to patients with more novel AVHs. It is, however, also highly possible that the inconsistent finding of Broca’s area activation may be due to the different tasks or paradigms that have been used. To allow comparability of findings it may be valuable for neuroimaging studies in future to report more details on the specific phenomenology of their patient’s AVHs, and to analyse their data by sub­groups of hallucinators based on phenomenology.

The distinction between inner speech-based AVHs and memory-based AVHs should also lead to detectable electrophysiological differences between such AVHs. Electrophysiological studies have found some evidence that when inner speech is produced in those with AVHs, dampening of the activity of the auditory cortex is impaired (Ford & Mathalon, 2005; see Chapter 7). It can be hypothesised that such a relation should be found only in AVHs which are phenomenologically consistent with inner speech. Conversely, in those which are more phenomenologically consistent with verbatim intrusions from memory such a disconnection would not be predicted. Again, the failure to employ such a distinction, and to hence treat AVHs as a homogenous phenomenon, may explain the mixed findings that electrophysiological studies have reported to date in investigating dampening of auditory responses in those with AVHs (Ford & Mathalon, 2005).

One factor that is likely to complicate testing such hypotheses is the fact that phenomenologically different types of AVHs often co-occur. For example, AVHs with parallels to inner speech often occur in the presence of seemingly more ictal-based AHs,
such as environmental noises. This is suggestive that there may indeed be an underlying neurological mechanism which cuts through across many cognitive, metacognitive, linguistic and/or perceptual capacities. Another factor to consider is that, although this paper has contrasted a AVHs-as-memories model with inner speech-based models, these two models may, to some degree overlap. The concept of a dynamic developmental progression, which I turn to next, helps illustrate this point.

**Dynamic developmental progression**

AVHs tend to evolve over time with voices “fashioning increasingly detailed dialogues with or about the patient” (p. 187). The evolution of AVHs and AHs over time, which is currently a neglected and poorly understood area, I term their dynamic developmental progression (DDP). This can be applied to the change in the nature of the AVHs/AHs themselves, as well as the change in the distress associated with them, and the beliefs held about them. As detailed published accounts of the DDP of AVHs are rare, an example from personal experience will be given. Adam (not their real name), who had been diagnosed with schizophrenia, initially developed an AVH the content of which was a voice saying (verbatim) phrases which had actually been said to them during a traumatic event. However, over time Adam’s AVH changed to become the voice of the same individual (from the traumatic event) but now saying novel things. It is hence worth considering the possibility that the mechanisms that may form the original basis for an AVH (e.g., a verbal intrusion of a specific memory of trauma) may come, over time, to be developed into a form unpredictable from its original content and potentially underpinned by different mechanisms (such as inner speech). Such transformations may be the result of cognitive, emotional, and neurological mechanisms. In the example above, it may be that memory-
based and inner speech-based processes interacted. Extensive activation of temporal lobe structures during spontaneous verbal thought has been taken to suggest that long-term memory processes may form the core of spontaneous thought (Christoff, Ream, & Gabrieli, 2004). Hence, if hyperaccessible memories exist (e.g. traumatic memories), it is plausible that these will be employed in inner speech processes. This could lead to cognitions with the properties of inner speech (e.g., action focused, relevant to ongoing events, etc.) but with content drawn from such memories. Such an interaction may result in an AVH involving a novel, action-related cognition involving the voice of an abuser. Although speculative, this highlights the possibility of synthesising AVH-as-memories models with inner speech-based models, to develop a synergistic account in line with a wider range of aspects of the phenomenology of AVHs than either of the models alone.

The concept of a DDP may prove valuable in a number of other ways. Given that the experience of AVHs stretches into the healthy population, it may be fruitful to consider how (and to what degree) AVHs may evolve from brief, benign experiences that can be considered more or less normal, to a distressing clinically relevant experience (Leudar et al., 1997). One possible DDP is that patients who come to develop schizophrenia initially experience benign, non-clinically relevant AVHs the content of which then becomes negative and distressing as a result of a traumatic/abusive experience. Personal experience indicates that a number of patients with schizophrenia report a DDP involving a positive, helpful voice in childhood which, after the individuals were exposed to a range of highly abusive experiences, became negative, abusive and entrenched in adulthood. Another (related) possibility is AVHs with benign content come to be negative, due to broader psychosocial factors affecting the individual. For example, how the individual relates to others in their social world, their social rank and levels of powerlessness, are associated
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with their relation to their AVHs (Hayward, 2003). It has also been noted (Bleuler, 1952) that some AVHs follow a DDP in which they “first appear as the ordinary dream; then they appear in the hypnagogic state; then finally in the full waking state”, a famous example being Emanuel Swedenborg (Jones & Fernyhough, 2008). A range of other DDPs are possible and encountered in patients, and it is likely that a large range of factors may interact dynamically to result in AVHs that are all-consuming and fiercely destructive. Phenomenological surveys which tend to take a ‘snapshot in time’ of individual’s AVHs, without assessing in detail the DDP of such experiences, are poorly equipped to address such issues. This highlights the need for future studies of the DDP of AVHs and AHs.

Conclusions

A key challenge for the study of AVHs is to derive models that fit the phenomenology of the experience, rather than fitting models to a phenomenology that does not pertain to it (Woodruff, 2004). This paper has observed that although models are created to fit the phenomenology of AVHs, due to the diverse nature of the experience they inevitably end up only fitting specific types of AVHs. Whilst it is possible a grand unified theory of AVHs and AHs may be developed, possibly involving a neurological mechanism cutting across many cognitive, metacognitive, linguistic and/or perceptual capacities, at present no such model exists. In contrast, I have proposed that to honour the phenomenological diversity of the experience of AVHs, a fruitful strategy may be subcategorisation. If phenomenology can indeed give clues to the etiology of the experience (David, 2004), then each subcategory may be accounted for by a different model involving different neurocognitive processes.
Specifically, it has been argued that neurocognitive intrusive memory-based models of AVHs account well for AVHs that have content directly linked to verbalisations during and surrounding earlier traumatic experiences. Purely bottom-up ictal neurological models have been argued to fit other specific AVHs, such as those involving crowds, noises, music, and brief stereotyped phrases. Finally, inner speech-based models have been proposed to underpin the typical form of AVHs, involving the regulation of everyday activities, which are engaged in the apprehension of objective reality, and have the same dialogical structures found in ordinary speech. A number of empirical tests have been suggested to test the proposal that subcategorisations of AVH exist that are underpinned by different neurocognitive mechanisms. If correct, this highlights the potential for future research designs to not just to contain groups of ‘hallucinators’ and ‘non-hallucinators’, but to group individuals with AVHs according to phenomenological subcategorisations.

I have also highlighted the need to study the dynamic developmental progression of AVHs, and how such an analysis opens up the possibility of synthesising of memory- and inner speech-based models of AVHs. Future work along these lines would likely benefit our understanding of the causes of AVHs.

If the proposal of subcategorisation of AVHs, underpinned by different mechanisms, can be empirically supported, this would importantly also have clear implications for treatment. For example, some therapeutic strategies aim to get voice-hearers to engage in dialogues with their AVHs, in an attempt to alter the individual’s relationship with their voices (Davies et al., 1999). Such an approach may be less appropriate for voices that are the result of intrusive memories, or bottom-up neurological factors, with instead techniques suitable to PTSD type flashbacks being more appropriate. The reverse would be the case for those with a phenomenology similar to inner speech.
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Also, given that up to a quarter of patients experience persistent hallucinations that are resistant to medication (Shergill, Murray, & McGuire, 1998) it may be fruitful to investigate whether the different phenomenological subcategorisations of AVHs proposed here respond differentially to antipsychotic medication.
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References


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hallucinations and perception of human speech in schizophrenia: a PET correlation study. *Psychiatry Research: Neuroimaging*, 122(3), 139-152.


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_Trauma and psychosis: New directions for theory and therapy._ (pp. 101-124).
London: Routledge.


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GENERAL DISCUSSION

Summary of findings

The studies reported in this thesis were designed to investigate a number of the cognitive mechanisms underlying the experiences of auditory verbal hallucinations and persecutory ideation. A guiding assumption has been what Aleman and Laroi (2008) have termed the continuum hypothesis, which holds that these experiences lie on a continuum with normal experiences.

Persecutory ideation

The first study (Chapter 1) built on previous work demonstrating the paradoxical impact of thought suppression, namely its tendency to make suppressed thoughts hyperaccessible and susceptible to post-suppression rebound (e.g., Wegner, 1989; Wegner, Schneider, Carter, & White, 1987; Wegner & Smart, 1997). This led to the hypothesis that thought suppression may be involved in the development and/or maintenance of persecutory delusions (PDs). The results of this study provided the first evidence of a relation between thought suppression and persecutory delusion-like beliefs (PDLBs). No main effect of thought suppression was found, but instead thought suppression was found to interact with anxiety in predicting levels of PDLBs. Thought suppression only predicted levels of PDLBs when anxiety levels were high. This study also found that anxiety, but not negative affect, accounted for a significant amount of unique variance in PDLBs, supporting Freeman’s (2007) contention that anxiety is the key emotion in persecutory ideation.

This second study (Chapter 2) examined interactions among factors proposed to be specifically associated with first- or second-stage factors of persecutory delusion.
formation. Through its focus on these interactions, this study performed a test of the two-factor model of PDLBs. In line with this model, the jumping to conclusions (JTC) bias was found not to be an independent predictor of PDLBs. Also in line with predictions, an interaction was found between a first-stage factor, social rank—which has been proposed to contribute to seeding the initial implausible idea—and a second-stage factor, the JTC bias, which is proposed to encourage the uncritical acceptance of such beliefs. This finding was replicated for two separate measures of PDLBs, strengthening confidence in the reliability of this conclusion.

**Hallucinations and intrusions**

Chapters 3 and 4 investigated the proposal that thought suppression and ensuing intrusive thoughts, would also be implicated in hallucinatory experiences. Chapter 3 began by noting that research into a little-studied form of hallucinations, those associated with the hypnagogic and hypnopompic (H&H) states, was limited by the lack of a brief, psychometrically reliable and valid self-report assessment tool. This study led to the development of the Durham Hypnagogic and Hypnopompic Hallucinations Questionnaire (DHQ) which was found to have satisfactory psychometric properties. This tool had a three-factor structure, indexing auditory H&H hallucinations, visual H&H hallucinations, and the experience of a felt presence. When scores on this new instrument were analysed in relation to measures of the tendency to experience cognitive intrusions it was found that transliminality, a measure of the extent to which material, typically processed unconsciously, is able to cross the threshold into consciousness, was positively related to all subscales of the DHQ. However, only auditory (not visual or felt-presence) H&H experiences were associated with the self-reported susceptibility to intrusive thoughts and tendency to
undertake thought suppression. It was proposed that this finding could be taken to suggest that different explanatory models are needed for each modality of H&H phenomena.

Chapter 4 examined the relation between thought suppression, intrusive thoughts and general hallucination-proneness (i.e., not limited to the H&H states). Fowler et al. (2006) had proposed that rumination or inner dialogues about interactions with the people involved in previous traumatic events could form the raw material of some AVHs. This study constituted the first direct empirical test of the relation between rumination and hallucination-proneness. A model was developed where rumination led to hallucinations due to its propensity to directly cause intrusive thoughts, and also to indirectly cause intrusive thoughts through the encouragement of thought suppression. Using path analyses it was found that the relation between rumination and hallucination-proneness was not direct but rather mediated by intrusive thoughts. These findings were consistent with a model of hallucination-proneness that sees intrusive thoughts playing a key role, with levels of intrusive thoughts being influenced by ruminative and thought suppression processes. Given that the measure of hallucination-proneness employed was heavily weighted towards AVHs, it was suggested that this model was likely to be more appropriate for AVHs than visual hallucinations.

**Hallucinations and agency**

Chapter 5 turned to a consideration of how self-produced thoughts and ruminative inner dialogue, highlighted in the previous chapter as candidates for the raw material of AVHs, may come to be experienced as alien. This chapter offered an integration of the neurocognitive action self-monitoring system (NASS) proposed by
General Discussion

Blakemore, Wolpert, and Frith (1998), with Wegner’s (2002) concept of apparent mental causation, and an application of this to the phenomenon of hallucinations. Central to this analysis was the idea of a predicted state, a prediction created by the brain of what will happen if a motor plan is executed, which is proposed to be involved in the creation of the felt-emotion of causing an event (agency). A critique was presented of Seal, Aleman, and McGuire’s (2004) proposal that an absence (or distortion) of the predicted state mechanism causes a cognition to be experienced as unintended, which is then resolved into a feeling of self or other authorship by preconscious attributions. Instead it was proposed, contra Seal et al., that cognitions involved in AVHs do not require that there be a feeling of unintendedness, the ambiguity of which must then be resolved into self/other authorship by preconscious attributions. Instead it was argued that cognitions come with a self/other label attached, as a result of the operation of the predicted state mechanism. The benefits of a Vygotskian, developmental approach to inner speech for the understanding of AVHs were highlighted. However, it was noted that a key problem, which Gallagher (2004) has termed the selectivity problem, remained unanswered. Specifically, if inner speech is the raw material of AVHs, why is it only some, not all, inner speech utterances in such individuals that are experienced as AVHs?

Chapter 6 then performed an empirical test of the predictions of the model developed in Chapter 5. It was noted that there are at least two potential ways in which the predicted state mechanism could lead to a breakdown in the feeling of authorship. Firstly, there may be a problem upstream of the predicted state, resulting in a complete or partial failure to generate a predicted state. Secondly, there may be a problem downstream of the predicted state. In this latter case, the creation of the predicted state may be normal, but the individual may be unable to use this
information to generate the feeling of agency. The Wheel of Fortune procedure (Aarts, Custer, & Wegner, 2005) utilised in this study offered a way of distinguishing which of the above possibilities is the best explanation of passivity experiences and AVHs. It was proposed that that the extent to which individuals could have their experience of causing an event enhanced by subliminal primes could be seen as a proxy measure of their ability to utilise predicted states. The results of the study showed that there was no significant correlation between participants' susceptibility to having their experience of causation enhanced by subliminal primes and either their levels of self-reported passivity experiences or AVH-proneness. However, it was noted that there was a trend for women who were less able to utilise effect primes to be more hallucination-prone. Furthermore, a main effect of priming was found in women and not in men. It was proposed that this may be due to women's increased propensity to take the intentional stance.

Hallucinations and inner speech

The final section of the thesis addressed multiple issues surrounding inner speech and AVHs. The study reported in Chapter 7 aimed to replicate the findings of Ford et al. (2001) which found dampening of the N1 response to auditory stimuli during inner speech, as compared to a silent baseline task. It was found that dampening of the N1 response occurred in all inner speech conditions employed (subvocal rehearsal, dialogic inner speech, and auditory verbal imagery). However, the finding of a dampened N1 response during mental rotation suggested that the dampening seen in response to inner speech tasks was not specific to the concurrent performance of inner speech, but was instead due to a reduction in attention to the auditory stimuli. These findings were taken as evidence that caution should be taken
in interpreting Ford et al.’s (2001) findings as evidence of a corollary discharge mechanism being defective in voice-hearing patients with schizophrenia. Instead it was suggested that the specificity of decreased dampening of the N1 response during inner speech by such patients may reflect underlying attentional differences in this group.

Having found electrophysiological evidence in Chapter 7 which questioned Ford et al.’s (2001) claim to have provided evidence in support of inner speech models of AVHs, the final chapter of the thesis (Chapter 8) evaluated inner speech-based models of AVHs on the basis of what was termed an argument-from-phenomenology. Specifically, it was investigated whether AVHs were phenomenologically consistent with inner speech. It was firstly argued that a competing model, which conceived of AVHs as a failure to inhibit memories of prior events (Badcock, Waters, & Maybery, 2007; Waters, Badcock, Michie, & Maybery, 2006), was only consistent with the phenomenology of a minority of AVHs, such as the approximately 10-20% (Fowler, 1997; Hardy et al., 2005) of individuals whose voices include content which can be linked directly back to memories of trauma.

In contrast to this, it was argued that many facets of the phenomenology of inner speech, including its regulatory nature, its linkage to ongoing events, and its tendency to involve the voices and perspectives of others, were consistent with the phenomenological properties of a large number of AVHs. However, it was also noted that inner speech-based models had a number of limitations when compared to the phenomenology of AVHs. They did not seem appropriate for the aforementioned ~10-20% of individuals whose voices have content which can be linked directly back to memories of trauma. These instead appeared better modelled as verbatim intrusions from memory. Inner speech models could also not account for other types of AVH,
such as the voices of crowds, or other auditory non-verbal hallucinations, such as environmental noise and music. Despite these limitations, it was argued that inner speech-based models had the starting advantage of being in accordance with the phenomenology of a significant number of AVHs.

An argument for the subcategorisation of auditory hallucinations was then made. The first category of AVH highlighted was that with content directly linked to intrusive memories of early abuse/trauma, which it was suggested were best understood via a neurocognitive model that saw AVHs as a failure to inhibit memories. The second category was auditory hallucinations involving seemingly random auditions, such crowds, noises, and music. It was proposed that these could be best explained at the neurological level, using an ictal-based (random seizure activation) bottom-up neurological model. Finally, a class of AVHs was identified, typified by novel statements that attempt to regulate the actions of the voice-hearer, which were linked to their ongoing activities, and that could not be linked directly to a verbatim memory. It was proposed that these AVHs could be best accounted for by a neurocognitive inner speech-based model. It was also noted that AVHs tend to evolve over time. This evolution of AVHs and AHs over time, currently a neglected and poorly understood area, was termed their dynamic developmental progression (DDP).

**Directions for future research**

*Persecutory ideation*

Chapters 1 and 2 of this thesis suggest a number of fruitful avenues for future research into persecutory ideation, and novel interventions for those who seek assistance for the distress such beliefs cause. Firstly, further research is needed to establish whether thought suppression plays a causal role in the development and/or
maintenance of clinically relevant PDs. Such a proposal could be experimentally tested through an examination of levels of thought suppression in patients with PDs and patients with generalised anxiety disorder. The latter would be a suitable control group as they show comparable anxiety levels to those with PDs (Freeman & Garety, 1999). If thought suppression plays a causal role in PD formation, it can be hypothesised that patients with PDs would have higher thought suppression levels than patients with generalised anxiety disorder.

Another approach would be to examine thought suppression levels longitudinally in individuals at high risk of developing PDs (for example those with a family history of psychosis), with a view to determining whether the presence of thought suppression predicts later onset of dysfunctional beliefs. If a causal role for thought suppression could be established, then one therapeutic possibility may be to encourage patients to openly express the thoughts they have been suppressing. In light of Sparrow and Wegner's (2006) findings that intrusive thoughts may be deactivated by merely expressing them, this would be expected to have beneficial effects for those with persecutory ideation. Such a study could also help establish whether thought suppression does play a causal role in the maintenance of such beliefs.

Secondly, the two-factor model requires empirical testing in those with clinically relevant PDs. One way in which this model could be tested would again be through a comparison of the presence of the JTC bias in patients with PDs, and patients with generalised anxiety disorder (but no persecutory ideation). The two-factor model would predict that, although social rank and other factors associated with anxiety and threat perception may not differ between these two groups, greater levels of the JTC bias would be found in the patients with PDs.
Thirdly, a multidimensional model of persecutory ideation (Freeman, 2007), which takes into account factors such as the conviction with which the belief is held and the distress it causes, offers further ways of considering how factors such as the JTC bias may impact upon persecutory ideation. Given that the JTC bias is thought to be involved in converting the initial implausible ideas associated with persecutory ideation into uncritically accepted beliefs, it may be hypothesised that levels of the JTC bias will directly impact upon the conviction with which PDs are held, but not on the levels of associated distress. Studies of such relations would be a first step towards enabling cognitive-behavioural interventions to take place at the level of specific dimensions of persecutory ideation.

The finding that the JTC bias was associated with increased levels of PDLBs in those with low social rank, suggests that direct attempts to correct the JTC bias may prove beneficial. Techniques for doing this could be based around cognitive behavioural interventions or forms of mindfulness training (e.g., Baer, 2006). In addition to interventions which try to directly alter reasoning styles, other more indirect psychosocial interventions may also prove important. For example, Freeman (2007) has suggested that social isolation may contribute to a failure to fully review persecutory thoughts. This has the implication that reasoning biases may be improved by increased social interaction. This highlights the potential importance of service-user led organisations, such as the Paranoia Network, which offers peer-support to those with severe persecutory ideation. Such service-user led groups offer those with PDs a route away from social isolation, and entrenchment of such persecutory beliefs. This is in line with evidence that interventions based around increased social interaction in supportive, peer-led, non-medicalised settings may be beneficial in
helping individuals recover from psychotic experiences (e.g., Mosher & Menn, 1978; Mosher, Vallone & Menn, 1995).

The finding that social rank was related to levels of PDLBs, both as an independent predictor and through its interaction with the jumping to conclusions bias, is a finding that should have important implications for future research. Firstly, it holds out the potential that psychosocial interventions which aim to build up social competence (e.g., Gould, Buckminster, Pollack, Otto, & Yap, 2006), which may help improve perceptions of social rank, may play a valuable role in reducing persecutory ideation. Also, given the positive correlation between social rank and serotonin levels found in primates (Raleigh, McGuire, Brammer, Pollack, & Yuwiler, 1991), this offers the prediction that selective serotonin reuptake inhibitors (SSRIs) may be effective in decreasing persecutory ideation. However, problems of treating persecutory ideation with SSRIs have been noted (Kantor, 2004), and psychosocial interventions targeted at social rank would appear to be preferable.

**Hypnagogic and hypnopompic hallucinations**

The development of the DHQ should allow a range of hypotheses in this area to be tested. Firstly, due to the demonstrated association between trauma and AVHs in clinical and non-clinical samples (Read & Argyle, 1999; Morrison & Peterson, 2003) it may be hypothesised that trauma contributes to an increased probability of experiencing hypnagogic and hypnopompic (H&H) hallucinations. This could be simply tested through an examination of the relation between levels of trauma and DHQ scores. However, trauma would need to be carefully assessed through detailed interviews using standardised tools such as the Childhood Experiences of Care Abuse Questionnaire (Bifulco, Bernazzani, Moran, & Jacobs, 2005) rather than simple self-
report measures. Secondly, it may be hypothesised that H&H hallucinations constitute a risk factor for the development of hallucinations in clear, waking consciousness (Jones & Fernyhough, 2008). To test this hypothesis, it would be necessary to perform a longitudinal study of whether early H&H experiences predict later development of hallucinations in clear consciousness. If this were found to be the case, it would be important to attempt to pinpoint what factors lead some, but not all individuals to have hallucinations that make the transition into clear, waking consciousness. One possibility is that trauma may increase the risk of benign H&H hallucinations intruding into waking consciousness, although at this time such a proposal remains speculative. Thirdly, given that thought suppression has been found to result in greater numbers of intrusions in those with insecure attachment styles (Mikulincer, Dolev, & Shaver, 2004), it would be profitable to test the hypothesis that thought suppression is more likely to lead to H&H hallucinations in those with an insecure (as opposed to a secure) attachment style.

Finally, an intriguing question is to what extent such experiences can be consciously induced. Richard Feynman, the Nobel Prize winning physicist, reported an ability to linger on the borders of sleep and to thus consciously induce hypnagogic hallucinations (Feynman, 1985). This seems to offer an intriguing method of studying such experiences, which are usually fragmentary and brief (Mavromatis, 1988). A study in which participants are trained to remain in a semi-conscious state between wake and sleep, to induce hypnagogic hallucinations which can then be recorded in a bedside diary, may offer valuable insights in the phenomenology of the experience. Obtaining a more detailed understanding of the phenomenology of H&H experiences may help us understand the relation between H&H hallucinations and those experienced in clear, waking consciousness.
Trauma and hallucinations

Chapter 4 proposed a model in which rumination upon traumatic events may play a role in AVHs. This model raises a number of questions for our understanding of the relation between trauma and AVHs. Firstly, what factors differentiate those who experience traumatic events but do not later develop hallucinations, from those who do? At a biological level it would be valuable to examine whether there is a genetic predisposition for trauma to lead to later hallucinatory experiences. Specifically, it may be hypothesised that a gene-environment interaction exists, with a specific genotype conferring a significantly greater probability of experiencing hallucinations following trauma. A useful parallel can be drawn here with research into gene-environment interactions in the relation between cannabis and psychosis. Studies in this area have focussed on the COMT gene, as its product (catechol-O-methyltransferase) is involved in the metabolism of dopamine, which has been implicated by many studies to play a role in psychosis (Kapur, 2003). In brief, a specific codon on the COMT gene may involve one of three potential combinations of valine (Val) and methionine (Met), Val-Val, Val-Met, or Met-Met. Those with the Val-Val form of the gene break down dopamine more slowly than those with the Val-Met form of the gene, who in turn break down dopamine more slowly than those with the Met-Met form of the gene (Männisto & Kaakkola, 1999). It has been found that a diagnosis of schizophrenia is significantly more likely to be received later in life in cannabis users who have a Val-Val combination form of the COMT gene, as opposed to Val-Met, or Met-Met forms (Caspi et al., 2005). The COMT gene would hence be a logical starting point for an examination of gene-environment interactions in the relation between trauma and hallucinations. Indeed, given the failure to establish clear
links between specific genes and psychiatric diagnoses, a complaint-based approach may be expected to be more profitable (Bentall, 2003; Crow, 2007).

Secondly, it may be asked what cognitive factors make trauma more likely to result in hallucinations. It has been argued in this thesis (Chapter 4) that rumination and thought suppression may play a role in encouraging the formation of hallucinations. However, a range of other factors such as attributional style, metacognitive factors, and source-monitoring deficits are also likely to be involved (Chapter 4). This highlights the need for a detailed longitudinal study to establish what cognitive factors prospectively predict whether later trauma results in the development of hallucinations. It may also be considered whether a 3-way gene-environment-cognitive style interaction may be important. It may be that those with a, as yet unknown, genetic predisposition to AVHs, who have experienced trauma, and who have a specific cognitive style which may include a tendency to ruminate, may be at greatest risk of developing hallucinations.

**Agency and hallucinations**

The failure to find a clear effect of subliminal priming using the Wheel of Fortune methodology (Aarts et al., 2005) employed in Chapter 6, suggested this methodology was not ideally suited to test the proposal that hallucinations result from a failure to utilise predicted states. This highlights the need to devise a simple, replicable task for assessing susceptibility to the illusion of conscious will, which can be shown to be reliable in a non-clinical sample before being extended to a clinical sample.

A recent study by Asai, Sugimori, and Tanno (2008) found that errors in predicted motor movements, specifically predicting where one's hand had moved to
when unseen, correlated with AVH-proneness in the general population. This can be seen to be consistent with the model advanced in Chapter 5, which carried the implication that problems with predicted motor actions should be experienced with hallucinations. The model proposed in Chapter 5 would also predict that impairments in motor prediction skills (as assessed by the task employed by Asai et al.) should be associated with the presence of AVHs, but not persecutory ideation. Future research is required to test such a proposal.

Subcategorisation of AVHs and dynamic developmental progression

The proposal that subcategorisations of AVHs are necessary (Jones, in press; see Chapter 8), each involving different underlying neurocognitive mechanisms, leads to a number of falsifiable hypotheses. Firstly, different patterns of neural activation would be likely to underlie these different forms of AVHs. For example, a patient with an AVH identifiable as a verbatim memory of abuse would be hypothesised to show a different pattern of neural activation to a patient with a more dynamic AVH which commented on current actions. The first type of AVH may show activation more similar to that seen during traumatic autobiographical memory recall. As Broca’s area activation decreases during recall of traumatic (as compared to neutral) memories in patients with PTSD (Rauch et al., 1996), and increases during inner speech production (Shergill et al., 2001), less activation should be seen in this area during AVHs involving intrusive memories of abuse, than during AVHs involving novel comments on current actions.

The concept of a dynamic developmental progression (DDP) also opens up a number of avenues for future research. For example, given that the experience of AVHs stretches into the healthy population (Aleman & Laroi, 2008; see General
General Discussion

Introduction, it may be fruitful to consider how (and to what degree) AVHs may evolve from brief, benign hallucinatory experiences that can be considered more or less normal, to a distressing clinically relevant experience. The identification of factors that engender a transition from benign to malign AVHs would likely have clinical implications for management of such AVHs. There is hence the need for more longitudinal studies of the DDP of voices, and factors that prospectively predict the course that AVHs will take.

Shared mechanisms in hallucinations and delusions

The studies in this thesis, following a complaint-based account of psychotic experiences (Bentall, 2006; see General Introduction), have focussed on studies of either AVHs or PDLBs. However, it is well known that levels of hallucinations correlate with levels of persecutory ideation in clinical and non-clinical sample (e.g., Bell, Halligan, & Ellis, 2006). This is suggestive that such experiences and beliefs may share some common causal factors. For example, this thesis has already shown links between thought suppression and both PDLBs (see Chapter 1, Chapter 2) and hallucination-proneness (see Chapter 4). Other factors are also likely to be associated with both PDLBs and hallucination-proneness. For example, this thesis found a link between rumination and hallucination-proneness (Chapter 4). Given that social rank is associated with both PDLBs (Chapter 2), and rumination (Cheung, Gilbert, & Irons, 2004), it may be that rumination plays a role in persecutory ideation, as well as hallucination-proneness. Thus, when performing studies of specific complaints such as hallucinations, researchers may also wish to assess how other correlated complaints, such as persecutory ideation, can be incorporated into the model of the primary complaint under investigation.
Interventions for those with hallucinations

A key goal of research into hallucinations is to develop interventions that can help individuals with such experiences. For those with hallucinations, such intervention may be divided into two categories based upon Romme’s proposal that voice-hearers are in need of “liberation, not cure” (Romme, as cited in Bentall, 2003, p. 511). While one class of interventions may aim at eliminating hallucinations, a second may simply aim at helping the individual to manage and cope with the experience better. In terms of eliminating hallucinations, the studies reported in the present thesis (Chapter 3; Chapter 4) suggest that one way to help reduce hallucinations is to prevent attempted suppression of thoughts of trauma and related cognitions. It would hence be useful to trial the use of acceptance-based approaches to intrusive cognitions (e.g., Marcks & Woods, 2005) with those with hallucinations.

As noted in the General Introduction, there is a long historical tradition of understanding hallucinations outside the context of a mental illness discourse, and not as a symptom of a mental illness. Following the approach of the UK Hearing Voices Network, who run groups for those with AVHs aiming to normalise the experience and allow sharing of coping strategies, there have been some randomised controlled trials of group cognitive-behavioural therapy (CBT) based along the same lines. However, Wykes et al. (2005) found that group CBT did not reduce either the frequency of AVHs, or the distress associated with them. The arguments developed in this thesis suggest a number of reasons why such trials may not have been effective. Firstly, it may be that trauma was involved in the development of the participants’ AVHs. A group-based intervention may not have facilitated discussion of such sensitive issues. This would have prevented the CBT from touching on key emotional
triggers behind the AVHs, and/or discouraged the introduction of alternative cognitive strategies, such as prevention of thought suppression.

The present considerations thus suggest some possible improvements to this form of CBT. Firstly, the therapy in Wykes et al.'s study lasted only seven weeks. This seems a short period for the group members to identify, and gain the confidence to talk about, any related traumatic events, and change how the experiences were dealt with. Secondly, it may be that one-to-one therapy would prove more beneficial in detailing with complex and often emotionally sensitive precursors to the experience. Thirdly, the CBT facilitators were therapists from the local mental health service who had been trained to administer this therapy programme in a manualised format. It may prove more effective to have such sessions delivered by ex-service users who may better be able to communicate the experience of hearing voices, give more hope for recovery, and remove the element of medicalisation from the sessions. For such reasons, service-user led sessions may prove more effective than clinician led-sessions (Mosher & Mann, 1978). This remains an issue to be addressed by future research.

Hope

Throughout the course of researching the material in this thesis, I have encountered many reports from service-users, individuals with AVHs and PDs, of being told by mental health professionals that they will "never recover" (Dillon, 2008), "never work again" (Alcock, 2009), that they "do not know what is best for [them]" (Dillon, 2008) and having being treated with a "total lack of hope" (Hilpem, 2007). The evolution in our scientific and political conceptions of such experiences, driven by both academics (e.g., Bentall, 2003) and service-users (e.g., Romme &
Escher, 1993; James, 2001), means there is reason to believe such attitudes are in the process of disappearing. Not only is there hope, but *contra* Obama (2008), it is no longer audacious to hope. Hope is a reality.
References


APPENDICES
Appendix A

A Message in the Medium?

Assessing the Reliability of Psychopathology E-questionnaires
A message in the medium? Assessing the reliability of psychopathology e-questionnaires

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Received 29 January 2007; received in revised form 19 August 2007; accepted 23 August 2007
Available online 10 October 2007

Abstract

Internet-based questionnaires (e-questionnaires) have become widely used in psychopathology research, but there is currently little evidence on their reliability as compared with pen-and-paper equivalents. Data from separate samples of young adults completing e-questionnaires on hallucination-proneness (N = 751) and persecutory ideation (N = 183) were compared with data from pen-and-paper versions of the same questionnaires completed by a third, unrelated sample (N = 188). There was no effect of modality of presentation on mean scores on these questionnaires. Multi-group confirmatory factor analysis indicated mode invariance of factor structure for the persecutory ideation questionnaire. Internal reliability of both questionnaire formats was satisfactory. We conclude that the administration of traditional measures of psychopathology via e-questionnaire is a reliable method of data collection.

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Keywords: E-questionnaires; Hallucination; Pen-and-paper; Persecutory delusions; Psychopathology; Reliability

1. Introduction

The use of internet-based questionnaires, also known as “e-questionnaires” (Denscombe, 2006, p. 252), as a research tool has increased significantly over the past decade. Compared with their traditional pen-and-paper counterparts, e-questionnaires are relatively inexpensive, easy to administer,
can easily cover wide geographical areas, and tend to have higher completion rates (Denscombe, 2006). Furthermore, this method of data collection avoids a number of the disadvantages of standard pen-and-paper administration, such as the potential for participants to fail to answer questions and the time-consuming need to transcribe data (Mangunkusumo et al., 2005). Responses given anonymously via e-questionnaires, when compared to anonymous mail-returned pen-and-paper methods, have been found to result in lower scores on social desirability measures (Joinson, 1999), and have been found to generate greater self-disclosure from participants (Stanton, 1998).

E-questionnaires are typically on-line versions of widely used, popular questionnaires that have been shown to have good psychometric properties when completed by participants via pen-and-paper methods. However, it cannot be assumed a priori that the psychometric properties of an internet-based version of tried and tested questionnaires will be identical to those of the pen-and-paper version (Ritter, Lorig, Laurent, & Matthews, 2004). There may exist what has been termed a “mode effect”, a differential pattern of responding by participants depending on the medium of administration of the questionnaire (e.g., Denscombe, 2006; McCabe, 2004). Richman, Kiesler, Weisband, and Drasgow (1999) have noted that certain properties of e-questionnaires such as the display of plain text on a screen, the ephemeral nature of responses, and the absence of social context cues may affect participant response patterns. However, preliminary evidence suggests that this mode effect is not significant (Richman et al., 1999). For example, studies have compared the data obtained from questionnaires administered both as e-questionnaires and via pen-and-paper format in areas such as personality (Buchanan & Smith, 1999), health assessment (Ritter et al., 2004), illicit drug taking (McCabe, 2004), and even library quality (Hayslett & Wildemuth, 2004). Such studies have not found significant differences in participant responses.

One area in which e-questionnaires are likely to be particularly useful is in the assessment of psychopathological experiences in the healthy population. The study of such experiences has been given a renewed momentum by findings that they exist on a continuum extending into the normal population (Johns & van Os, 2001; Peters, Joseph, Day, & Garety, 2004). Finding reliable self-report instruments for assessing these experiences is thus likely to be important for the future development of research in this area. Given that psychopathology questionnaires are likely to be particularly sensitive to social desirability effects, it is possible that e-questionnaire versions of standard pen-and-paper psychopathology questionnaires may result in significantly different patterns of response. Investigating whether there is a mode effect for psychopathology questionnaires would therefore seem to be particularly timely given the number of studies that are starting to use this method (e.g., Allen et al., 2005; Berry, Wearden, Barrowclough, & Liversidge, 2006; Jones & Fernyhough, 2006, 2007).

The present study set out to examine if a mode effect was present when standard measures of psychopathology in the healthy population were administered as e-questionnaires. Equivalence between the two modes of administration was to be assessed in two ways. Firstly, the means of the psychopathology instruments were to be compared by mode of administration. This is a popular method of establishing mode equivalence of questionnaires (e.g., Cronk & West, 2002). We hypothesized that, due to the lower social desirability scores and greater self-disclosure associated with e-questionnaires, self-reported proneness to these unusual experiences would be higher than would be expected with pen-and-paper administration.

We also set out to examine if the factor structures of the psychopathology questionnaires differed by mode of administration. If equivalent factor structures are found for both modes of
administration this offers evidence that the same constructs are being measured in each mode (Buchanan & Smith, 1999). This comparison was to be performed using multi-group confirmatory factor analysis. We hypothesized that the factor structures of the psychopathology questionnaires employed here would be mode invariant. Finally, it was hypothesized that the e-questionnaires would be as satisfactory in their internal reliability as their pen-and-paper counterparts.

2. Method

2.1. Participants

2.1.1. Internet administration
A circular e-mail was sent to undergraduates enrolled on a variety of programs at a British university informing them of a website where they could take part in a study. Participants were not offered an incentive to take part in the study, and responses were anonymous with only basic demographic data (age, gender, and university department) being requested. As a result of this 751 undergraduates (328 men, 423 women) completed an e-questionnaire assessing hallucination-proneness. (Note that descriptive statistics for the e-questionnaire hallucination-proneness data reported for this sample were previously reported in Jones & Fernyhough, 2006). The mean age of participants was 20.1 years (SD = 2.6). A separate sample of 183 undergraduates (107 men, 76 women) completed an e-questionnaire assessing levels of persecutory delusion-like beliefs. The mean age of these participants was 18.5 years (SD = 1.3).

2.1.2. Pen-and-paper administration
A convenience sample of 188 undergraduates (78 men, 110 women) enrolled on a variety of programs at a British university completed questionnaires assessing hallucination-proneness and delusion-proneness. The mean age of the participants was 20.6 years (SD = 3.2). Participants' responses were anonymous with only basic demographic data (age, gender, and department) being requested. No incentive was offered for participation. There was no overlap between participants in the pen-and-paper sample and the two e-questionnaire samples.

2.1.3. Design of questionnaires
The pen-and-paper and e-questionnaire formats had identical questions, presented in identical orders. The e-questionnaire was designed to be as similar in appearance as possible to the pen-and-paper format. As such, responses on the e-questionnaires did not involve drop-down menus, but simply required participants to click on a box which they would normally tick on the pen-and-paper questionnaires.

2.2. Measures

2.2.1. Proneness to persecutory delusion-like beliefs
The Persecutory Ideation Questionnaire (PIQ; McKay, Langdon, & Coltheart, 2006) is a 10-item questionnaire designed to measure persecutory ideation in both clinical and non-clinical samples. Items are rated on a 5-point Likert scale ranging from “Very True” (4) to “Very Untrue” (0).
Total scores can range from 0 to 40. Higher scores indicate greater proneness to persecutory delusion-like beliefs. This measure has been shown to have good reliability and validity (McKay et al., 2006).

2.2.2. Proneness to hallucinations

The revised Launay-Slade Hallucination Scale (LSHS-R; Launay & Slade, 1981; modified by Bentall & Slade, 1985) is a 12-item instrument designed to measure predisposition to hallucination-like experiences. Each item is scored on a 5-point Likert scale ranging from "Certainly applies to me" (4), to "Certainly does not apply to me" (0). Total scores can range from 0 to 48. Higher scores indicate a greater predisposition to hallucination-like experiences. This measure has been shown to have good reliability and validity (Bentall & Slade, 1985).

3. Results

3.1. Mean score comparisons

3.1.1. PIQ

The age of the PIQ paper-and-pen group was significantly higher than the PIQ e-questionnaire group, \( t(369) = 8.08, p < .001 \). There was also a significant gender difference between the two groups, \( \chi^2 = 10.7, p < .01 \), with a greater proportion of females in the paper-and-pen group. Cronbach's alpha for the PIQ delivered as an e-questionnaire was .88, and through traditional pen-and-paper administration it was .85. Descriptive statistics are presented in Table 1. Scores on the PIQ for both modes of questionnaire administration were comparable to the mean of 9.11 (SD = 5.95) found by previous research (using pen-and-paper administration) on undergraduate populations (McKay et al., 2006).

<table>
<thead>
<tr>
<th>Item #</th>
<th>E-questionnaire</th>
<th>Pen-and-paper</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Male (( n = 107 ))</td>
<td>Female (( n = 76 ))</td>
</tr>
<tr>
<td>PIQ Total</td>
<td>6.77 (4.35)</td>
<td>7.82 (6.04)</td>
</tr>
<tr>
<td>Item #1</td>
<td>0.69 (.79)</td>
<td>0.77 (.93)</td>
</tr>
<tr>
<td>Item #2</td>
<td>0.76 (.65)</td>
<td>0.84 (.63)</td>
</tr>
<tr>
<td>Item #3</td>
<td>0.61 (.62)</td>
<td>0.68 (.76)</td>
</tr>
<tr>
<td>Item #4</td>
<td>0.71 (.71)</td>
<td>0.92 (.90)</td>
</tr>
<tr>
<td>Item #5</td>
<td>0.32 (.54)</td>
<td>0.59 (.83)</td>
</tr>
<tr>
<td>Item #6</td>
<td>1.34 (.84)</td>
<td>1.31 (1.00)</td>
</tr>
<tr>
<td>Item #7</td>
<td>0.68 (.70)</td>
<td>0.76 (.84)</td>
</tr>
<tr>
<td>Item #8</td>
<td>0.31 (.50)</td>
<td>0.61 (.90)</td>
</tr>
<tr>
<td>Item #9</td>
<td>1.06 (1.03)</td>
<td>0.97 (1.10)</td>
</tr>
<tr>
<td>Item #10</td>
<td>0.25 (.55)</td>
<td>0.34 (.85)</td>
</tr>
</tbody>
</table>

*Note. PIQ = Persecutory Ideation Questionnaire.*
A one-way analysis of covariance (ANCOVA) was performed with PIQ total score as the dependent variable, and mode of administration (e-questionnaire, pen-and-paper) as the independent variable. Age and gender were entered as covariates. Levene’s test of equality of error variances indicated that the assumption of homogeneity of variance was met, $F(1, 369) = 2.55$, n.s. Visual graphical inspection of the distribution of PIQ total scores and a Kolmogorov-Smirnov test indicated PIQ scores deviated significantly from normality. However, as ANOVA is robust to deviations from normality (Field, 2005) parametric analysis was deemed appropriate. The ANCOVA showed there was no effect of mode of administration on PIQ score, $F(1, 367) = 2.98$, n.s.

A one-way multivariate analysis of covariance (MANCOVA) was then performed with the ten individual PIQ items as dependent variables, and mode of administration as the independent variable. Levene’s test of equality of error variances indicated that the assumption of homogeneity of variance was met for all individual PIQ item scores, except Item #10. PIQ item scores were found to be non-normally distributed (as indicated by Kolmogorov-Smirnov tests and visual inspection), however, as MANOVA is robust to violation of this assumption (Tabachnick & Fidell, 2007) parametric analysis was performed. There was no multivariate effect of mode of administration on PIQ item score, $F(10, 353) = 1.12$, n.s. However, it has been argued that using correlated dependent variables in a MANOVA (as was the case with the individual PIQ items) reduces its power to detect effects (Tabachnick & Fidell, 2007). Hence, univariate analyses were also examined. Follow-up univariate tests were performed with a Bonferroni correction to take account of the number of separate ANCOVAs performed, with alpha set as $\alpha' = .005$ (.05/10). None of the univariate tests were significant. This was taken as evidence that the mean PIQ scores, both in total, and at item level, did not differ between the two modes of administration.

3.1.2. LSHS-R

The age of the LSHS-R paper-and-pen group was significantly higher than the LSHS-R e-questionnaire group, $t(937) = 2.20$, $p < .05$. There was also a significant gender difference between the two groups, $\chi^2 = 13.3$, $p < .001$, with a greater proportion of females in the paper-and-pen group. Cronbach’s alpha for the LSHS-R delivered as an e-questionnaire was .81, and through traditional pen-and-paper administration it was .78. Descriptive statistics are presented in Table 2. Scores on the LSHS-R for both modes of questionnaire administration were also comparable to the mean of 17.72 (SD = 7.49) found in undergraduate populations (by pen-and-paper administration) by Waters, Badcock, and Maybery (2003: $N = 562$).

A one-way analysis of covariance (ANCOVA) was performed with LSHS-R total score as the dependent variable, and mode of administration (e-questionnaire, pen-and-paper) as the independent variable. Again, age and gender were entered as covariates. Levene’s test of equality of error variances indicated that the assumption of homogeneity of variance was met, $F(1, 937) = 3.66$, n.s. Visual graphical inspection of LSHS-R score distribution suggested that they were not normally distributed, as did a Kolmogorov-Smirnov test. There was no effect of mode of administration on LSHS-R score, $F(1, 937) = .23$, n.s.

A one-way MANCOVA was then performed with the twelve individual LSHS-R items as dependent variables, and mode of administration as the independent variable. Levene’s test of equality of error variances indicated that the assumption of homogeneity of variance was met for all individual LSHS-R item scores, except item #8. There was a significant multivariate effect of mode of administration on LSHS-R item score, $F(12, 924) = 1.77$, $p < .05$, $\eta^2 = .02$. This is
### Table 2
Means (standard deviations) of LSHS-R in both modes

<table>
<thead>
<tr>
<th></th>
<th>E-questionnaire</th>
<th>Pen-and-paper</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Male</td>
<td>Female</td>
</tr>
<tr>
<td>LSHS-R total</td>
<td>17.52 (8.23)</td>
<td>16.67 (8.12)</td>
</tr>
<tr>
<td>Item #1</td>
<td>2.57 (1.29)</td>
<td>2.73 (1.25)</td>
</tr>
<tr>
<td>Item #2</td>
<td>1.19 (1.25)</td>
<td>.99 (1.19)</td>
</tr>
<tr>
<td>Item #3</td>
<td>2.01 (1.32)</td>
<td>1.56 (1.29)</td>
</tr>
<tr>
<td>Item #4</td>
<td>.67 (1.00)</td>
<td>.58 (1.00)</td>
</tr>
<tr>
<td>Item #5</td>
<td>2.73 (1.15)</td>
<td>2.54 (1.24)</td>
</tr>
<tr>
<td>Item #6</td>
<td>2.17 (1.29)</td>
<td>1.93 (1.34)</td>
</tr>
<tr>
<td>Item #7</td>
<td>1.28 (1.26)</td>
<td>1.09 (1.22)</td>
</tr>
<tr>
<td>Item #8</td>
<td>.16 (.60)</td>
<td>.19 (.67)</td>
</tr>
<tr>
<td>Item #9</td>
<td>1.23 (1.39)</td>
<td>1.50 (1.52)</td>
</tr>
<tr>
<td>Item #10</td>
<td>1.04 (1.20)</td>
<td>1.18 (1.28)</td>
</tr>
<tr>
<td>Item #11</td>
<td>1.89 (1.26)</td>
<td>1.89 (1.29)</td>
</tr>
<tr>
<td>Item #12</td>
<td>.52 (1.01)</td>
<td>.44 (.96)</td>
</tr>
</tbody>
</table>

*Note. LSHS-R = Revised Launay-Slade Hallucination Scale.*

classified as a small effect by Cohen's (1977) criteria. Follow-up univariate tests were performed with a Bonferroni correction, $\alpha' = .004 (.05/12)$. None of the univariate tests were significant. This was taken as evidence that the mean LSHS-R scores, both in total, and at item level, did not differ between the two modes of administration.

### 3.2. Factor structure comparisons

#### 3.2.1. PIQ

No previous studies have investigated the factor structure of the PIQ, and hence such methods have not yet been utilized to assess its psychometric properties. Initial analysis was performed on the pen-and-paper PIQ data set using exploratory factor analysis with principal components analysis. Inspection of the scree-plot of the eigenvalues (Cattell, 1966) suggested a one-factor solution. As the first two eigenvalues were 5.17 and .94, this conclusion was consistent with the number of factors to extract by both Kaiser's (1960) rule and, more importantly, parallel analysis. All items loaded (> .4) onto this factor, however, item 7 ('Some people try to steal my ideas and take credit for them') had a low communality of .27.

A confirmatory factor analysis (CFA) was performed using AMOS 6.0 to establish the fit of a one-factor solution to the PIQ for the pen-and-paper group. As noted in Section 3.1.2, scores on the PIQ deviated significantly from normality. The most common method used to estimate CFA model fit, maximum likelihood (ML) estimation, has been found to become increasingly impaired as non-normality increases (Curran, West, & Finch, 1996). With non-normal data ML typically leads to an increased chi-squared statistic, and is likely to lead to the rejection of accurate models (Curran et al., 1996). In response to this problem, Yuan and Bentler (1999) have developed an $F_{YB}$ statistic which is a modification of the asymptotically distribution free (ADF) estimation method. This has been shown to perform well with both non-normal data and sample sizes as...
The one-factor model of the PIQ was found to be a poor fit to the pen-and-paper data, \( F_{VB} \) (35, 113) = 1.88, \( p < .05 \), CFI = .93, RMSEA = .11. Modification indices (MI) indicated that improvement in the model could be achieved if the errors of items 8, 9, and 10 were allowed to be correlated. This indicated redundancy in two of these items. Items 9 and 10 were hence removed from the scale, with item 8 ('I sometimes feel I am being persecuted in some way') being retained. MI also indicated that the removal of item 7 would improve the model, and given its low communality reported above, this was also removed. The remaining seven-item PIQ (with items 1,2,3,4,5,6,8) was subject to CFA. This indicated a good fit to the data, \( F_{VB}(35, 113) = 2.12 \), n.s., CFI = .97, RMSEA = .08. The revised seven-item PIQ (PIQ-7) was hence seen to be a preferable psychometric tool to the original PIQ. The PIQ-7 had a Cronbach's \( \alpha = .88 \) for the paper-and-pen sample, and \( \alpha = .84 \) for the e-questionnaire sample.

Mode invariance of PIQ-7 factor structure was examined using a series of model comparisons between baseline, and increasingly constrained, models (Table 3). A non-significant change in model fit between models indicates that invariance exists on the new constraint (Byrne, 1998). Before multi-group analyses were performed, fit of the one-factor solution of the PIQ-7, derived from the pen-and-paper group, was examined for the e-questionnaire group. A good fit was found (see Table 3).

Nested multi-group CFAs were then performed (see Table 3). In the baseline model (Model A), the validity of the one-factor structure of the PIQ-7 across the pen-and-paper and e-questionnaire groups was assessed. A good fit was found for this model. In Model B, factor variance was constrained to be equal. There was no significant difference in the goodness of fit between Model A and Model B, suggesting that the factor variance was equivalent between the two groups. In Model C, factor loadings, as well as factor variance, were constrained to be equal. The addition of this constraint did not lead to a significant change in the goodness of fit of the model. It was hence concluded that the factor structure of the PIQ-7 did not differ between the pen-and-paper and e-questionnaire administered groups.

When the analyses performed in Section 3.1.1 examining mean scores for mode effects on the PIQ were performed for the PIQ-7, again no mode effect was seen on either PIQ-7 total score, or at an item-level analysis.

Table 3
CFA model Fit statistics for the PIQ-7

<table>
<thead>
<tr>
<th>Model</th>
<th>( df )</th>
<th>( F_{VB} )</th>
<th>( p )</th>
<th>( \Delta df )</th>
<th>( \Delta F_{VB} )</th>
<th>( p )</th>
<th>CFI</th>
<th>GFI</th>
<th>RMSEA</th>
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<tr>
<td>Pen-and-paper</td>
<td>14</td>
<td>2.12</td>
<td>n.s.</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>.97</td>
<td>.96</td>
<td>.08</td>
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<tr>
<td>Online group</td>
<td>14</td>
<td>1.37</td>
<td>n.s.</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>.98</td>
<td>.96</td>
<td>.06</td>
</tr>
<tr>
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<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>A (baseline)</td>
<td>28</td>
<td>1.59</td>
<td>( p &lt; .05 )</td>
<td>-</td>
<td>-</td>
<td>.97</td>
<td>.96</td>
<td>.05</td>
<td></td>
</tr>
<tr>
<td>B (variances)</td>
<td>29</td>
<td>1.68</td>
<td>( p &lt; .05 )</td>
<td>1</td>
<td>.09</td>
<td>n.s.</td>
<td>.97</td>
<td>.96</td>
<td>.05</td>
</tr>
<tr>
<td>C (variances and loadings)</td>
<td>35</td>
<td>1.77</td>
<td>( p &lt; .01 )</td>
<td>6</td>
<td>.09</td>
<td>n.s.</td>
<td>.96</td>
<td>.95</td>
<td>.06</td>
</tr>
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</table>
Table 4
Confirmatory factor analyses of LSHS-R factor structure

<table>
<thead>
<tr>
<th>Factor structure</th>
<th>Pen-and-paper</th>
<th>Internet</th>
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<tr>
<td></td>
<td>df</td>
<td>$F_{YB}$</td>
</tr>
<tr>
<td>One-factor</td>
<td>54</td>
<td>1.53*</td>
</tr>
<tr>
<td>Aleman et al. (2001)</td>
<td>53</td>
<td>21.53**</td>
</tr>
<tr>
<td>Waters et al. (2003)</td>
<td>52</td>
<td>4.56**</td>
</tr>
<tr>
<td>2-factor (present study)</td>
<td>53</td>
<td>1.56*</td>
</tr>
</tbody>
</table>

*p < .05.

**p < .01.

3.2.2, LSHS-R

The only study of the factor structure of the LSHS-R in its original English form with a non-clinical population ($N = 562$; Waters et al., 2003), and hence comparable to the present study, concluded that this instrument had a three-factor solution. This replicated the number of factors found by Aleman, Nieuwenstein, Böcker, and De Haan (2001) who examined the factor structure of a Dutch translation of the LSHS-R in a non-clinical population. However, the items that these two studies found to load onto these factors differed, as did the meaning of the factors themselves. Before mode effects of the LSHS-R factor structure could be examined it hence needed to be shown which of Waters et al.’s and Aleman et al.’s factor structures best fitted our data. CFAs were performed on our paper-and-pen group data for a basic one-factor solution, and the three-factor structures of Aleman et al. and Waters et al. This analysis is shown in Table 4. None of these factor structures was found to be a good fit to our paper-and-pen group data. As an exploratory exercise it was also examined if any of these factor structures fitted our e-questionnaire data. They did not (Table 4).

We hence performed exploratory factor analysis based on our paper-and-pen sample. Parallel analysis indicated that the extraction of 2-factors was appropriate. The only items to load onto the second factor were items 8 and 12, relating to experiencing the voice of the Devil and God respectively. A CFA was performed on the paper-and-pen data based on this two-factor structure. As indicated in Table 4, this model was a poor fit to the data. It was hence concluded that it was not meaningful to examine mode effects on factor structure as no structure could be established upon which any such comparison could be made.

4. Discussion

The present study set out to investigate whether the administration of questionnaires measuring psychopathology in healthy participants via either e-questionnaires or traditional pen-and-paper methods led to significantly different patterns of response. No mode effect was found for the PIQ either on PIQ total score or through analyses of individual PIQ item scores. Through the use of exploratory and confirmatory factor analytic techniques with the pen-and-paper group, the original ten-item PIQ was revised into a more parsimonious seven-item form (PIQ-7). This revised PIQ-7 showed satisfactory internal reliability in both modes of administration. In parallel with
Appendix A


our findings for the original PIQ, the PIQ-7 did not demonstrate a mode effect either in total or at item level. The one-factor structure of the PIQ-7 was found to be a good fit to both the pen-and-paper and e-questionnaire group data. This factor structure was also found to be mode invariant, not differing significantly between the pen-and-paper group and the e-questionnaire group when factor variance and loading was constrained. We hence conclude that there is no evidence of a mode effect for the PIQ-7.

There was no mode effect for LSHS-R total score. A significant multivariate effect was found when item level analysis was performed; however follow-up univariate tests indicated that none of the LSHS-R items showed a mode effect. No factor structure could be found that was a good fit to the LSHS-R, and hence factor structure mode invariance could not be assessed. The LSHS-R showed satisfactory internal reliability in both modes of administration.

We conclude that the present study provides no evidence that the administration of traditional measures of psychopathology via e-questionnaire is an unreliable method of data collection in the healthy population. The present study has also highlighted a number of psychometric properties of the PIQ and LSHS-R. We have shown the original ten-item version of the PIQ (McKay et al., 2006) may be refined into a more parsimonious seven-item version, the PIQ-7. However, it remains for future research to assess other psychometric properties of this tool (such as test–retest validity). The present study has also highlighted problems in establishing a reliable factor structure for the LSHS-R. This suggests that if research requires measurement of separate aspects of sub-clinical hallucination proneness the development of dedicated instruments may be required.

A number of potential caveats need to be examined in regard to these conclusions. Firstly, the study was not a repeated-measures design. Although age and gender were controlled for, this still leaves the possibility that other differences between the groups who completed the e-questionnaires and the paper-and-pen versions could have obscured any effects of mode of administration. However, as convenience samples from similar demographics were used for each group, there is no reason to believe that this alone could account for our findings. In addition, we note that the use of a repeated-measures design to investigate this problem would introduce the potential confound of administering the two versions of the instruments at different time-points, leading to the risk of conflating test–retest differences in scores with any evidence for a mode effect. Secondly, while it is possible that individual participants may have completed the e-questionnaires more than once, as no incentive was offered for participation it appears highly unlikely that this would have occurred.

In summary, with their ability to produce inexpensive, complete, easily transcribed data, e-questionnaires are likely to prove an efficient tool for data collection in the future. It is however, worth considering if this conclusion is likely to be generalizable to populations with clinically relevant psychopathology. Such populations are likely to have fewer years of formal education than the healthy population (e.g., Sumich, Chitnis, & Fannon, 2002) and correspondingly less experience of information technologies. These differences in background, in conjunction with clinically relevant delusional ideation, might affect such individuals' perception of the anonymity and privacy (or lack thereof) of e-questionnaires. Any study proposing to utilize e-questionnaires in samples with clinically relevant psychopathology (such as patients with schizophrenia in an out-patient setting) would hence be advised first to investigate the presence of such mode effects.
Acknowledgements

The authors would like to thank Steven Muncer for his advice during the development of this article. Simon R. Jones is supported by an Economic and Social Research Council and Medical Research Council Interdisciplinary Research Studentship. Lee de-Wit is supported by an Economic and Social Research Council Studentship.

References


Appendix B

Neural Correlates of Inner Speech and Auditory Verbal Hallucinations: A Critical Review and Theoretical Integration
Neural correlates of inner speech and auditory verbal hallucinations: A critical review and theoretical integration

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Received 11 May 2006; received in revised form 15 August 2006; accepted 2 October 2006

Abstract

The neuroimaging and neurophysiological literature on inner speech in healthy participants and those who experience auditory verbal hallucinations (AVHs) is reviewed. AVH-bearers in remission and controls do not differ neurologically on tasks involving low levels of verbal self-monitoring (VSM), such as reciting sentences in inner speech. In contrast, on tasks involving high levels of VSM, such as auditory verbal imagery, AVH-bearers in remission show less activation in areas including the middle and superior temporal gyri. This pattern of findings leads to a conundrum, given that mentation involving low levels of VSM is typically held to form the raw material for AVHs. We address this by noting that existing neuroimaging and neurophysiological studies have been based on unexamined assumptions about the form and developmental significance of inner speech. We set out a Vygotskian approach to AVHs which can account for why they are generally experienced as the voice of another person, with specific acoustic properties, and a tendency to take the form of commands. On this approach, which we argue is consistent with the neural correlates evidence, AVHs result from abnormalities in the transition between condensed and expanded dialogic inner speech. Further potential empirical tests of this model are discussed. © 2006 Elsevier Ltd. All rights reserved.

Keywords: Auditory verbal hallucinations; Dialogue; Inner speech; Verbal self-monitoring; Vygotsky

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doi:10.1016/j.cpr.2006.10.001

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Causes of auditory verbal hallucinations (AVHs), the experience of perceiving speech without corresponding external stimulation (Stephane, Barton, & Boutros, 2001), are still not well understood. Any theory claiming to offer an explanation of AVHs must be able to explain why a voice is heard in the absence of an external stimulus. In addition, any such account must explain why such a voice is experienced as generated or authored by an individual other than one’s own self. A complete theory of AVHs must further be able to account for key features of the phenomenology of the experience. While the phenomenology of AVHs varies widely between different voice-hearers, broad themes can be extracted. One issue concerns the personal identity of the voice heard. While 28% of voice-hearers in a sample interviewed by Leudar, Thomas, McNally, and Glinski (1997) reported hearing only incognito voices, most (64%) reported hearing a voice which they could clearly identify as being that of a family member or acquaintance. Others, notably only those with a diagnosis of schizophrenia, reported hearing voices belonging to public figures.

Surveys of the numbers of different voices heard by voice-hearers have found an average of 2-3 voices (Leudar et al., 1997; Nayani & David, 1996). Ninety-six percent of voice-hearers in Leudar et al.’s (1997) sample reported being the target of the voice, which spoke to them rather than addressing another voice or another person. This highlights the fact that issues of the content and pragmatics of voices must also be addressed by a complete theory. Voices can attempt to perform a wide range of functions, including advising on possible actions, requesting specific actions, and inhibiting actions. However, AVHs most commonly take the form of commands, for example, ‘Get the milk’ or ‘Go to the hospital’ (Nayani & David, 1996). These command AVHs were reported by 84% of voice-hearers in Nayani and David’s phenomenological survey and appear common in the autobiographical literature too. For example, North (1990) recalls how, during the period of her life when she was diagnosed with schizophrenia, she heard distinct voices issuing commands such as, ‘Be good’, ‘Do bad’, ‘Stand up’, and ‘Sit down’ (p. 60).

In summary, any satisfactory account of AVHs must be able to explain the following aspects of the experience: (1) why the AVH-hearer experiences a voice in the absence of any external stimulus; (2) why this voice is experienced as generated/authored by an individual other than one’s own self; (3) why the voice is often perceived as having person-specific (e.g., acoustic) properties, distinct from one’s own; and (4) why AVHs have characteristic contents and pragmatics, particularly their tendency to appear as second-person assertive utterances (e.g., commands). To date, no theory has been entirely successful in accounting for all of these aspects of the phenomenon, with the third and fourth criteria, in particular, rarely addressed. Our aim in this article is to present a theoretical framework which, we suggest, is sufficiently broad to do justice to the rich and varied phenomenology of AVHs. In doing so, we attempt what Bentall (1990) recommends a scientific theory of hallucinations should do, namely, “explain the experiences of the hallucinator in terms of underlying cognitive mechanisms” (p. 83).

In setting out our arguments, we will be drawing upon an assumption which has guided much thinking on AVHs, namely that they are a form of inner speech (Bentall, 2003; Bick & Kinsbourne, 1987; Fernyhough, 2004; Jones & Fernyhough, in press; Leudar & Thomas, 2000; Maudsley, 1886; Seal, Aleman, & McGuire, 2004). A prima facie case for the involvement of inner speech in AVHs concerns a basic commonality between the two kinds of experience, namely that both involve some form of internal verbal mentalization, or ‘voice in the head.’ Furthermore, like inner speech, AVHs are often relevant to the voice-hearer’s ongoing activities, and may attempt to comment on or regulate behavior (Leudar & Thomas, 2000). While other explanations of AVHs are possible (e.g., Hoffman & McGlashan, 1997; Lennox et al., 2000), the strong conceptual and empirical support for a relation between the two phenomena suggests that considering AVHs as a disorder of inner speech remains a fruitful line of enquiry. In this article, we propose that further light may be shone on the puzzle of AVHs through a more careful investigation of the nature and developmental significance of inner speech. Our arguments will be informed by the claims of L. S Vygotsky (1934/1987) that inner speech can best be understood in terms of its being the endpoint of a developmental process. A guiding assumption, which we will argue is supported by empirical evidence from the study of children’s speech, is that inner speech in healthy individuals is irreducibly dialogic in nature. We contend that AVHs result from an abnormality in the typical process of inner speech production, specifically the transition between condensed and expanded varieties of inner speech. These ideas require substantial unpacking and will now be more fully explicated.

1. Neuroimaging of inner speech and AVHs

Those working within the cognitive neuropsychology tradition typically view experiences such as AVHs as being underpinned by specific cognitive deficits or biases, which in turn must have neural instantiation. As remarked, many authors have proposed that the cognitive deficit involved in AVHs is a disorder of inner speech. Such a proposal is
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supported by evidence that brain areas implicated in inner speech, such as the left inferior frontal gyrus (including Broca’s area) and the right temporal cortex (Huang, Carr, & Cao, 2001; Jones, in press; McGuire, Silbersweig, Murray, et al., 1996) are activated during AVHs (McGuire, Shah, & Murray, 1993; Shergill et al., 2004; Shergill, Brammer, Williams, Murray, & McGuire, 2000). Although some have questioned whether the finding of inferior frontal gyrus activation during AVHs is an artifact of using a button-press to signal onset of AVHs during imaging (Hunter & Spence, 2005), other studies which have not used a button-press to signal onset, but have instead asked post-imaging whether an AVH was experienced, have also shown inferior frontal gyrus activation in AVHs (Shergill, Brammer, et al., 2000). This said, several other imaging studies have failed to replicate the finding that inferior frontal gyrus activation is associated with the experience of AVHs (e.g., Lennox et al., 2000; Silbersweig et al., 1995). For the present we will make the working assumption that the neural substrates underlying inner speech are implicated in the genesis of AVHs. We will return to offer an explanation for these seemingly contradictory findings about the involvement of the inferior frontal gyrus later in this article.

Several specific proposals have been put forward within a cognitive neuropsychological framework to understand how atypical processing of inner speech may cause AVHs (Frith, Rees, & Friston, 1998; Jones & Fernyhough, in press; Seal et al., 2004). At the base of some of these proposals is the neuropsychological mechanism originally proposed by Frith and Done (1988), in which faulty monitoring of inner speech results in verbal thoughts not being recognized as one’s own and being perceived instead as someone else’s. The cognitive capacity responsible for monitoring inner speech is termed verbal self-monitoring (VSM). VSM has been proposed to operate through a corollary discharge system whereby discharges from the motor speech-producing areas in the frontal lobes ‘warn’ the auditory cortex that self-produced speech is about to be produced by dampening its response (Creutzfeldt, Ojemann, & Lettich, 1989; Ford & Mathalon, 2005; Martikainen, Kaneko, & Hari, 2005). Such a system may signal through the white matter tracts of the arcuate fasciculus which has pathways (among others) that originate in Broca’s area and project to Wernicke’s area (Hubl et al., 2004).

VSM can also be construed as entailing the appraisal of what one has said in relation to what one intended to say (McGuire, Silbersweig, & Frith, 1996). McGuire et al. (1996) used a PET imaging study to attempt to pin down the brain regions involved in VSM, by experimentally manipulating auditory verbal feedback while participants read aloud, and hence inducing a disparity between what the participant expected to hear and what they actually heard. McGuire et al. concluded that the VSM system resides primarily in the lateral temporal cortex bilaterally. This cortical region has been shown to be differentially activated by different tasks involving elicited verbal mentation (McGuire, Silbersweig, Murray, et al., 1996). Whereas silent reading does not activate this region, it is activated by participants’ imagining speech in another person’s voice (Shergill et al., 2001). While it seems plausible that generating verbal mentation in one’s own voice will be a relatively automatic process, mentally imitating another voice presumably requires external inspection of this imagined speech (necessary to assess whether the voice has the prosody, tone, pitch, and rhythms of the voice it is intended to be; McGuire, Silbersweig, Murray, et al., 1996), placing high demands on the VSM system. Greater demands of mentally imitating another voice are consistent with healthy participants rating such a task as subjectively more difficult than silent articulation (Shergill et al., 2001). The implication of the lateral temporal cortex in VSM accords with findings that the temporal lobe is associated with speech perception (Wise et al., 1991; Zatorre, Evans, Meyer, & Gjedde, 1992) and the phonological processing of heard words (Demonet et al., 1992). Furthermore, patients with left temporal lesions have been found to have impairments in imagining others speak (Zatorre & Hapler, 1993).

In addition to imaging studies, other techniques, such as EEG studies (e.g., Ford & Mathalon, 2005), have been employed to examine the neurophysiological signaling (corollary discharge) mechanisms involved in VSM. These techniques have also been applied to the study of individuals assumed to have a deficit in VSM, such as those who experience AVHs, with the result that a wide range of neuroimaging and neurophysiological studies with normal and AVH-hearing populations (primarily those with a diagnosis of schizophrenia) have been published. In the first part of this article, we review these findings and evaluate their significance for any comprehensive theory of AVHs.

1.1. Neuroimaging of silent articulation

A number of neuroimaging studies have been performed to investigate the neural correlates of silent articulation in healthy participants (e.g., Bullmore et al., 2000; Friedman et al., 1998; McGuire, Silbersweig, Murray, et al., 1996; Shergill et al., 2001). In Shergill et al.’s (2001) fMRI study, participants listened to single words, presented via...
headphones, read out by a neutral voice at 3 s intervals. In the baseline condition subjects were simply instructed to listen to each word carefully. In what was termed the Inner Speech condition, participants were asked silently to complete sentences of the form 'I like x' or 'I like being y' using the presented word. Functional MRI scans of brain activation during the Inner Speech condition were compared to baseline, and the Inner Speech condition was found to be associated with left-sided activation, in Broca's area (BA 44), Wernicke's area (BA 22), the SMA (BA 6), and the insula as well as the superior parietal lobe (BA 7) and right posterior cerebellar cortex. These findings accord with those of an earlier fMRI study by Bullmore et al. (2000), which found similar neural regions to be specifically associated with inner speech. In this experiment, participants were presented with a word, such as 'goat', during scanning, and then had to decide whether it was a living or nonliving object. This decision then had to be articulated in inner speech. However, rather than just examining the neural correlates of this inner speech compared to a control condition (fixating on a blank screen), Bullmore et al. (2000) attempted to link the activation they found in specific neural regions to the function that it was associated with. This led them to propose that activation in a series of connections from the prefrontal cortex to the left inferior frontal gyrus/Broca's area (BA 44, 45) via the SMA (BA 6) was responsible for subvocal planning and articulation. Inferior parietal lobule (BA 39, 40) activation extending to the precuneus (BA 7) and the superior temporal gyrus (BA 21) was presumed to represent the monitoring of the subvocal output of Broca's area.

The question of whether AVH-hearers have different patterns of brain activation during silent articulation as compared to healthy controls was first broached by McGuire et al. (1995). The psychiatric sample in this study consisted of patients diagnosed with schizophrenia who had consistently experienced AVHs during their illness, but who were in remission and had low levels of psychotic symptoms at the time of testing. PET scans were performed on both healthy participants and these remitting AVH-hearers as they silently read single words presented on a visual display unit. Analysis of the results found no differences in the areas activated by AVH-hearers and controls during silent articulation. This result was replicated by Shergill, Bullmore, Simmons, Murray, and McGuire (2000), using the same paradigm as Shergill et al. (2001) described above. In one part of Shergill, Bullmore, et al.'s (2000) study, the presented words were used to complete sentences such as 'I like x', or 'I like being y', which were silently articulated by the participants. fMRI imaging was used to compare the neural correlates of this silent articulation between healthy controls and AVH-hearers. It was found that patients showed no differences to controls in neurological activation when performing this task.

Such findings suggest that silent recitation of sentences is not abnormal in AVH-hearers in remission. Because silent articulation is believed to result in low levels of activation of the VSM system (McGuire, Silbersweig, Murray, et al., 1996), this suggests that tasks involving low levels of VSM are not performed abnormally by AVH-hearers. However, Shergill et al. (2003) hypothesized that, if the VSM load involved in silent articulation could be increased, then differences would be found between AVH-hearers and healthy controls. Accordingly, Shergill et al. (2003) asked participants to covertly articulate the word 'rest' either once every 4 s (low-VSM condition) or once every second (high-VSM condition). It was found that lateral temporal cortex activation (signifying higher levels of VSM) increased with the faster rate of covert generation in healthy participants. In contrast, in patients with a history of AVHs (but, at the time of study, in clinical remission) less activation was shown in the right (but not left) superior temporal gyrus, the right parahippocampal gyrus, and the right cerebellar cortex when compared to controls performing the faster rate of covert generation.

In conclusion, evidence suggests that neural correlates of inner speech during silent recitation of sentences are not abnormal in AVH-hearers in remission. Because silent articulation is thought to involve low levels of activation of the VSM system (McGuire, Silbersweig, Murray, et al., 1996), it seems likely that any neural differences between AVH-hearers and healthy controls will remain obscured unless methods other than low-VSM tasks are employed.

1.2. Neuroimaging of first-, second-, and third-person inner speech: auditory verbal imagery

In addition to studies of silent articulation of sentences, a number of studies have investigated the neural correlates of inner speech in the form of imagined speech, termed auditory verbal imagery (AVI). Such a study was performed by Shergill et al. (2001) with healthy participants, using the paradigm of completing sentences such as 'I like x', or 'I like being y', as outlined above. However, in addition to silently articulating such sentences, participants were also asked to imagine either the sentence being spoken in their own voice (first-person AVI), or sentences of the form 'You like x', being spoken to them (second-person AVI), or sentences of the form 'He likes x', as if spoken about them (third-person AVI). When imagining the sentences being spoken to them or about them, participants were instructed to imagine the
voice as being the same voice that spoke the words that were played to them. Shergill et al.'s (2001) fMRI analysis found that auditory verbal imagery in general, as indexed by the three imagery tasks combined, was associated with activation typically found in 'inner speech' tasks (left inferior frontal and parietal cortex and SMA). In addition, activation was seen in the left precentral and superior temporal gyri, and the right homologues of all these areas. The involvement of the right hemisphere homologues was presumed to occur due to retrieving and processing the prosody of the imagined other's speech.

McGuire et al. (1995) were the first to address the question whether, when producing auditory verbal imagery, patients with schizophrenia who have experienced AVHs have a different pattern of activation to healthy controls. Their study required participants to listen to recorded single words and then to imagine sentences of the form 'You are x', or 'You are a y', being spoken to them by the voice on the recording (this study only addressed second-person AVI). Using the results of their PET scans, it was found that AVH-hearers in remission showed reduced activation in the left middle temporal gyrus (MTG), the rostral SMA, and the left medial prefrontal cortex compared to healthy controls. It was also found that AVH-hearers in remission, compared to patients with schizophrenia who did not experience AVHs, showed reduced activation in the left MTG, the rostral SMA, and a posterior region spanning primary and secondary visual areas and the adjacent cerebellar cortex.

A difference in neural activation during AVI between patients with schizophrenia who had experienced AVHs and healthy controls was also found by Shergill, Bullmore, et al. (2000). As reviewed above, participants were asked silently to articulate sentences of the form 'I like x', or 'I like being y', and additionally to generate first-, second-, and third-person AVI. Functional MRI analysis of the AVI conditions, using an activation map for all three types of AVI combined, showed differences between patients with schizophrenia who had heard voices (but who were in remission) and healthy controls. Specifically, when AVH-hearers in remission imagined others speaking to them, there was less activation in the posterior cerebellum, hippocampal complex, and lenticular nuclei bilaterally, and also the right thalamus, middle and superior temporal gyri, and left nucleus accumbens.

In conclusion, evidence suggests that mechanisms underlying the ability to silently imagine a voice speaking are atypical in AVH-hearers in remission. Because imagining others speak is thought to be associated with high levels of activation of the VSM system (McGuire, Silbersweig, Murray, et al., 1996; Shergill et al., 2001), these conclusions are consistent with the findings of Shergill et al. (2003) that neural activation on tasks involving high levels of VSM (in this instance, producing inner speech at a high rate) may be abnormal in AVH-hearers in remission.

1.3. Event-related potential studies of silent articulation

Whereas scanning techniques such as PET and fMRI are able to provide indirect evidence that VSM may be atypical in individuals affected by AVHs, studies using event-related potentials (ERPs) are able more directly to test this hypothesis. As noted earlier, the VSM system is thought to work through a corollary discharge system whereby discharges from the motor speech-producing areas in the frontal lobes 'warn' the auditory cortex that self-produced speech is about to be produced (Creutzfeldt et al., 1989). This is postulated to work through the motor speech-producing areas' corollary discharge deactivating the auditory cortex when self-produced speech is about to occur (Ford & Mathalon, 2005).

Ford et al. have used ERPs to study the activation of such areas during inner speech in patients diagnosed with schizophrenia (Ford et al., 2001). In this experiment, N1 ERPs were used as a measure of engagement of the auditory cortex during inner speech. N1 is generated by the superior temporal gyrus of the auditory cortex in response to auditory stimuli of all kinds (ibid). Participants were asked to repeat typical hallucinatory statements (e.g., 'That was really stupid') silently to themselves for 30 s, while brief auditory stimuli were presented via headphones. The instruction to participants to use silent articulation in this study would presumably have entailed a low load on the VSM system (as opposed to the high load that would have resulted from instructions to use AVI). It was found that, in healthy participants, the N1 response to brief auditory sounds during 'inner speech' (contrasted here as repeating silently the hallucinatory type statements) decreased significantly as compared to a baseline condition involving silently fixating on a point. In contrast, patients with schizophrenia showed no significant difference in N1 response between these two conditions. Ford et al. interpreted this to show that, whereas healthy individuals dampen their auditory cortex response during self-produced speech, as if to warn the auditory cortex that the sound is internally produced, patients with schizophrenia do not. As a consequence this may lead patients with schizophrenia to experience their own self-produced speech as having an external source. However, Ford et al. failed to find a significant correlation between levels of hallucinations (as per SAPS summary score for hallucinations) and the N1 effect (baseline less 'inner speech').
suggesting that other factors must be causally involved in AVHs beyond damping of the auditory cortex response during the silent recitation of sentences.

No electrophysiological studies have been performed to examine directly whether corollary discharge is abnormal in AVH-hearers during high-VSM inner speech tasks. One study that used overt speech rather than inner speech attempted to examine whether corollary discharge was abnormal in AVH-hearing patients with schizophrenia (Ford & Mathalon, 2005, Section 6). These researchers assessed the involvement of frontal lobe speech production areas with speech reception areas in the temporal lobe, using measures of EEG coherence (frontal–temporal gamma synchrony). In the Talking condition participants uttered aloud the syllable [a] every 1–2 s. They then heard playback of their speech through earphones at various degrees of distortion (no distortion, half a semitone lower, and a full semitone lower). In the Listening condition, they did not speak but simply heard undistorted pre-recorded sounds from the Talking condition. The difference in coherence between the Talking and Listening conditions (a measure of the interdependence of the frontal and temporal lobes during talking), although different in participants with schizophrenia when compared to normal controls, did not correlate with hallucination severity (Ford & Mathalon, 2005, Section 8). Furthermore, the difference in coherence between the distorted and undistorted Talking conditions also did not correlate with hallucination severity. However, this study used overt speech rather than inner speech, and there is persuasive neuroimaging evidence that different neural regions (aside from motor areas) are activated in covert and covert speech (Huang et al., 2001; Soltysik & Hyde, 2006). As Ford and Mathalon (2005) note, "talking may be a poor proxy for thoughts and voices" (p. 188).

From this series of experiments Ford and Mathalon (2005) concluded that "we have not been able to demonstrate a clear relationship between neurobiological indicators of dysfunctional corollary discharge and the extent to which a patient currently reports experiencing auditory verbal hallucinations" (p. 187). Thus, ERP studies provide good evidence that low-VSM inner speech tasks do not appear to result in differential neural activation specific to the experience of AVHs. It still remains for ERP studies to examine neurophysiological differences between AVH-hearers and controls when performing high-VSM inner speech tasks such as AVI. We would predict that, on the basis of the neuroimaging findings reviewed above, hallucination severity would correlate with the N1 effect when the participants are imagining others speak.

1.4. Summing up

The neuroimaging and neurophysiological findings reviewed above provide evidence that the neural correlates of inner speech, conceptualized as the silent articulation of sentences, are not fundamentally different in AVH-hearers in remission compared to healthy controls. The neuroimaging studies also show that, when one’s own self or others are imagined talking in inner speech, there is a neural difference between AVH-hearers in remission and healthy controls without AVHs. As noted above, the silent articulation of sentences places a low load on the VSM system, whereas repeating single words at an increased pace or imagining a sentence being spoken place a high load on the VSM system. This leads to the conclusion that predisposition to AVHs is associated with abnormal neurological activation during tasks that involve high-VSM loads.

An important caveat to make at this point is that the neuroimaging evidence showing abnormal neurological activation in remitting AVH-hearers during high-VSM inner speech tasks does not necessarily translate into different patterns of judgment about the authorship of externally heard voices. Using a well-established paradigm (Johns, Gregg, Vythelingum, & McGuire, 2003), Johns, Gregg, Allen, and McGuire (2006) examined such judgments in patients with schizophrenia with and without current AVHs. Participants spoke into a microphone and then had the sound played back to them over headphones in real time. The voice they heard played back to them could be their own voice, their own distorted voice, another person’s voice (saying the same word), or another person’s distorted voice. The study found that patients who had previously experienced AVHs, but who were currently hallucination-free, did not misattribute the source of the spoken voice any more than healthy controls. However, although the neurological activation on high-VSM tasks differs between such patients compared to healthy controls, this does not appear to result in a detectable difference in tasks that require judgments about the authorship of heard voices. In contrast, Johns et al. (2006) found that patients currently experiencing regular AVHs were more likely (compared both to controls and to remitting AVH-hearers) to misattribute the source of the heard voices.

There are at least two possible ways of accounting for this apparent discrepancy between the neural correlates findings and the work of Johns et al. (2006) on judgments of voice authorship. First, it may be that both remitting and current AVH-
Baddeley and Hitch's model, the articulatory loop is responsible for the short-term maintenance of up to two seconds' recitation of predefined sentences. There has been a certain degree of consistency in the equation of inner speech with et al. defined inner speech as the "subjective phenomenon of talking to oneself, of developing an auditory-articulatory theoretical work on inner speech and its developmental precursors.

AVH-hearers, but that this difference only obtains on tasks involving high-VSM demands, such as auditory verbal imagery (AVI). In contrast, paradigms involving low-VSM demands, such as silent articulation, do not distinguish (neurologically, at least) AVH-hearers from healthy controls. And yet it is precisely these paradigms that are commonly held to be the best experimental analogues of normal inner speech (e.g., McGuire et al., 1995). This leads us to a conundrum: if we wish to account for the genesis of AVHs in terms of neurally instantiated VSM deficits leading to the impaired self-monitoring of inner speech, this cannot involve the kinds of verbal mentation (low-VSM tasks such as silent articulation) that are typically held to be the best experimental approximations to typical inner speech. Specifically, it needs to be explained why AVH-hearers might be performing verbal mentation in inner speech associated with high levels of VSM. Why, during typical inner speech, should they be performing tasks utilizing the same cognitive resources that are involved with imagining people speak? We suggest that this conundrum can be resolved through a careful examination of the nature, forms, and developmental significance of inner speech.

2. What is this thing called inner speech?

We propose that at least some of the apparent contradictions that have emerged from empirical research on inner speech stem from two related sets of unexamined assumptions: First, about what inner speech is, and second, about how it can be elicited artificially. In this section, we examine these assumptions in light of contemporary empirical and theoretical work on inner speech and its developmental precursors.

Many neuroimaging studies of AVHs have drawn on a conception of inner speech which derives from Levine, Calvanio, and Popovics (1982). In a paper examining the possibility of language in the absence of inner speech, Levine et al. defined inner speech as the "subjective phenomenon of talking to oneself, of developing an auditory–articulatory image of speech without uttering a sound" (p. 391). Some studies explicitly refer to this definition (e.g. Evans, McGuire, & David, 2000), while others assume it implicitly by holding the silent articulation of sentences to represent inner speech (e.g. Shergill et al., 2001). Other studies have initially defined inner speech as "thinking in words" (McGuire et al., 1995, p. 596), while going on to operationalize inner speech for experimental purposes as the subvocal recitation of predefined sentences. There has been a certain degree of consistency in the equation of inner speech with subvocal recitation. By these definitions, patients and controls are said to be performing inner speech when they mentally recite sentences such as "You are stupid" (McGuire et al., 1995, p. 597).

Such a subvocal rehearsal/recitation conception of inner speech has a clear historical foundation in (Baddeley and Hitch’s, 1974) model of working memory (Baddeley, 1986). This model proposed the existence of an articulatory loop consisting of a phonological store, capable of holding speech-based information, and an articulatory control process. In Baddeley and Hitch's model, the articulatory loop is responsible for the short-term maintenance of up to two seconds’
worth of verbal information in working memory. It thus corresponds to the part of the phonological information-processing system that Baddeley (1986) described as the “voice in the head”, or inner speech. The large body of empirical support for Baddeley and Hitch’s working memory model (e.g., Baddeley, 2001) may be one reason why the subvocal rehearsal/recitation conception of inner speech has influenced experimental attempts to elicit inner speech in neuroimaging studies. A second, related reason is that the elicitation methodologies that derive from this conception are relatively straightforward and thus appropriate for the considerable practical constraints associated with neuroimaging studies.

Baddeley and Hitch’s conception of inner speech as resulting from activation of the articulatory loop is not, however, the only conception of inner speech to have proved valuable to cognitive scientists. In Vygotsky’s (1934/1987) theory, inner speech represents the endpoint of a developmental process in which external discourse gradually becomes internalized to form verbal thought. Generally speaking, Vygotsky’s hypotheses about the development of inner speech (and its semi-covert precursor, private speech) have been supported by empirical research (Berk, 1992; Winsler, 2004). Of particular interest for our purposes is the finding that private speech frequently reflects the dialogic structure of interpersonal verbal exchanges (e.g., Ramirez, 1992; Wertsch, 1980).

Vygotsky’s (1934/1987) ideas about inner speech form part of a broader theory of the social origins of higher mental processes. This theory is encapsulated in his well-known claim that every mental function appears twice in development: First on the interpsychological plane, as a function distributed between individuals, and second on the intrapsychological plane, as an internalized version of that previously external function (Vygotsky, 1931/1997). In the case of the developmental transition from external speech to inner speech, this internalization can be seen at work, for example, in a mother and child solving a jigsaw puzzle. Initially, the puzzle-solving process will involve an external dialogue between mother and child, with the mother typically asking the child questions about which piece should be placed where, the child answering, and so on. Later on this dialogic pattern of exchange is internalized as inner speech (Fernyhough, 2004). Mental dialogue is, therefore, an internal version of the interplay of perspectives that takes place between individuals on the external plane (Fernyhough, 1996).

For Vygotsky, internalization of previously external verbal activity was an essential component in children’s developing self-regulation of behavior and cognition. Accordingly, studies of children’s private speech have shown that it often contains utterances that have a self-directive function (Luria, 1961). The dialogic nature of private speech (and, by extension, inner speech) guarantees that children become able to take the role of the questioner, advisor, and director in the regulation of their activity. As Vygotsky (1930–1935/1978) put it, the use of verbal mediation means that humans are able to “control their behavior from the outside” (p. 40).

Central to the Vygotskian conception of inner speech is the assumption that, like its developmental precursor private speech, it will retain certain characteristics of the external discourse from which it is derived. Several authors (e.g., Wertsch, 1980) have noted that one implication of Vygotsky’s theory is that inner speech will have a dialogic structure. In arguing against a view of inner speech as a homogeneous phenomenon, one of us (Fernyhough, 2004; submitted for publication) has previously suggested that at least two distinct forms of dialogic inner speech might be predicted on the basis of Vygotsky’s theory. These are expanded inner speech, where the flow of verbal mentation retains the give-and-take quality of external dialogue, and condensed inner speech, where the linguistic accoutrements of dialogue become jettisoned and inner speech becomes a process of “thinking in pure meanings” (Vygotsky, 1934/1987).

Fernyhough’s (2004) four-stage model of the development of inner speech is represented diagrammatically in Fig. 1. At Level 1 (external dialogue), the child and caregiver engage in verbally mediated reciprocal exchanges such as might arise in the collaborative solving of a jigsaw puzzle. At Level 2 (private speech), children conduct these dialogues in overt self-directed speech. This linguistic activity gradually becomes internalized, leading to the creation of expanded dialogic inner speech (Level 3). Finally, the syntactic and semantic abbreviation processes described by Vygotsky effect its transformation into condensed dialogic inner speech (Level 4), corresponding to Vygotsky’s stage of “thinking in pure meanings”.

This account entails that thinking retains the dialogic, semiotically mediated qualities of interpersonal discourse even when, phenomenologically speaking, it does not share the give-and-take character of external dialogue. To put it another way, the conversation that we have with ourselves does not cease when it ceases to be conducted in explicit, syntactically expanded questions and answers. One of the present authors (Fernyhough, 1996, 2004, 2005, submitted for publication) has previously examined the implications of a dialogic view of the higher mental processes for our understanding of a range of different cognitive processes, including executive functioning and social understanding. As Carruthers (2002) notes, most members of the cognitive science community endorse a purely communicative conception of language, which sees language as an input–output system for central cognition, with thinking undertaken
in some other form of representation. For example, Fodor (1975) proposed that we think, not in natural language, but in what is commonly termed mentalese. At the other end of the spectrum exist views, such as those held by early behaviorists, which equate thought with inner speech, claiming thought to be merely speech without sound (e.g., Watson, 1920). Treading a middle path between these two poles, a Vygotskian account of inner speech fits with cognitive conceptions of language which see it as having a constitutive role in cognition that goes beyond the straightforward communication of information (e.g., Carruthers, 2002). Vygotsky (1934/1987) proposed that thought and speech could be visualized as two intersecting circles. The overlapping region of thought and speech represents what may be termed verbal thought or inner speech. Such a conception allows that, while thought may occur in the medium of inner speech, nonetheless "[t]here is a large range of thinking that has no direct relationship to verbal thinking" (Vygotsky, 1934/1987, p. 115). This position is consistent with evidence of the need for verbal thought in some forms of cognition (Hermer-Vazquez, Spelke, & Katsnelson, 1999), as well as the possibility of thought without language (Weiskrantz, 1988). In line with this conclusion, an elegant neuroimaging experiment performed by Dehaene, Spelke, Pinel, Stanescu, and Tsivkin (1999) has demonstrated the involvement of language in performing exact mathematical calculations in one's head, but not in the performance of approximate arithmetic. We thus adopt a Vygotskian position that inner speech (both expanded and condensed versions) forms a subset of human thought processes.

We argue that Fernyhough's (2004) extension of Vygotsky's conception of inner speech has at least two important implications for our understanding of AVHs. First, the distinction between condensed and expanded inner speech allows us to understand the dynamic relation between AVHs, where inner speech is experienced with its full complement of acoustic properties, and much of regular human mentation, where the acoustic properties of the voices in inner dialogue are less apparent. In expanded inner dialogue, which we suggest forms the raw material of AVHs, human thought incorporates a multiplicity of internalized voices. If such expanded dialogues do indeed form the raw material of AVHs, it is unsurprising that the existing thoughts and ideas of voice-hearers may come to be reflected in part or much of the content of the AVHs (Leudar & Thomas, 2000). In terms of the neural correlates evidence discussed above, the dialogic conception of inner speech consequently allows us to see how different forms of inner speech entail different levels of VSM demands. Specifically, conducting expanded inner dialogues will require high levels of VSM, while condensed inner speech (where the acoustic properties of inner speech are attenuated) does not make such demands on the VSM system.

A second implication of this conception of inner speech is that it predicts the characteristic pragmatic qualities of AVHs. Specifically, it allows us to understand how AVHs frequently take the form of commands. As inner speech is
developmentally linked with the control of action (Luria, 1961; Vygotsky, 1934/1987), we should not be surprised that AVHs frequently have a similar regulatory quality.

3. Neural correlates of inner dialogue: testing a Vygotskian model

We turn now to the question of how the paradigms used to elicit inner speech in neuroimaging and neurophysiological studies relate to this alternative, dialogic conception of inner speech. Recall that the majority of such studies have elicited inner speech by asking participants to repeat sentences (such as ‘I like swimming’) subvocally. At first glance the resulting subvocal speech would appear closer in form to Level 3 (expanded) inner speech than Level 4 (condensed) inner speech. Crucially, though, the dialogic quality of typical spontaneous inner speech is absent in such experimentally elicited utterances. If our Vygotskian characterization of inner speech is accurate, we would have to conclude that the methods used for eliciting subvocal speech in typical neuroimaging studies do not lead to the generation of anything resembling naturally occurring spontaneous inner speech.

Furthermore, Vygotsky’s ideas about the semiotic mediation of higher mental processes entail that verbal mentation should continue even when inner speech loses its explicit linguistic form. Specifically, Femyhough’s (2004) extension of Vygotsky’s ideas on this issue suggest that, with the exception of episodes when inner speech is temporarily expanded to form Level 3 (expanded) inner speech, verbal mentation should consist of ongoing Level 4 (condensed) inner speech. The implications of this for neuroimaging studies are, we suggest, profound. In a study such as that of Shergill et al. (2001), the neural correlates of inner speech are examined by comparing a baseline condition (subjects listening to single words presented at 3 s intervals) to a condition where they are silently articulating sentences. However, what such studies treat as a baseline condition is likely to involve ongoing condensed inner speech. The results of such studies would therefore appear to be contaminated by the persistence of a form of inner speech even into the baseline condition.

The dialogic conception of inner speech has value, however, beyond pointing up possible methodological limitations of existing neural correlates studies. One of the present authors (Femyhough, 2004) has put forward a theory of AVHs that draws on Vygotskian ideas about the developmental significance of inner speech. According to this hypothesis, AVHs result from the temporary re-expansion of Level 4 (condensed) inner speech into Level 3 (expanded) inner speech, particularly under conditions of stress and cognitive challenge. Such movement between forms of inner speech is held to be a typical feature of human mental life. In individuals who report AVHs, in contrast, the re-expansion of condensed inner speech interacts with pre-existing source monitoring and other cognitive biases to result in the expanded dialogue being experienced as involving an alien voice. Femyhough (2004) further proposed that such hallucinations in psychiatric patients may be shaped (in terms of their content and structure) by the experience of trauma.

Femyhough (2004) suggested that the re-expansion model of AVHs could be tested (and distinguished from other possible hypotheses drawn from a Vygotskian account of inner speech) in four main ways. First, the re-expansion model entails that AVH-hearers should experience normal Level 4 (condensed) inner speech. To date, this issue has not been investigated empirically. Second, the re-expansion model would predict that AVH-hearers should not experience normal Level 3 (expanded) inner speech, as such speech would typically be experienced as having an alien quality. Again, this prediction awaits empirical examination. Third, the model would predict that AVHs would be associated with stress and cognitive challenge, a finding that receives support from a range of sources (e.g., Cooklin, Sturgeon, & Leff, 1983). Finally, the model holds that previously unaffected individuals should occasionally experience AVHs under conditions of very great stress, a prediction that is supported by reports of auditory hallucinations following bereavement and extreme stress (Balan et al., 1996; Reese, 1971).

Much more empirical research needs to be done before the re-expansion model of AVHs can be properly evaluated. In the meantime, several resulting hypotheses about the neural correlates of inner speech and AVHs can be assessed in light of the neuroimaging and neurophysiological evidence reviewed above.

Hypothesis 1. Neural correlates of condensed inner speech in AVH-hearers will be identical to those in healthy controls.

Femyhough’s (2004) re-expansion model holds that AVH-hearers should experience normal condensed inner speech. The prediction can thus be made that the neural correlates of this speech should not differ from those observed in healthy controls. One way of making sense of this prediction is in terms of our earlier distinction between tasks
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involving low and high levels of VSM demands. The attenuation of the acoustic characteristics of speech in Level 4 (condensed) inner speech is, we suggest, likely to result in a reduced VSM load in inner speech of this kind. One would therefore expect to see Broca's area activation accompanied by only minor corresponding activation in the VSM system (i.e., bilaterally in the lateral temporal cortex) in both AVH-hearers and non-AVH-hearers. The testing of this hypothesis would require a task that elicits condensed inner speech, with no expanded (Level 3) component. Currently no ecologically valid procedures exist for examining condensed inner speech, although the procedure employed by Ford et al. (2001) might be construed as offering an approximation to such a condition. Recall that, in Ford et al.'s baseline condition, participants had to fixate on a screen, while auditory stimuli (speech sounds, noises, etc.) were played to them. As Ford et al. themselves note, "spontaneous inner speech" (p. 1914) could occur during this baseline condition. From a Vygotskian perspective, we would expect condensed (Level 4) inner speech to be occurring during this part of the task. Thus, we would predict no N1 activation differences between AVH-hearers and healthy controls during this condition. In line with our prediction, Ford et al. found that N1 responses during the baseline condition, although being slightly (but not significantly) lower in patients than in controls, were not related to the presence of AVHs.

Hypothesis 2. Neural correlates of inner speech in AVH-hearers will differ from those of controls in tasks involving the re-expansion of condensed inner speech.

The central proposal of the re-expansion model is that AVHs stem from the re-expansion of condensed inner speech. AVH-hearers are suggested to differ from their unaffected counterparts in their experience of Level 3, but not Level 4, inner speech. To date, no neural correlates studies have examined the transition between these forms of inner speech. The paradigm that would lend itself most easily to being adapted to such an investigation is that of Bullmore et al. (2000). In this study, participants viewed a word on a screen, then had to articulate internally a semantic judgment about the stimulus. We speculate that the semantic judgment process might involve Level 4 (condensed) inner speech, with a transition to Level 3 (expanded) inner speech during the subvocal articulation of the judgment. In line with our first hypothesis, we would predict that the neural activation of AVH-hearers would not differ from controls up to the point when condensed inner speech stops and the conclusion becomes subvocally articulated. In contrast, the transition to Level 3 inner speech during the subvocal articulation of the semantic judgment should lead to observable differences between AVH-hearers and controls. In addition to typical patterns of activation associated with high-VSM tasks (corresponding here to the occurrence of Level 3 inner speech), one might also expect AVH-hearers to show greater levels of parietal cortex activation, representing the experience of alienness (Spence et al., 1997). Time-sequenced fMRI studies might be best placed to detect any such group differences in neural correlates of inner speech in the transition between Level 3 and Level 4 inner speech.

Hypothesis 3. Neural correlates of elicited expanded inner speech should differ between AVH-hearers and controls.

As noted above, the re-expansion model entails that AVH-hearers should not experience typical Level 3 inner speech. To date, this prediction has been tested neither by examination of subjective reports of AVH-hearers' inner speech, nor by the attempted experimental elicitation of expanded inner speech. We have noted that no existing neural correlates studies have included explicit instructions to generate expanded dialogic inner speech. The closest approximations in the existing literature would appear to be the studies of McGuire et al. (1995) and Shergill, Bullmore, et al. (2000), where second-person AVI has indeed been shown to be associated with neural differences between AVH-hearers and controls. In contrast, no such group differences are found in the neural correlates of silent (non-dialogic) articulation. Our hypothesis here could be tested simply by asking participants to conduct an expanded dialogue in their heads while undergoing imaging. We would predict that such a condition would lead to similar differences in neural activation to those predicted by Hypothesis 2. We would also predict, in line with the re-expansion model, that the experience of expanded inner dialogues, even when experimentally elicited, should, subjectively, have some of the hallucinatory quality of the AVHs held by the re-expansion model to result from the re-expansion of condensed inner speech.

Hypothesis 4. Neural correlates of the experience of an AVH should be similar to patterns of activation in healthy individuals undertaking expanded inner speech.

We noted above (Hypothesis 3) that approximations to the elicitation of expanded inner speech exist in studies that have required participants to undertake AVI. A pertinent observation in this respect is one made by Shergill, Brammer, et al. (2000). Reporting their use of fMRI scanning of patients with schizophrenia while they were experiencing AVHs,
Shergill, Brammer, et al. commented that “The pattern of activation we observed during auditory hallucinations is remarkably similar to that seen when healthy volunteers imagine another person talking to them (auditory verbal imagery)” (p. 1036). Specifically, they observed common activation of the bilateral frontal and temporal gyri, along with right-sided precentral and inferior parietal gyri. One important difference between patients hearing AVHs and healthy participants generating AVI, however, is that the latter is associated with increased SMA activation, while this region is only weakly activated during AVHs. We can therefore conclude that Hypothesis 4 receives some support from studies that involve approximations to expanded inner speech (through the elicitation of AVI in healthy controls), but that the involvement of the SMA may differ between AVH-hearing patients and healthy controls generating AVI.

Finally, we suggest that the re-expansion hypothesis may be able to resolve the controversy about the involvement of Level 3 (expanded) inner speech in both AVH-hearers and healthy controls. While a number of studies have found increased left inferior frontal cortex activation compared to baseline in AVH-hearers experiencing hallucinations (Shergill et al., 2004; Shergill, Brammer, et al., 2000), others have failed to find such an increase in activation during AVHs (Lennox et al., 2000; Silbersweig et al., 1995). These negative findings on the involvement of Broca’s area activation in AVHs have been interpreted as providing “no support to the theory that auditory hallucinations arise from abnormalities of inner speech” (Lennox et al., 2000, p. 19).

We would argue that these contradictory findings may be accounted for by the persistence of Level 4 (condensed) inner speech into baseline conditions in typical neuroimaging studies. Given that the re-expansion model predicts that AVH-hearers will experience normal Level 4 (condensed) inner speech, this result would support continued activation of Broca’s area even when patients are being scanned at rest, without any current experience of hallucinations. Broca’s area activation will also be a feature of the experience of hallucinations, held by the re-expansion model to involve Level 3 (expanded) inner speech. Thus, the difference in Broca’s area activation between the AVH and non-AVH states will depend on how much condensed inner speech patients have been performing during their non-hallucinating rest period in the scanner. If patients are undertaking continuous condensed inner speech at rest, this would be likely to lead to no difference in Broca’s area activation between AVH and non-AVH phases. In contrast, if low levels of condensed inner speech are being performed at rest, then an increase in Broca’s area activation is likely to accompany AVHs. In order to resolve this question, we recommend that future neuroimaging studies incorporate post-scanning self-report measures of the thinking and inner speech that occurred during participants’ time in the scanner. If such subjective reports are taken into account, it should be possible to determine the relative contributions of condensed and expanded inner speech to Broca’s area activation in both hallucination and rest phases of such imaging studies.

4. Conclusions

At the start of this paper we set out certain key features of AVHs that we believe a comprehensive account of the phenomenon must explain. We reviewed the neuroimaging and neurophysiological evidence that has to date been used to study neural correlates of inner speech in AVH-hearers and healthy controls. We proposed that an apparent conundrum posed by this research, namely that differences between these groups only obtain on tasks involving high levels of VSM, can be resolved by considering the different possible forms that inner speech can take. Fernyhough’s (2004) re-expansion model of AVHs was used to generate testable hypotheses for future studies of the neural correlates of inner speech in both AVH-hearers and healthy controls.

Before drawing any final conclusions about the value of the theoretical ideas considered here, we return to our earlier caveat about the assumed, but not proven, involvement of inner speech in AVHs. It has previously been argued that research showing that neither the articulatory loop (David & Lucas, 1993; Haddock, Slade, Prasae, & Bentall, 1996) nor the inner-voice/inner-ear partnership is impaired in patients with schizophrenia means that, if any relationship exists between AVHs and inner speech, it is not simplistic or direct (Evans et al., 2000). While this conclusion appears sensible, we note that it assumes that working memory conceptions of inner speech (as silent articulation/rehearsal) map precisely onto those derived from Vygotsky’s theory. Rather, we have argued that spontaneous inner speech has varieties of forms, as well as syntactic and semantic properties, that make it a richer phenomenon, both in its subjective qualities and its neural instantiation, than the subvocal articulation of external utterances. Lack of any strong evidence for articulatory loop deficits in schizophrenia cannot therefore be taken to invalidate the re-expansion model, as both condensed and expanded inner speech will be reliant on a functioning phonological apparatus.

We now return to the four key challenges for any comprehensive account of AVHs. The first task is to explain why AVH-hearers experience a voice in the absence of any external stimulus. Our answer to this question, in line with those
of many other authors (e.g., Bentall, 2003; Bick & Kinsboume, 1987), is that the raw material of AVHs is internally generated inner speech. We go beyond these existing accounts, however, in claiming that inner speech is inherently dialogic. Two specific forms of dialogic inner speech are identified: Level 3 (expanded) inner speech, requiring high levels of VSM, and Level 4 (condensed) inner speech, associated with low-VSM demands. Specifically, it is Level 3 (expanded) inner speech that forms the raw material for AVHs. Such an account can account for the paradoxical 'alien-yet-self' quality of AVHs (Fernyhough, 2004), as well as fitting with the evidence from neural correlate studies that AVH-hearers differ from healthy controls only on tasks involving high levels of VSM.

The second challenge for any comprehensive account of AVHs is to explain why they are experienced as authored by a person other than one's self. Following Frith and Done (1988) and others, we attribute this feature of AVHs in part to a failure in a neurocognitive action self-monitoring system (Jones & Fernyhough, in press), which is put under particular pressure in high-VSM conditions, such as the generation of expanded inner speech. Beyond explaining errors of judgment about authorship, however, a comprehensive account of AVHs must meet a third challenge, namely to explain why voices are often perceived as having person-specific acoustic properties, distinct from those of the AVH-hearer. The Vygotskian approach to inner speech allows us to account for this third feature of AVHs with reference to the dialogic nature of inner speech. That is, human mentation involves the dialogic interplay of semiotically manifested perspectives on reality, such that our thinking is literally shot through with other voices. When combined with atypical VSM, of the kind that we and others propose to characterize AVH-hearers, in addition to further potential factors such as anterior cingulate and parietal cortex activation, these voices are perceived as having an alien quality.

The fourth challenge for a comprehensive theory is to explain why AVHs have characteristic contents and pragmatics, particularly their tendency to appear as second-person assertive utterances (e.g., commands). We have suggested that the developmental foundation of inner speech in private speech and external dialogue, where children gradually acquire linguistic means for regulating their own behavior, can account for the specific pragmatic qualities of AVHs.

We have set out a number of suggestions for how future studies involving AVH-hearers and healthy controls might test these hypotheses. We propose that further light may be shed on these issues through the use of a neglected population in AVH research. Many voice-hearers do not attract a clinical diagnosis such as schizophrenia and are able to deal with the experience of hearing voices in living normal lives (Romme & Escher, 1993). To our knowledge this population has not yet been invited to participate in neuroimaging studies. Our hypotheses would predict, for example, that neural correlates of expanded inner speech in healthy voice-hearers would be similar to those of AVH-hearers diagnosed with schizophrenia. In terms of existing neuroimaging paradigms, we would predict less SMA and temporal lobe activation than healthy controls when generating (particularly second-person) AVI. In addition, we note the need for much more research on the neural correlates of the experience of AVHs, to complement the rich body of research conducted with AVH-hearers in remission. There is also an urgent need to devise ecologically valid procedures for eliciting both forms of dialogic inner speech. Inner speech is, we suggest, a rich and heterogeneous phenomenon, and empirical studies of it are weakened by a continued reliance on unexamined assumptions about its form and nature.

References


Appendix B
Appendix B


Appendix C

Scale for assessment of passivity experiences in the general population (SAPE-GP)

1. Do you ever feel as if you are under the control of some force or power other than yourself?

2. Do you ever feel as if you are possessed by someone or something else?

3. When making movements (such as reaching for something) do you ever feel as if someone else inside you caused you to move?

4. Do you ever feel as if you are a robot or zombie without a will of your own?

5. Do you ever feel as if your feelings or actions are not under your control?
Appendix D

Participant Information Sheet

**Electrophysiological investigations of inner speech**

I am a PhD student at Durham University where I am trying to find out more about what happens in the brain when we perform inner speech in our head. You have received this Information Sheet as part of an invitation to take part in a study. Before you decide whether to take part, it is important for you to understand why the research is being done and what it will involve. If there is anything that is not clear, or that you would like to discuss further, please let me know. Take as much time as you like to decide whether you would like to take part. Thank you.

*Why have I been chosen?*

Participants are being recruited randomly from the student population.

*Do I have to take part?*

It is up to you to decide whether or not to take part. If you do decide to take part you will be asked to sign a Consent Form before the start of the experiment. All your data will be kept strictly confidential. If you decide to take part you are still free to withdraw at any time without having to give your reasons, and your data will not be used.

*What will happen to me if I take part?*

The following information sets out what will happen if you agree to take part in the study. Please read it carefully and ask me if you need any more information. The experiment consists of one approximately 1 hour session which will take part in the Durham Psychology Department. Firstly, we need to give you a special cap to wear. Like the picture below. This cap contains equipment that can sense what electrical...
activity is happening in your brain. It does not send any signals into your brain but just measures the electrical activity on its way out, so your brain won't be interfered with in any way. This is a completely safe and non-invasive procedure. Once you've put the cap on we will need to squirt some gel into it through some holes in the top. This gel just contains water and salt and its purpose is to increase the contact to your scalp. Although the gel will wash out of your hair easily, it may look a little messy afterwards. Given this potential messiness, testing can be done at a time that suits you, e.g. after lectures in the afternoon before you head back to halls / home. Once the cap is on, you will be asked to perform a number of simple tasks, whilst a range of sounds are played to you via headphones. These will involve repeating a number of sentences to yourself, silently in your head, and imagining objects rotating. The total time for the experiment will be 1 hour, and you will be paid £7 for participating.

What are the possible advantages and disadvantages of taking part?

There will be no direct benefits of taking part, except for the chance to advance our knowledge of the neural activity associated with inner speech. There are no risks or side-effects of participating and the study has been approved by the Psychology Department Ethics Committee.
Appendix D

If you think you would like to take part in this experiment, please drop me an e-mail (s.r.jones@durham.ac.uk) and we can arrange a time convenient for you to come into the Department to participate in the experiment.

Many thanks

Si Jones (e-mail at s.r.jones@durham.ac.uk)
Appendix E

Visual stimuli utilised in mental rotation task

A

B

C