The Nature of Schizotypal Symptoms and Social Recovery in Psychosis

Joanne Michelle Hodgekins

Thesis submitted for the degree of Doctor of Philosophy

University of East Anglia

School of Medicine, Health Policy and Practice

March 2009

ABSTRACT

Schizotypy is traditionally conceptualised as a personality trait reflecting vulnerability to the development of psychosis. This thesis introduces the concept of schizotypal symptoms as state phenomena, related to both the development of psychotic symptoms, and to long-term recovery from the disorder. It is argued that schizotypal symptoms may be at the core of psychosis, occurring both prior to onset and following the remission of an acute psychotic episode. Schizotypal symptoms may therefore provide a bridge for the symptom-disability gap which has long been established in psychosis.

The first study in this thesis reports on the psychometric properties of the Schizotypal Symptoms Inventory (SSI), a modified schizotypy assessment tool designed to measure current low-level positive psychotic symptoms in clinical and non-clinical populations. Levels of schizotypal symptoms are then compared in clinical and non-clinical samples. Following this, differential relationships between schizotypal symptom types and emotional, psychological, and neuropsychological variables are investigated. Schizotypal symptoms are then examined in relation to existing psychosis outcomes, before being integrated into a dimensional model of recovery. The final study of this thesis investigates the role of schizotypal symptoms as mediators of social recovery from psychosis, in the context of a randomised controlled trial.

The findings of this thesis suggest that social anxiety schizotypal symptoms are highly prevalent in individuals recovering from acute psychosis. Moreover, differential relationships exist between schizotypal symptom types and emotional, psychological, and neuropsychological variables. Anomalous experiences are associated with a visual processing perceptual anomaly, whereas social anxiety and paranoid schizotypal symptoms are associated with emotional processing. Differential relationships also exist between schizotypal symptom types and recovery dimensions. Social anxiety mediates social recovery from psychosis, whereas anomalous experiences are associated with positive symptoms. Paranoid schizotypal symptoms are associated with both symptomatic and social recovery. These findings are combined in a psychological model of recovery in the discussion chapter.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

ACKNOWLEDGEMENTS

I would firstly like to thank David Fowler, my supervisor, for his continual guidance, feedback, and encouragement; and for teaching me a great deal. Our discussions have been invaluable in shaping my understanding of psychosis and in my development as a researcher. I would also like to thank Siân Coker for her support throughout the PhD process and for providing feedback on drafts of this thesis.

I would like to express my gratitude to all of the people who agreed to participate in this research, without whom the studies described in this thesis would not have been possible. I am also extremely grateful to all of the teams and services who supported the research, particularly the Norfolk Early Intervention Service and the Assistant Psychologists who assisted with data collection and recruitment.

Special thanks go to colleagues at the Institute of Psychiatry and University College London (PRP group: Daniel Freeman, Philippa Garety, Elizabeth Kuipers, Paul Bebbington) and the University of Cambridge (ISREP study: Peter Jones, Carolyn Crane, Michelle Painter). In particular I would like to acknowledge Katarzyna Ray-Glover for her help with the creation of the website for the internet study; and Tim Croudace and Graham Dunn for their statistical wisdom. I feel very privileged to be involved with such a scholarly and inspiring research team.

Finally, I would like to thank my friends and family for their continued support and tolerance whilst I have been completing this thesis. I wish to give very special thanks to my parents for always believing in me and for their many years of unwavering personal encouragement and emotional backing.

CHAPTER ONE: INTRODUCTION

1.1 GENERAL OVERVIEW

There is evidence to suggest that low-level psychotic, or schizotypal, signs are common both in the general population and in the prodromal stages of psychosis (e.g. van Os, 2003). Thus, schizotypy is often considered as an index of an individual's underlying vulnerability to developing psychosis (Meehl, 1990). Although little research has been conducted into the area, schizotypal phenomena may also be important in the recovery phase of psychosis. Rather than a complete and immediate resolution of all symptoms following an acute psychotic episode, the frequency and intensity of experiences and beliefs gradually reduce (Drury, 1992). This often results in the presence of residual, low-level traces of symptoms which could be argued to resemble schizotypal-like experiences. These phenomena may not be highlighted by traditional assessment tools but are nonetheless important as could influence an individual's social and functional recovery from the effects of psychosis; and also leave them vulnerable to potential relapse.

It could be argued that given their similarities with schizotypal phenomena and in line with the psychosis continuum hypothesis, the prodromal and residual symptoms of psychosis may be most effectively assessed using a measure of schizotypy. However, current assessment tools in this domain perceive schizotypy as a personality trait as opposed to a subclinical manifestation of psychosis (i.e. a mental state). As such they are not reflective of fluctuations in schizotypal symptoms over time. Introducing a trait-state distinction to the schizotypy concept would allow it to be used to assess not only an individual's underlying vulnerability but also the presence of current low-level psychotic symptomatology.

This thesis examines and compares the prevalence and frequency of low-level psychotic phenomena in both clinical and non-clinical populations using a modified measure of schizotypal symptoms. It further aims to examine the potential underlying mechanisms associated with different types of such phenomena, with particular reference to the domain of social disability. This opening chapter addresses key issues concerning the context in which the research that follows is set. First, the major concepts of interest –

predominantly psychosis and schizotypy – are defined, and an overview of the current theories proposed for these concepts is provided. The importance of assessing schizotypal symptoms following an episode of psychosis is then discussed. Finally, the studies to be conducted in this thesis are outlined, along with their associated aims.

1.2 DEFINITION OF PSYCHOSIS

Psychosis is a broadly defined concept relating to a set of symptoms which exist across a range of diagnostic categories; including schizophrenia, schizo-affective disorder, and bipolar disorder (Sims, 2002). Psychotic symptoms can also occur outside of these diagnoses and, furthermore, secondary psychotic symptoms (i.e. those not occurring from psychiatric conditions) have been found to exist in a range of other disorders, including dementia and in individuals with brain tumours (Cummings, 1988). A psychotic episode is often described as involving a "loss of contact with reality" (Overall & Gorham, 1962) and this can be taken as a reflection of the level of disruption occurring to an individual's perceptual and thought processes. Characteristic symptoms of psychosis include hallucinations, delusional ideation, and disordered thoughts and speech. In addition, these symptoms are frequently accompanied by impaired social interaction, poor functioning, and a lack of insight (Cassano, Pini, Saettoni, Rucci, & Dell'Oso, 1998; Pini, Cassano, Dell'Oso, & Amador, 2001). When occurring in conjunction with disorders of mood (i.e. affective psychosis), the content of symptoms is also generally influenced by the nature of the mood (S. Jones & Bentall, 2006).

There are a number of different ways of conceptualising psychosis including the diagnostic approach, in which psychotic disorders are studied as single entities; and the single symptom approach, which argues that there is utility in investigating the individual symptoms of psychosis separately. In contrast to these symptom-focused approaches, and due to the varied nature of outcome from psychosis, it may also be useful to investigate different types of recovery from psychosis. These three different approaches will now be outlined and discussed.

1.2.1 Diagnostic Approach

The term *psychosis* is often used interchangeably with diagnostic terms for psychotic disorders, such as *schizophrenia*, *schizoaffective disorder* and *bipolar disorder* (Cutting, 1985). Traditional psychiatric assessment instruments are used to diagnose these disorders and are designed to assess the presence of clinically definable symptoms, outlined in psychiatric diagnostic criteria such as the Diagnostic Statistical Manual of Mental Disorders (DSM; American Psychiatric Association, 1994) and the International Classification of Diseases (ICD; World Health Organisation, 1990). Using these criteria, the occurrence of symptoms is viewed as a deviation from an individual's habitual mode of behaviour, i.e. their presence is "abnormal".

A range of diagnostic tools exist, including the Structured Clinical Interview for DSM (SCID; First, Spitzer, Gibbon, & Williams, 1996; Spitzer, Williams, & Gibbon, 1987); the Present State Examination (PSE; Wing, Cooper, & Sartorius, 1974); the Schedules for Clinical Assessment in Neuropsychiatry (SCAN; Wing et al., 1990); and the Brief Psychiatric Rating Scale (BPRS; Overall & Gorham, 1962). These general psychopathology measures assess a range of clinically defined disorders and have a wider remit than the assessment of psychotic symptoms. Conversely, the Diagnostic Interview for Psychosis Diagnostic Module (DIP-DM; Jablensky et al., 1999) is a semistructured interview specifically designed for the assessment of psychosis in epidemiological and clinical settings. Items are derived and adapted from the SCAN, and diagnoses are generated from the Operational Criteria Checklist for Psychotic Illness (OPCRIT; McGuffin, Farmer, & Harvey, 1991).

Measures such as those outlined above have been used to examine the epidemiology of psychotic disorders and also to investigate their underlying aetiology. These studies will now be outlined.

1.2.1.1 Epidemiology

Prevalence rates of psychosis vary depending on the diagnostic category under investigation (e.g. schizophrenia, schizoaffective disorder, bipolar disorder, etc). The

lifetime prevalence of schizophrenia and bipolar disorder is approximately 0.7-1% (Kendler, Gallagher, Abelson, & Kessler, 1996; Woods, 2000), although individual psychotic symptoms have been found to be present in as much as 10-15% of the general population (Tien, 1991). The incidence of schizophrenia and other psychotic disorders is highest in late adolescence to early adulthood and at this time point, it is males who are most affected (Riecher et al., 1989). However, the gender distribution of bipolar disorder is more equal (Walden & Grunze, 2004). The incidence of psychosis has been found to be associated with a lower socio-economic status; although it is arguable as to whether this is a cause or an effect of the disorder (Dohrewend et al., 1992). Psychosocial stress is thought to be a prominent trigger factor in psychosis, with numerous studies highlighting an association between trauma, significant life events and the onset of psychosis (Bebbington et al., 1993; Read & Ross, 2003; Ventura, Nuechterlein, Lukoff, & Hardesty, 1989). Social isolation, migration, and victimisation are also thought to be important risk factors (Bebbington et al., 2004; Cantor-Graae & Selten, 2005; Thornicroft, Bisoffi, de Salva, & Tansella, 1993).

1.2.1.2 Aetiology

The aetiology of psychosis is somewhat debatable, with no singular theory providing a definitive account of its occurrence. In the psychiatric literature, there is argument for a large organic component to psychosis, focusing particularly on the neurotransmitter dopamine (Davis, Kahn, Ko, & Davidson, 1991). Furthermore, neurodevelopmental and genetic hypotheses for psychosis have been suggested (Gottesman & Shields, 1982; Murray & Lewis, 1987), as well as theories relating to psychological processes (Garety, Kuipers, Fowler, Freeman, & Bebbington, 2001). These will now be outlined in more detail.

Dopamine Hypothesis. The dopamine hypothesis attempts to explain the biological processes accompanying the onset of psychosis. An over-activity of dopamine systems in the mesolimbic pathway has been argued to have an influence on the positive symptoms of psychosis. Conversely, problems with dopaminergic function in the mesocortical pathway have been thought to play a role in the occurrence of negative symptoms (Davis et al., 1991). Evidence for the dopamine hypothesis initially came from the finding that medications reducing excess levels of dopamine proved

successful in reducing psychotic symptoms (Carlsson & Lindquist, 1963). Complementary support was provided by the occurrence of amphetamine-induced psychosis; a psychotic episode experienced under the influence of amphetamines, which act by increasing dopamine levels in the brain (J. A. Lieberman, Kane, & Alvir, 1987). Furthermore, neuroimaging studies have shown that when psychotic, patients with schizophrenia show a heightened synthesis of dopamine (Reith et al., 1994).

Kapur (2003) further developed the dopamine hypothesis in an attempt to clarify the role of the neurotransmitter in psychosis. Mesolimbic dopamine is argued to be involved in attributing salience or significance to affectively neutral stimuli, transforming them to be either aversive or attractive (Berridge & Robinson, 1998). Kapur (2003) suggests that in psychosis there is an increased, often stimulus-independent release of dopamine, resulting in the aberrant attribution of salience to relatively innocuous events and stimuli. It is postulated that this induces a "somewhat novel and perplexing state marked by exaggerated importance of certain percepts and ideas" (p. 15), leaving individuals confused and searching for an explanation. This is argued to form the basis for the development of symptoms such as hallucinations and delusions.

Neurodevelopmental Hypothesis. The neurodevelopmental theory of psychosis suggests that aberrant brain development; occurring due to the influence of faulty genes and/or early neurological insults; leads to a predisposition to the later development and onset of the disorder (Murray & Lewis, 1987). The nature of this aberrant brain development has been proposed to stem from excessive cortical pruning during postnatal development, resulting in a loss of neural elements (Keshavan, Anderson, & Pettegrew, 1994). Evidence for this approach comes from studies which have demonstrated a higher level of developmental delays in children who later go on to develop psychosis (Cannon et al., 2002). Further evidence of a neurological basis to psychosis comes from studies demonstrating anomalies of brain structure and function in individuals suffering from psychotic disorder (e.g. Chua & McKenna, 1995). More recent elaborations of the neurodevelopmental hypothesis postulate the incorporation of social factors, such as social isolation and urban upbringing, into the model (e.g. Boydell, van Os, McKenzie, & Murray, 2004). This suggests the possibility of an

exacerbation of aberrant neurodevelopment due to the interaction of environmental influences with biological factors.

Genetic Theories. Genetic theories of psychosis propose an underlying genetic component to the disorder, perhaps resulting in a vulnerability to the biological and neurological disturbances outlined above. Evidence for this comes from twin studies which suggest that a high proportion of the variance in liability to psychosis is genetic (Gottesman & Shields, 1982). Individual genes, including neuregulin and dysbindin, have recently been highlighted as playing an explicit role in increasing the risk of schizophrenia (P. J. Harrison & Owen, 2003). The exact function of these genes is unclear but it is suggested that they may have an impact upon dopamine regulation. It must be remembered however that genetic predisposition is unlikely to be sufficient for the expression of psychosis and that an interaction with environmental factors is most probably also required (van Os & Sham, 2003).

Psychological models of psychosis. These models consider the role that psychological processes may play in the formation and maintenance of psychotic symptoms, and how these can be modified in a therapeutic context. The cognitive model of psychosis proposed by Garety, Kuipers, Fowler, Freeman, and Bebbington (2001) suggests that a "basic cognitive dysfunction" results in the occurrence of anomalous experiences, which are then interpreted and appraised in the context of an individual's life experiences and affective state. Cognitive biases are also argued to influence the interpretation of such anomalous experiences. However, rather than one inclusive psychological model of psychosis, differential hypotheses exist for the role of psychological processes in the development and maintenance of different psychotic symptom types. As such, psychological models will be discussed in more detail in a later section of this chapter.

Summary. Research has highlighted an underlying biological or organic basis to psychosis which, via an influence on early development, creates an individual level of vulnerability to the experience of psychotic symptoms in later life; usually in adolescence or early adulthood. This vulnerability may be triggered by stress, emotional factors, and/or the occurrence of major life events. In addition to this, psychological processes play a role in the interpretation of the product of basic biological disturbances,

and thus the formation of more specific symptoms. The aetiology of psychosis appears to be multi-factorial, with each factor having a variable level of impact amongst different individuals, depending on their vulnerability threshold.

1.2.1.3 Treatments for psychosis

Pharmacological treatment. Treatment for psychosis can take a variety of forms but the dominant approach often involves the use of antipsychotic medication to respond to the excess levels of dopamine thought to be responsible for psychotic symptoms (Hirsch & Weinberger, 2003). Pharmacological treatments for psychosis were first developed in the 1950s, with the discovery of "typical" antipsychotic medications such as chlorpromazine (Cole, Klerman, & Goldberg, 1969). As opposed to targeting specific areas of the brain, the action of these medications is somewhat generalised. As such, their benefits are accompanied by a range of side effects that can cause equal amounts of distress as the psychosis itself. To some extent these problems have been overcome by the introduction of "atypical" and more specific antipsychotic medications, which only target the parts of the brain thought to be important in the occurrence of psychotic illnesses, such as the limbic system (Moghaddam & Bunney, 1990). These medications include clozapine, olanzapine and risperidone and large scale randomised controlled trials have suggested that as well as producing less side-effects, they are also more effective than typical antipsychotic drugs in reducing psychotic symptoms (Kane, Honigfield, Singer, & Meltzer, 1988).

Although there are distinct benefits to the use of medication in psychosis, drugs are not a cure for the disorder, but instead alleviate the associated symptoms (Gitlin et al., 2001). Due to unpleasant side effects, such as involuntary movements, weight gain, drowsiness, physical health problems and sexual dysfunction; compliance with medication is often problematic (Coldham, Addington, & Addington, 2002; Kampman et al., 2002). Furthermore, some individuals are found to be unresponsive to medication altogether (Garety, Fowler, & Kuipers, 2000; Whitaker, 2004).

Psychological treatment. Psychological treatments have recently been utilised in addition to drugs in an attempt to successfully target the symptoms and impact of psychosis. Although medication may reduce the intensity and frequency of

experiences such as voices by reducing dopamine release; it cannot alter the interpretation or psychological impact of these symptoms (Kapur, 2003). Similarly, whilst psychological therapies may not directly affect dopaminergic activity; they can change the way an individual views themselves and the world, and therefore how they interpret their psychotic experiences. Psychosocial interventions can also reduce the distress associated with psychotic experiences; promote awareness of early warning signs; and provide coping strategies to be used when these signs occur (Rector & Beck, 2001). Thus, it is increasingly recognised that psychological treatments can reduce the probability of psychotic relapse (Kuipers et al., 1997). Cognitive-behaviour therapy (CBT) is perhaps the most well-documented psychological approach in psychosis although other interventions include family therapy and social, cognitive and occupational rehabilitation approaches (Pilling, Bebbington, Kuipers, Garety, Geddes, Martindale et al., 2002; Pilling, Bebbington, Kuipers, Garety, Geddes, Orbach et al., 2002). Many of these therapies are evidence-based, utilising research highlighting the importance of particular psychological processes in psychosis (Brenner & Pfammatter, 2000).

1.2.1.4 Problems with the diagnostic approach

Psychiatric diagnoses provide a way of labelling certain types of behaviour and experiences and are also useful in terms of deciding the appropriate course of treatment for a given disorder. However, despite highlighting the presence of psychotic disorder, the diagnostic approach does not provide an explanation of the causes of psychotic symptoms (Bentall, 2004). In addition it does not reflect the variability present within psychotic disorders. Indeed, two individuals may have the same diagnosis but yet have both completely different presentations and outcomes (Bentall, Jackson, & Pilgrim, 1988). Moreover, studies have suggested that clinicians often disagree about diagnostic classification, and that diagnostic practices can differ cross-culturally (Brockington, 1992; van Os, Gilvarry et al., 1999). These flaws have led researchers to suggest that a "single symptom" approach may provide a better way of investigating the underlying mechanisms responsible for the development and maintenance of psychotic phenomena (Persons, 1986). This approach will be discussed in the next section.

1.2.2 Single Symptom Approach

The single symptom approach to psychosis proposes the importance of studying single symptoms of psychosis in their own right (e.g. Freeman, Garety, Kuipers, Fowler, & Bebbington, 2002; Oltmanns & Maher, 1988; Slade & Bentall, 1988). This method of study combats the flaws of the diagnostic approach and also enables the isolation of single elements of pathology for study (Persons, 1986). Using this approach, individuals with psychosis can be classified in terms of their current problems, and treated accordingly, rather than being considered as a homogenous group (Allardyce, Suppes, & van Os, 2007).

1.2.2.1 Types of psychotic symptoms

Despite the idiosyncratic nature of psychotic symptoms, common themes have been highlighted. This has enabled the broad categorisation of psychotic phenomena into three different types: "positive symptoms", "negative symptoms", and "disorganised symptoms". This idea was first proposed by Strauss, Carpenter, and Bartko (1974) who built upon Crow's (1980) positive and negative dimensions of psychosis, and has since been supported by numerous factor analytic studies (e.g. Arndt, Alliger, & Andreasen, 1991; Liddle, 1987; Peralta, de Leon, & Cuesta, 1992). The different symptom types present in psychosis will now be discussed further. It is important to note that there is great variability both within and between patients. Thus, not all of types of symptoms will be experienced by every individual suffering with psychosis and some symptoms may be more prominent than others.

Positive Symptoms. Positive psychotic symptoms reflect the presence of experiences in the individual suffering with psychosis which are not present in the experiences of a non-psychotic individual (Cutting, 2003). Positive symptoms include hallucinations and delusions (Sims, 2002). Hallucinations are defined as sensory perceptions occurring in the absence of external stimuli. Delusions are defined as fixed false beliefs which are held with strong conviction and not shared by the individual's social environment. As with all types of human experience, the form and content of hallucinations and delusions varies considerably between individuals. Hallucinations can occur across a range of sensory modalities, including visual, auditory, gustatory,

olfactory and tactile domains (Bentall, 1990). Similarly, delusions can adopt numerous themes and fall into a variety of categories; including persecutory, religious and grandiose ideation (Garety & Hemsley, 1994). Other forms of positive psychotic symptoms include the so-called *first-rank* symptoms of schizophrenia, as defined by Schneider (1959). These are symptoms once thought to be particularly characteristic of schizophrenia. In addition to auditory hallucinations and delusions, first-rank symptoms include phenomena such as thought insertion, thought withdrawal and thought broadcast.

Negative Symptoms. In contrast to positive symptoms, negative symptoms refer to abilities that are "lost" or diminished in sufferers of psychosis (Cutting, 2003). Negative symptoms may remain even in periods of remission from positive symptoms and can be very debilitating (Ellenbroek & Cools, 2000). They are often linked with a poor outcome and problems with functioning (Blanchard, Mueser, & Bellack, 1998). These symptoms include a lack of motivation, flattened affect, social withdrawal, poverty of speech, and reduced emotion. Furthermore it may appear that an individual is unable to experience pleasure and this is referred to as physical and social anhedonia (Kirkpatrick & Buchanan, 1990).

Disorganised Symptoms. These symptoms refer to the disordered behaviour, speech and thought, often displayed by individuals suffering with conditions such as schizophrenia (Cutting, 2003). It could be argued that disorganised symptoms are a reflection of the broader cognitive disorganisation which potentially underlies psychosis (Basso, Nasrallah, Olson, & Bornstein, 1998). Disordered thought is often indicated by abnormal spoken language, whereby conversation may erratically jump from one topic to another. Alternatively, there may be a breakdown in grammatical structure such that speech appears illogical (Sims, 2002). Disorganised behaviour may include that which is considered inappropriate according to social norms, or abnormalities in mannerisms or posture (M. F. Green, 2001). These phenomena may appear objectively bizarre and as such can lead to problems in conducting the activities of daily life. Despite this, disorganised symptoms are not thought to be a predictor for subsequent quality of life following a psychotic episode (Ho, Nopoulos, Flaum, Arndt, & Andreasen, 1998).

1.2.2.2 Assessing single symptoms of psychosis

Single symptoms of psychosis can be assessed using a range of measures, including the Positive and Negative Syndrome Scale (PANSS; Kay, Fiszbein, & Opler, 1987). Rather than a diagnostic tool, the PANSS is designed to assess the presence and severity of positive, negative and general features of schizophrenia, based on items adapted from the BPRS. Operational criteria are provided for each item and ratings are made on the basis of both an interview and observations made by care staff and relatives. The authors have reported acceptable inter-rater, test-retest and internal reliability and the measure is often used to rate psychotic symptoms both clinically and for research purposes. Scales of a similar nature to the PANSS include the Scale for the Assessment of Negative Symptoms and the Scale for the Assessment of Positive Symptoms (SANS/SAPS; Andreasen, 1981, 1984). Devised as part of the Comprehensive Assessment of Symptoms and History (CASH; Andreasen, Flaum, & Arndt, 1992), these scales rate the presence and severity of negative and positive symptoms of schizophrenia using both interview and observational techniques. Both scales provide detailed clinical definitions for a large number of symptoms; have been demonstrated to have good internal reliability and validity; and are widely used in psychosis research (Grube, Bilder, & Goldman, 1998; Malla, Norman, & Williamson, 1993).

1.2.2.3 Aetiology of single symptoms of psychosis

Rather than proposing an all-inclusive explanation of the occurrence of psychosis, the single symptom approach aims to outline how specific psychological (or biological) processes may be involved in the development and maintenance of specific symptoms. Research in this area has mostly focused on the positive symptoms of psychosis. The finding that there are differential associations between psychological processes and different symptom types suggests that there may be independent mechanisms, and thus causal pathways, underlying the symptoms of psychosis. These will now be discussed in more detail.

Hallucinations. Hallucinations have been argued to stem from both difficulties integrating information into its temporal and spatial context (Hemsley, 1993); and problems with self-monitoring, such that the individual's own actions are

experienced as external and alien (Frith, 1992). In addition, other cognitive deficits have been highlighted in psychosis, including problems with executive function, memory and attention (M. F. Green, 1992). It is suggested that subtle deficits in information processing may leave individuals vulnerable to hallucinatory experiences, potentially via an increased sensitivity to stress (Cosway et al., 2000; McEwen & Sapolsky, 1995; Myin-Germeys, Krabbendam, Jolles, Delespaul, & van Os, 2002). Morrison (2001) builds on this notion of increased stress sensitivity, suggesting that hallucinations may be intrusions, similar to those occurring in anxiety disorders, but which have been misinterpreted as external events.

Whilst the presence of hallucinations has been attributed to a cognitive dysfunction, their content and associated distress have been linked to core beliefs that an individual holds about themselves and others based on their previous experiences (Smith et al., 2006). This fits with the cognitive model of psychosis (Garety et al., 2001) which suggests that psychotic symptoms are often personally meaningful and can be understood in the context of the individual's personal history.

Delusions. Delusions have been conceptualised as interpretations of anomalous experiences, arising as a result of normal cognitive processes (Garety et al., 2001). Maher (1988) suggests that extreme emotional reactions to anomalous experiences may trigger a search for explanation and meaning, potentially resulting in delusion formation. Furthermore, Garety et al. (2001) describe the role of cognitive biases in the development and maintenance of delusions. These are hypothesised to include a "jumping to conclusions" data gathering bias, which acts by prematurely terminating the search for meaning before full evidence has been acquired, often resulting in the adoption of a delusional appraisal (Garety & Hemsley, 1994). An externalising attribution bias has also been found to exist in individuals with psychosis, such that negative events are often attributed to an external source, e.g. other people or a conspiracy (Bentall, Kinderman, & Kaney, 1994). This may fuel the development of persecutory beliefs. Furthermore, Frith (1992) suggests that people with psychosis may suffer from a "theory of mind" deficit, such that they find it difficult to understand the mental representations of others (i.e. what another person is thinking based on clues from their actions and conversation). This could potentially result in a paranoid misinterpretation of the intentions of other people.

The cognitive model of psychosis suggests that cognitive biases operate in the context of an individual's core beliefs about both themselves and the world around them, also known as *schema* (Fowler, Freeman, Smith et al., 2006). Schematic beliefs develop based on life experiences and may in turn have an effect on the way any anomalous experiences are interpreted (Garety et al., 2001). For example, if events in an individual's life have led them to believe that other people are bad and that they are vulnerable, for example as a result of victimisation or trauma exposure; they may have a tendency to feel paranoid in anxiety provoking and ambiguous situations. Different schematic beliefs have been found to underlie different delusion subtypes (Fowler, Freeman, Smith et al., 2006).

1.2.2.4 Problems with the single symptom approach

Although the single symptom approach is more informative than the diagnostic approach in terms of highlighting the potential mechanisms underlying psychotic phenomena, it remains focused on symptoms as the major problem in psychosis. Moreover, it is mainly concerned with acute psychotic symptoms, with measures such as the SAPS and PANSS being relatively insensitive to minor symptom fluctuations (Leucht et al., 2005; Santor, Ascher-Svanum, Lindenmayer, & Obenchain, 2007). As such, symptom approaches ignore other aspects of psychosis, such as an individual's emotional response to the experience of psychosis as a major life event (Anthony, 1993; Gumley, White, & Power, 1999). Moreover, symptom approaches alone do not account for the heterogeneity of outcome present in psychotic disorders (Allardyce et al., 2007; Liberman & Kopelowicz, 2002). Therefore, a further way in which psychosis can be conceptualised is in terms of different types of recovery or outcome. This approach will be discussed in the next section.

1.2.3 Recovery from Psychosis

Psychosis is traditionally viewed as a debilitating condition with a poor outcome (Bleuler, 1908; Kraepelin, 1919). However, more recent studies have highlighted the heterogeneous nature of recovery from psychosis (Harding, Brooks, Ashikaga, Strauss, & Breier, 1987; G. Harrison et al., 2001). A study by Davidson and McGlashan (1997)

investigated the different outcomes experienced by individuals with psychotic diagnoses. About one third of patients were shown to experience a "good outcome", involving full remission of symptoms and limited problems with social and occupational functioning. Many other individuals experienced continuing residual symptoms and a relatively high level of social disability, with a smaller proportion of patients suffering repeated episodes of psychosis throughout their lives. Whatever the outcome, experiencing an episode of psychosis can be a very traumatic life event affecting an individual's confidence, self-esteem and functioning (McGorry et al., 1991). The complex nature of recovery from psychosis is reflected heavily in the literature. Studies generally focus around three main aspects of recovery: symptomatic recovery; functional recovery; and emotional and psychological well-being. These will now be discussed in more detail.

1.2.3.1 Positive symptom recovery

Symptomatic recovery from psychosis (i.e. the remission of hallucinations and delusions) is an important aspect of the recovery process and often forms the initial phase. This is what McGorry (1992) refers to as recovering from the "primary impairment" of psychosis. Recovery from positive psychotic symptoms is often dependent on an individual's response to medication (Johnstone, Crow, Frith, Carney, & Price, 1978; Kapur & Mamo, 2004), although psychological therapies have also been shown to be efficacious in symptom reduction (Fowler, Garety, & Kuipers, 1995; Kuipers et al., 1997; Wykes, Steel, Everitt, & Tarrier, 2008). Estimates vary in the literature, but it is suggested that around 12-50% of individuals make a full symptomatic recovery from an initial episode of psychosis, depending on when it was diagnosed and how it was treated (Jablensky et al., 1992; P. Mason et al., 1995; Rosen & Garety, 2005; M. Shepherd, Watt, Falloon, & Smeeton, 1989; Whitehorn, Lazier, & Kopala, 1998; Wiersma, Nienhuis, Slooff, & Giel, 1998). However, length of follow-up varies between studies and figures do not always take into account the prevalence of future psychotic relapse.

Recovery from psychotic symptoms has been hypothesised to occur through a number of different stages, and these have been argued to be a mirror image of the stages occurring in the development of psychotic symptoms. This is known as the "rollback

phenomenon" (Detre & Jarecki, 1971; Fava, 1999). Symptomatic recovery is proposed to involve a reduction in the frequency and severity of symptoms, combined with a gradual increase in insight and awareness. This may be accompanied by anxiety and depression as the individual begins to come to terms with what has happened to them (Carr, 1983; Drury, 1992; Sacks, Carpenter, & Strauss, 1974). These symptom recovery stages have been supported by research conducted on a large sample of individuals following the administration of antipsychotic medication (Mizrahi, Bagby, Zipursky, & Kapur, 2005). In this study, participants reported less cognitive and emotional preoccupation with symptoms shortly after the administration of medication, e.g. the idea or percept "doesn't bother me as much" (Winkelman, 1954). However, complete resolution of symptoms took longer to achieve and the authors suggest that, although antipsychotic medication may dampen the salience of psychotic phenomena, symptoms may require additional psychological deconstruction (Kapur, 2003).

Based on the above literature, individuals in recovery from a psychotic episode may experience residual symptoms which are low-level and arguably subclinical in nature, even when full-blown psychotic symptoms have remitted. This supports the use of a schizotypy measure to assess such phenomena, rather than a traditional psychosis assessment tool such as the PANSS (Kay et al., 1987). Low-level symptoms may have implications for relapse (Jorgensen, 1998; Subotnik & Nuechterlein, 1988) and also for quality of life and social recovery; although this is less clear (Malla & Payne, 2005). Thus, it is important that they are monitored. However, it must be remembered that symptomatic recovery is only one part of the wider recovery process. Indeed, although psycho-education about symptom management has been shown to be important, particularly in terms of preventing relapse by the acknowledgement of early warning signs (Birchwood et al., 1989; Gumley et al., 2003; Leete, 1989); the psychological impact of psychosis also needs to be considered (Mueser, Corrigan et al., 2002).

1.2.3.2 Functional recovery

Although variable, social and functional outcome in psychosis is frequently reported as poor, with long-term follow-up studies suggesting that less than 50% of people with non-affective psychosis achieve a social recovery, and only 10-20% of people return to competitive employment (G. Harrison, Croudace, Mason, Glazebrook, & Medley, 1996;

Jablensky et al., 1992; Johnstone, Macmillan, Frith, Benn, & Crow, 1990), despite the majority suggesting that they wish to work (Mueser, Salyers, & Mueser, 2001). Around 50% of people with bipolar disorder also fail to return to work and remain disabled (Tsai et al., 2001). Thus, many aspects of social functioning are affected by psychosis including employment, relationships and recreational activities (Birchwood, Smith, Cochrane, Wetton, & Copestake, 1990).

Studies have suggested numerous predictors of poor functional recovery from psychosis, including poor premorbid adjustment, adolescent onset of psychosis, lack of social support, and high levels of negative symptoms (Addington, Young, & Addington, 2003; Ho et al., 1998; Liberman, Kopelowicz, Ventura, & Gutkind, 2002; Schmidt, Blanz, Dippe, Koppe, & Lay, 1995). In addition, external societal factors have been hypothesised to be important, including the availability of roles in education and work within the local labour marker (Warner, 1985). Local economic factors, government policy, and individual variations in cultural values may also affect social recovery. It is likely that these relationships are mediated by societal effects on personal psychological factors, such as feelings of stigmatisation, beliefs about self and others, and the experience of social anxiety. Indeed, the emotional impact of psychosis has been postulated as a further explanation for poor social functioning (C. Jackson & Iqbal, 2002).

Published criteria state that functional improvement should be a core component when defining recovery from psychosis (e.g. Liberman et al., 2002; Whitehorn, Brown, Richard, Rui, & Kopala, 2002). However, this aspect of the recovery concept is seldom defined in psychiatric literature, with different studies using different measures to assess social outcome (Malla & Payne, 2005). For example, some studies utilise strict definitions when assessing functional recovery (i.e. at least ten hours per week in paid employment for at least a 12-month period); whereas others are much more subjective and define recovery as "getting back to normal" (Anthony, 1993; Hoffmann & Kupper, 2002). A major problem with defining and assessing functional recovery in psychosis is the lack of appropriate measures to do so. Existing measures designed to assess this dimension rely heavily on whether or not people have returned to competitive employment (e.g. American Psychiatric Association, 2000b; Beecham & Knapp, 1992; Goldman, Skodol, & Lave, 1992). Although work will always be a key marker of social

recovery, it is not the only marker of social improvement and there is a need to consider engagement in other domains of activity, such as voluntary work, education, and structured social activity. These activities may have a positive impact on confidence and self-esteem and thus be an important precursor to more formal involvement in economic activity such as work or education. In a longitudinal study, Wing and Brown (1970) showed that reduced time spent doing nothing, and increased social contact were the most reliable predictors of improvement in psychosis. However, few social functioning measures for use in psychosis measure daily activity and as such are insensitive to change on these domains.

In addition to research outlined above, service user literature suggests that "rebuilding life" and "reconnecting with the environment" are important aspects of recovery from psychosis (Chadwick, 1997; Pitt, Kilbride, Nothard, Welford, & Morrison, 2007). In a review of the recovery literature, Mueser et al. (2002) suggest that "recovery refers not only to short-term and long-term relief from symptoms, but also to social success and personal accomplishment in areas that the person defines as important" (p. 1273). This is corroborated in the service user literature and particularly by a personal account by Leete (1989), who states that:

> As I work, I become increasingly self-confident, and my self-image is bolstered. I feel important and grown-up, which replaces my usual sense of vulnerability, weakness and incompetence. Being a member of a work force decreases stigma and contributes to acceptance by my community, which in turn makes my life easier. (p. 197)

Thus, although symptomatic improvement does not necessarily result in functional improvement following psychosis; increased activity, and particularly work, can be therapeutic and have a positive impact on symptoms (Burns et al., 2007). Indeed, in another personal account, a service user explained how increased social contact actually reduced their feelings of paranoia (Anonymous, 1989). Research suggests that increased activity promotes other aspects of recovery via its positive effects of confidence and self-esteem; and potentially as a result of providing distraction from persistent symptoms (Waddell & Burton, 2006). Thus, functional recovery is an important area for research which should be considered in its own right rather than as an epiphenomenon of psychosis.

1.2.3.3 Emotional and psychological well-being

Psychosis has been described as an experience of sheer terror and panic (Forchuk, Jewell, Tweedell, & Steinnagel, 2003). Thus, encountering an episode of psychosis can be considered as a major life event with the potential to impact heavily upon emotional status and evaluative beliefs about self and others (McGorry et al., 1991; Morrison, Frame, & Larkin, 2003). Indeed, the experience of psychosis can be extremely personally threatening, particularly if the episode involved feelings of persecution (Shaner & Eth, 1989). This in turn can have a wider impact on long-term recovery from psychosis, even when symptoms have subsided (Chadwick, 1997; Fowler, 2002). Studies investigating the emotional and psychological impact of psychosis can be broadly split into two types: those examining the prevalence of emotional and psychological distress in large samples using standardised assessment tools; and smaller qualitative studies describing personal accounts of psychosis.

Social anxiety has been highlighted as a common feature of psychosis, with Social Anxiety Disorder (SAD) proposed to be present in up to one in three individuals with a diagnosis of schizophrenia (Birchwood et al., 2006). Social anxiety often emerges during the recovery phase and is argued to be reactive to the psychotic episode (Pallanti, Quercieli, & Hollander, 2004). Such anxiety is hypothesised to contaminate social interaction, thus leading to social withdrawal and poor functioning (Birchwood et al., 2006). Social withdrawal following psychosis is hypothesised to protect the self from the stigmatising views of society (Strauss, 1989). However, this may result in social isolation which can have devastating effects on an individual's self-esteem and in turn lead to increased social withdrawal (Garety et al., 2001). Indeed, there is evidence to suggest that an acute episode of psychosis is often accompanied by a reduction in social networks (Erickson, Beiser, & Iacono, 1999) which are very rarely replaced (H. J. Jackson & Edwards, 1992).

Depression is a further emotional disturbance which has been found to arise in the recovery stages of psychosis. A prospective study of post-psychotic depression (PPD) found that 36% of a group of 115 participants experienced low mood following psychosis (Birchwood, Iqbal, Chadwick, & Trower, 2000). Moreover, PPD has been found to be related to more frequent psychotic relapses, poorer social functioning, and

even suicide (Drayton, Birchwood, & Trower, 1998; Power et al., 2003). Inextricably linked with anxiety and depression, low self-esteem and elevated negative beliefs about self have also been highlighted as common in individuals recovering from psychosis (Gumley, O'Grady, Power, & Schwannauer, 2004; Gureje, Harvey, & Herrman, 2004). Furthermore, societal stigma may contribute towards negative beliefs about others (i.e. feelings that other people are hostile), thus producing threat responses and exacerbating residual paranoia (Birchwood, 2003; Trower & Gilbert, 1989).

Emotional disturbance during the recovery stages of psychosis is hypothesised to occur as a result of cognitive appraisals of psychosis, including loss of role, and feelings of hopelessness, shame and stigma (Birchwood, Mason, MacMillan, & Healy, 1993). Birchwood et al. (2006) illustrated that individuals experiencing social anxiety following an episode of psychosis, experienced greater shame attached to their diagnosis and also felt more socially marginalised than individuals without social anxiety. These associations remained even when controlling for depression. Estroff (1989) further postulates that social anxiety in schizophrenia may be triggered by the loss of social status that the stigma of "becoming a schizophrenic" often entails. Moreover, PPD is hypothesised to be linked with loss of autonomy and social role, combined with feelings of being entrapped in psychosis (Rooske & Birchwood, 1998). Anthony (1993) further elaborates on the concept of loss and suggests that recovery from the consequences of mental distress can be more difficult than recovery from symptom-related distress itself.

Personal accounts of recovery from psychosis also describe the lack of positive emotion present following an episode of psychosis (e.g. Chadwick, 1997; Deegan, 1997), including disempowerment and a loss of hope with regard to the future (Noordsy et al., 2002). This feeling of hopelessness is often instilled at a very early stage of the illness and is hypothesised to be related to the traditional idea that individuals with psychosis, particularly schizophrenia, will experience an inevitable and progressive downhill course (Corrigan, Giffort, Rashid, Leary, & Okeke, 1999). Overcoming this preconception is one of the main elements of recovery outlined in service user literature, a particular focus of which is the recovery of self-identity and regaining a sense of control and mastery over one's life (Deegan, 1988; Leete, 1989; Lovejoy, 1984; Pitt et al., 2007; Unzicker, 1989). In a review of this literature, Anthony (1993) defines

recovery as "a way of living a satisfying, hopeful, and contributing life even with the limitations caused by mental illness" (p. 14). He also refers to "the development of new meaning and purpose in one's life as one grows beyond the catastrophic effects of mental illness" (p. 14).

1.2.3.4 Summary of recovery literature

In summary, psychosis recovery literature focuses on numerous factors which are likely to be important when considering recovery from a psychotic episode. These include symptomatic recovery, functional recovery, and emotional and psychological well-being. Most studies tend to focus on these individual components in isolation when investigating recovery. However, it is likely that the different dimensions of recovery are overlapping and that individuals will encounter elements of numerous dimensions but at varying levels of severity, and at different time points throughout the recovery process (Liberman & Kopelowicz, 2002). Despite this, social disability and emotional recovery have always been argued to be somewhat separate issues to symptoms in psychosis, with improvement in the latter not necessarily dictating improvement in the former (e.g. Ganev, 2000). This thesis will argue that a psychological model incorporating low-level psychotic or *schizotypal* symptoms, which exist after the remission of the acute phase, may help to bridge this symptom-disability gap. The concept of schizotypal symptoms will be defined and discussed in the next section.

1.3 DEFINITION OF SCHIZOTYPY

Schizotypy is traditionally viewed as a non-pathological personality trait, akin to Schizotypal Personality Disorder (SPD) but occurring at a much lower level of severity; with lower levels of distress; and where social and occupational functioning are unaffected (Claridge, 1997b). Schizotypal symptoms resemble those outlined in DSM-IV criteria for SPD (e.g. social isolation, odd behaviour and thinking, and unusual perceptual experiences; see Table 1.1) and can also be thought of as being phenomenologically similar to low-level psychotic symptoms. Indeed, the presence of schizotypy is thought to denote an underlying genetic or biological vulnerability to psychosis (Meehl, 1990). Evidence for this approach comes from studies highlighting the increased presence of schizotypal personality traits in the relatives of patients with

psychotic disorders (Grove et al., 1991). In addition to this, longitudinal studies have suggested that transition rates to psychosis are higher in schizotypal than non-schizotypal individuals (Morrison et al., 2002). Whether or not an individual goes on to develop psychosis is thought to depend upon the interaction of their schizotypal predisposition with social and environmental conditions (Meehl, 1990). For example, if adverse life events occur, the individual may make the transition to psychosis. However, if environmental conditions are benign then it is likely that the individual will remain schizotypal, displaying characteristics which are reminiscent of psychosis but where the disorder itself is not present. Thus, it is argued that schizotypy exists on a continuum with psychosis (Meehl, 1990).

Table 1.1

DSM-IV Diagnostic Criteria for Schizotypal Personality Disorder (American Psychiatric Association, 1994)

A. A pervasive pattern of social and interpersonal deficits marked by acute discomfort with, and reduced capacity for, close relationships as well as by cognitive or perceptual distortions and eccentricities of behaviour, beginning by early adulthood and present in a variety of contexts, as indicated by five (or more) of the following:
   1. ideas of reference (excluding delusions of reference)
   2. odd beliefs or magical thinking that influences behaviour and is inconsistent with subcultural norms (e.g. superstitiousness, belief in clairvoyance, telepathy, or "sixth sense"; in children and adolescents, bizarre fantasies or preoccupations
   3. unusual perceptual experiences, including bodily illusions
   4. odd thinking and speech (e.g. vague, circumstantial, metaphorical, overelaborate, or stereotyped)
   5. suspiciousness or paranoid ideation
   6. inappropriate or constricted affect
   7. behaviour or appearance that is odd eccentric or peculiar
   8. lack of close friends or confidants, other than first-degree relatives
   9. excessive social anxiety that does not diminish with familiarity and tends to be associated with paranoid fears rather than negative judgments about self

B. Does not occur exclusively during the course of Schizophrenia, a Mood Disorder with Psychotic Features, another Psychotic Disorder, or a Pervasive Developmental Disorder.

Note: If criteria are met prior to the onset of Schizophrenia, add "Premorbid", e.g. "Schizotypal Personality Disorder (Premorbid)"

1.3.1 Epidemiology of Schizotypy

The prevalence of SPD is approximately 0.06-2.4%, depending on how it is assessed (Torgersen, Kringlen, & Cramer, 2001). Although this suggests that SPD is rare, a study conducted by Tien, Costa, and Eaton (1992) has proposed that personality traits in this domain are not. Forty percent of individuals surveyed were found to display DSM-III-R schizotypal personality traits without meeting full diagnostic criteria for personality disorder itself. This finding has since been replicated in other studies (e.g. Goulding, 2005; Johns & van Os, 2001; Joseph & Diduca, 2001; Verdoux et al., 1998).

Epidemiologically, the characteristics of individuals displaying schizotypal traits have been shown to be similar to those of individuals with psychosis. For example, gender and age differences found in schizophrenia have also been found to be reflected in dimensions of schizotypy (Spauwen, Krabbendam, Lieb, Wittchen, & van Os, 2003; Venables & Bailes, 1994). Furthermore, the social and environmental risk factors associated with schizophrenia, such as urbanicity and childhood trauma, have also been shown to be associated with increased levels of schizotypy (Krabbendam, Myin-Germeys, & van Os, 2004).

1.3.2 Aetiology of Schizotypy

The aetiology of schizotypy remains largely unknown, although it is argued that the construct shares similar aetiological mechanisms to those found in psychosis (e.g. Barkus, Stirling, Hopkins, & Lewis, 2006). Indeed, individuals with increased schizotypy scores have been shown to have increased levels of dopamine (Mohr, Landis, Sandor, Fathi, & Brugger, 2004), and to carry the COMT gene; a gene postulated to be important in the expression of schizophrenia (Avramopoulos et al., 2002). Moreover, schizotypal individuals show similar deficits in brain structure and function as individuals diagnosed with psychosis (Byrne, Hodges, Grant, Owens, & Johnstone, 1999; Raine, Sheard, Reynolds, & Lencz, 1992). On an individual symptom level, processes thought to underlie hallucinations and delusions in psychosis have also been shown to be related to anomalous experiences and low-level paranoia in non-

clinical samples (e.g. Freeman, Garety et al., 2005; Morrison, Wells, & Nothard, 2000). Individual schizotypal symptoms will now be discussed in more detail.

1.3.3 Types of Schizotypal Symptoms

Factor analytical studies of schizotypal phenomena in non-clinical populations have highlighted that they broadly fit into the same categories as psychotic symptoms (e.g. Bentall, Claridge, & Slade, 1989). The exact number of factors deemed to constitute schizotypy varies between studies, as do the labels attributed to them (Claridge, 1997a). Furthermore, there are methodological limitations in that different studies use different populations and measures of schizotypy to assess underlying factor structure (Suhr & Spitznagel, 2001). However, major reviews of factor analytic studies suggest that there is general evidence for "positive", "negative", and "disorganised" symptom dimensions of schizotypy (Claridge et al., 1996; Venables, 1995; Vollema & van den Bosch, 1995).

1.3.3.1 Positive schizotypal symptoms

The positive schizotypal symptoms factor is the most common factor emerging from factor analysis studies (Vollema & van den Bosch, 1995). It relates to unusual perceptual experiences and magical thinking styles and beliefs; arguably analogous to hallucinatory and delusional psychotic experiences. Unusual perceptual experiences do not specifically refer to hallucinatory-like occurrences, but also to perceptual distortions and hypersensitivities to sounds and smells, potentially forming the basis for later hallucinations (V. Bell, Halligan, & Ellis, 2006). Feelings of "de ja vu" and "presque vu" are other experiences included in this category (McCreery & Claridge, 2002). Odd beliefs and magical ideation may include extreme religious beliefs or conviction in ideas such as telepathy, ESP, and clairvoyancy (Chequers, Joseph, & Diduca, 1997; Clarke, 1991). It is important to remember that positive schizotypal symptoms are not necessarily pathological in themselves. Many individuals are not distressed by their experiences and can successfully integrate them into their lives (M. Jackson, 1997). However, these phenomena could be considered as analogous to the "anomalous conscious experiences" which feature in Garety et al's (2001) cognitive model of psychosis.

Some studies have renamed this factor the "cognitive-perceptual" component of schizotypy, reflecting the nature of the phenomena included in this domain and creating some ideological distance from psychotic symptomatology (Bergman et al., 1996; Raine et al., 1994). Furthermore, others have separated positive schizotypal symptoms into two factors, usually distinguishing unusual perceptual experiences from paranoid-like beliefs (Suhr & Spitznagel, 2001).

1.3.3.2 Negative schizotypal symptoms

Negative schizotypal symptoms refer to phenomena relating to social withdrawal and anhedonia (Bergman et al., 1996). It could be argued that these symptoms are reminiscent of the notion of the reduced ability or expectation of psychotic individuals to experience pleasure from social and physical stimulation. However, other studies suggest that in schizotypy, this factor has a more specific social anxiety and social impairment connotation rather than referring to physical anhedonia (Gruzelier, 1996; Venables & Rector, 2000). Indeed, Raine et al. (1994) labelled this factor "Interpersonal Schizotypy", with loadings from schizotypal symptom types such as excessive social anxiety, constricted affect, and a lack of close friends. Moreover, O. Mason, Claridge, and Williams (1997) refer to this factor as a tendency towards introverted, emotionally flat and asocial behaviour.

1.3.3.3 Disorganised schizotypal symptoms

This factor refers to a tendency for thoughts and speech to become disorganised, perhaps reflecting the same underlying cognitive disorganisation shown to be present in psychotic illness (Liddle, 1987). Types of schizotypal phenomena loading on this factor include odd behaviour and odd speech (Raine et al., 1994; Vollema & Hoijtink, 2000) including symptoms such as unusual mannerisms; eccentric habits; and bizarre usage of words. Gruzelier (1996) has suggested a link between this factor and eccentricity. Some studies have argued for the existence of an "asocial" component as well as, or as an alternative to, the disorganised factor of schizotypy (e.g. Bentall et al., 1989). Asocial schizotypy is thought to reflect impulsive nonconformity and the disposition to unstable mood, particularly with regard to rules and social conventions (Shean & Wais, 2000).

However, it could be argued that asociality may be linked with disorganised symptoms, the latter perhaps influencing the former.

1.3.4 Schizotypy and the Development of Psychosis

There is longitudinal evidence to suggest that individuals scoring high on trait schizotypy may be at increased risk for the development of psychosis. Indeed, the Edinburgh High Risk study highlighted that individuals with increased genetic risk of psychosis reported higher levels of schizotypy than a control group (P. Miller, Byrne et al., 2002). Furthermore, within the high-risk group, baseline levels of schizotypal symptoms (particularly items from the 'Social Withdrawal' factor) were reasonably accurate at predicting which individuals did and did not go on to develop psychosis at a later time point (Johnstone, Ebmeier, Miller, Owens, & Lawrie, 2005). Morrison et al. (2002) have also demonstrated that individuals in a wider high-risk group score higher on the O-LIFE than non-clinical controls. Other longitudinal studies have suggested a 16- to 60-fold increase in risk of transition to psychosis in individuals who reported having psychotic-like experiences in childhood and adolescence (van Os, Linscott, Myin-Germeys, Delespaul, & Krabbendam, 2009). For example, Poulton et al. (2000) report that 25% of children with psychotic-like experiences aged 11 had developed schizophreniform disorder at age 26.

Despite findings from these studies, it must be remembered that not all individuals with high schizotypy go on to develop psychosis. In a large survey of high school students, McGorry et al. (1995) showed that prodromal symptoms were extremely prevalent among older adolescents and are therefore unlikely to be a specific indicator, or an accurate predictor, of subsequent schizophrenia. There is also evidence to suggest that the presence of schizotypy may have positive effects (e.g. creativity; O'Reilly, Dunbar, & Bentall, 2001). Moreover, longitudinal studies have suggested that many people reporting schizotypal phenomena at baseline have a good outcome and report few or no schizotypal phenomena at follow-up (Hanssen, Bak, Bijl, Vollebergh, & van Os, 2005; Wiles et al., 2006). Thus, only a small proportion (approximately 8%; Hanssen et al., 2005) go on to develop a clinical disorder.

It may be the case that schizotypal symptoms reflect an underlying (and potentially genetic) vulnerability to psychosis which, when interacting with environmental risk factors, may result in the onset of psychosis. A recent review and model by van Os and colleagues (2009) supports this claim, suggesting that environmental risk factors such as trauma (Spauwen, Krabbendam, Lieb, Wittchen, & van Os, 2006), cannabis use (Henquet, Murray, Linszen, & van Os, 2005), and urbanicity (Spauwen, Krabbendam, Lieb, Wittchen, & van Os, 2004) act synergistically with underlying vulnerability to psychosis, potentially leading to the onset of psychotic disorder. It is argued that exposure to these environmental risk factors may have an impact on behavioural and neurotransmitter sensitisation, thus increasing both the persistence of schizotypal experiences and the need for care (Cougnard et al., 2007).

The psychosis-proneness-persistence-impairment model outlined by van Os et al. (2009) has clinical implications for both early intervention and at-risk mental state services. Moreover, it may be useful in interpreting longitudinal findings such as those from the Edinburgh High Risk Study (Johnstone et al., 2005). Indeed, as a group individuals at genetic high-risk of psychosis scored higher on schizotypy than non-clinical controls, whereas only a small proportion (around 12%) developed psychosis. The authors note that the vulnerability to schizophrenia occurs in many more people than will develop the illness. Thus, whilst informative, genetic risk and the mere presence of schizotypal symptoms are likely to be insufficient in defining transition to psychosis. As outlined by Morrison et al. (2002), screening for psychosis using schizotypy measures alone is likely to result in a high rate of false positives. Consideration of other environmental and psychological factors is therefore warranted.

1.3.5 Trait Schizotypy vs. State Schizotypal Symptoms

Schizotypy is a useful concept to measure in psychosis as it may help to illuminate the processes involved in the transition from a benign to disordered state (Raine & Lencz, 1995). However, schizotypy is currently conceptualised as a stable personality *trait* which acts as a potential risk factor for the later development of psychotic disorder. In contrast, this thesis is interested in the somewhat novel concept of *state* fluctuations in individual schizotypal symptoms following an episode of psychosis, and their relationship with recovery.

Yue, Bidwell, and Norton (2006) outline the differences between traits and states, with particular reference to psychiatric disorders. Traits are defined as markers of an individual's risk of developing a given disorder. Furthermore, they are thought to reflect the behavioural and biological processes which may play a causal role in the pathophysiology of the disorder. Traits are suggested to be present in the biological relatives of patients; to remain stable over time; and to be unresponsive to treatment, due to the fact that they are not overtly pathological. Conversely, states are argued to reflect the current clinical manifestation of a disorder in patients at a given time. States are suggested to vary over time and with clinical course and treatment.

When considering the trait-state distinction in terms of schizotypy, trait schizotypy can be viewed as a relatively stable personality trait reflecting individual differences in psychosis proneness and the likelihood of experiencing schizotypal symptoms. Thus, a high-trait schizotypal individual would be more likely to develop psychosis than a low-trait schizotypal individual (P. Miller, Byrne et al., 2002). However, high trait schizotypy is not sufficient for the development of psychosis. Two individuals may score exactly the same on a measure of trait schizotypy yet one may go on to develop clinically-defined psychosis whilst the other simply remains at risk. As such, a trait measure of schizotypy does not provide any information on the current level of schizotypal symptoms being experienced. Rather, trait measures focus on general dispositions or tendencies (Matthews & Deary, 1998).

Conversely, state schizotypy would provide information on the level of schizotypal phenomenology being experienced in a given situation, thus enabling fluctuations in symptoms to be monitored over time. Rather than referring to general tendencies and personality characteristics, a state measure would enquire whether a specific phenomenon (e.g. feelings of paranoia) had occurred recently (Yue et al., 2006). As such, a state measure would account for the limitations of trait assessments and allow heterogeneity to be detected amongst high trait schizotypal individuals who would otherwise appear as a homogenous group. For example, an individual who has felt consistently paranoid for the last week (high state) would arguably be at greater risk of descending into a spiral of psychosis than an individual who has a general tendency to feel paranoid in certain situations (high trait). A measure of state schizotypal symptoms

could be used not only to assess risk of transition to psychosis but also low-level psychotic experiences occurring as residual symptoms following an episode of psychosis. Thus, there is great utility in the state schizotypy concept. Despite this, a measure of state schizotypal symptoms does not currently exist.

The following section will argue for a relationship between state schizotypal symptoms and psychosis and thus for the development of a measure to assess their presence.

1.4 THE RELATIONSHIP BETWEEN SCHIZOTYPAL SYMPTOMS AND PSYCHOSIS

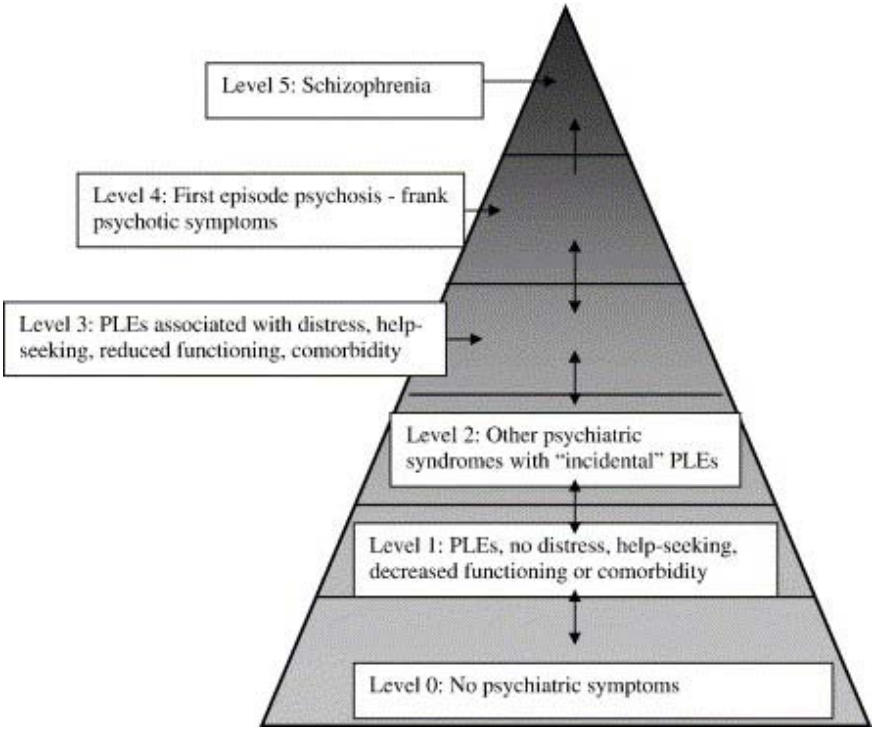
Although the notion of assessing state schizotypal symptoms is somewhat novel, trait schizotypy has long been linked with psychosis via the continuum hypothesis (Meehl, 1962; Strauss, 1969). The term "continuum" is used to refer to constructs which range from one condition to another condition via gradual transitions as opposed to abrupt changes. This is in contrast to the concept of "dichotomisation" whereby a construct can be sharply divided into two obviously distinguishable categories (Soanes & Stevenson, 2005).

1.4.1 The Continuum Hypothesis of Psychosis

The continuum hypothesis of psychosis states that "psychosis-like beliefs, perceptual distortions, and idiosyncrasies of thought and communication, considered the hallmark diagnostic criteria for psychosis, are distributed (albeit to varying degrees) throughout the general population" (V. Bell et al., 2006, p. 366). This is as opposed to traditional Kraepelinian categorical views of the disorder, which advocate the notion of a dividing line between sanity and madness. The concept of a continuum of psychosis was first suggested by Bleuler (1908) who proposed that schizophrenia was an "intensification of an already existing character". Rado (1953) later coined the term *schizotypal* in reference to the schizophrenia phenotype. Meehl (1962) elaborated on this suggesting that both schizotypy and schizophrenia arise from the existence of *schizotaxia*, an integrative neural defect, which is inherited. A predisposed schizotaxic individual will develop as schizotypal if the social learning conditions are benign. However, if adverse environmental events occur, the individual may develop schizophrenia.

This theory has since been supported by a wealth of research including studies providing evidence that schizotypal and psychotic symptoms have a similar epidemiology and share similar risk factors (Arseneault, Cannon, Witton, & Murray, 2004; Boydell et al., 2001; Johns et al., 2004; Morrison et al., 2003; van Os, Jones, Sham, Bebbington, & Murray, 1998; van Os, Verdoux et al., 1999; Verdoux & van Os, 2002). Longitudinal studies also indicate that individuals with high schizotypy are at an increased risk of developing a clinical disorder. Chapman, Chapman, Kwapil, Eckblad, and Zinser (1994) reported that high scores on schizotypy measures predicted higher frequencies of both psychosis and mood disorders over the next ten years. These results have since been replicated (Kwapil, Miller, Zinser, Chapman, & Chapman, 1997; Verdoux et al., 1999) and provide yet further evidence for the continuum hypothesis.

Yung et al. (2006) add further clarity to the continuum concept by outlining different dimensional stages on the psychosis continuum, i.e. different mental states (see Figure 1.1). These include the absence of psychotic experiences at the lowest end of the continuum; followed by the presence of psychotic-like experiences with and without distress; frank psychotic symptoms; and psychiatrically defined schizophrenia at the upper end. An important point of this model is the consideration that individuals can move between different groups and thus up and down the continuum over time (illustrated by the arrows on Figure 1.1). Where an individual lies on the continuum at one particular time denotes their current risk of a psychotic episode. This adds a state element to the continuum model and a consideration of the fact that an individual's mental state is not static but rather changes over time.



Note. PLE = Psychotic-like Experience

Figure 1.1

Theoretical model of the psychosis continuum (taken from Yung et al, 2006)

1.4.2 The Importance of Assessing Schizotypal Symptoms in Psychosis: Indictors of Recovery

Adopting a continuum view of psychosis has been shown in previous research to be important and useful for a number of reasons. First, individuals who are schizotypal but not psychotic provide an analogue sample in which processes thought to be potentially related to psychosis can be investigated without the influence of the disease process or medication effects (Raine & Lencz, 1995). Second, a dimensional approach may help to identify individuals who are "at-risk" of developing psychosis and thus enable potential early intervention (Gooding, Tallent, & Matts, 2005). Finally, from a therapeutic perspective, the idea that psychosis exists on a continuum with normality is both important in assisting individuals in adopting a normalising appraisal for their experiences (Freeman, Garety et al., 2005); and in supporting the use of techniques found useful in other, more common disorders, such as anxiety and depression, to treat psychosis (Kuipers et al., 2006).

At present, schizotypy is considered as being premorbid to psychosis and is not generally assessed after transition has been made. This thesis will argue that assessing state schizotypy in individuals already diagnosed with psychotic disorders may be particularly informative in relation to monitoring recovery from a psychotic episode. Although clinically definable psychotic symptoms dissipate following an episode of psychosis, individuals may still be experiencing residual psychotic symptoms of a subclinical nature, similar to those experienced in the prodromal phases of the disorder (Keitner et al., 1996). As outlined previously, there is evidence to suggest a relationship between residual and prodromal symptomatology via the "rollback phenomenon" (e.g. Detre & Jarecki, 1971; Fava, 1999). This suggests that individuals may inhabit a *postdromal* phase following the remission of an acute episode of psychosis, and supports the suggestion that it is possible to move up and down different points on the psychosis continuum over time (Yung et al., 2006). It is important that these low-level symptoms are measured as they may have implications for future relapse (Birchwood, 1995). In addition, it may be postulated that particular types of low-level psychotic, or schizotypal, symptoms (e.g. extreme social anxiety) may be directly related to impairments in social functioning, thus influencing longer-term recovery from

psychosis. However, due to their subclinical nature, such symptoms cannot be detected using traditional psychiatric assessment tools for psychosis (e.g. PANSS; Kay et al., 1987). It could therefore be argued that a measure of current schizotypal symptoms would be more appropriate in the assessment of these phenomena.

Currently, no measures exist which specifically assess low-level psychotic symptoms during the recovery phase. Studies investigating residual symptoms tend to set their own criteria for the definition of these phenomena (e.g. Buchanan et al., 2005). This usually consists of a specified cut-off point on psychiatric assessment tools for psychosis (e.g. PANSS, BPRS), thus conceptualising residual psychotic symptoms as attenuations of those symptoms present during the acute episode (Strakowski et al., 1998). As such, these measures do not assess any emotional dysfunction occurring as a result of the episode itself. Other methods for assessing low-level psychotic symptoms following psychosis include "early warning signs" tools (Birchwood, MacMillan, & Smith, 1992). Early warning signs (EWS) are described as subtle changes in thought, affect and behaviour preceding the onset of psychosis (Birchwood, Spencer, & McGovern, 2000). Research undertaken in this domain suggests that EWS can be observed in 50-70% of individuals up to a month prior to relapse (Birchwood, 1995; A. G. Jolley, Hirsch, Morrison, McRink, & Wilson, 1990; A. Tait, McNay, Gumley, & O'Grady, 2002). The Early Signs Scale (ESS; Birchwood et al., 1989) was developed to assess the prodromal signs of relapse, and combines non-specific prodromal signs (e.g. dysphoria, sleep and appetite problems) with an idiosyncratic relapse signature based on the events of the previous episode. Although the EWS literature highlights the importance of assessing low-level symptoms in individuals recovering from acute psychosis, the ESS was designed specifically to investigate processes associated with psychotic relapse, rather than other forms of recovery from psychosis (e.g. social recovery). Thus, there is a need for a measure which could be used to accurately assess and monitor low-level psychotic symptoms, both prior to and following a psychotic episode, which could then be used to investigate relationships with different dimensions of recovery from psychosis. This will be the main focus of this thesis.

1.5 SUMMARY AND AIMS OF THESIS

This introductory chapter has defined the major concepts of interest in this thesis, namely psychosis and schizotypy, and reviewed the current theories proposed for their existence. Moreover, the problem of social disability in psychosis has been highlighted, as has its potential relationship to schizotypal symptoms in the recovery phase of psychosis. The main focus of this thesis is the conceptualisation of schizotypal symptoms as both a prodromal and residual feature of psychosis. It is argued that schizotypal symptoms may be at the core of psychosis, potentially providing good predictors of prognosis. In the context of this somewhat novel approach, schizotypal symptoms are therefore viewed as being analogous to (or at least comparable with) low-level or residual psychotic phenomena. As such, the terms *schizotypal*, *low-level*, and *residual* will be used interchangeably. In order to advance our understanding of psychosis (and recovery from), this thesis will argue that there is a need to focus on the nature of schizotypal symptoms, and to examine how they are associated with psychological processes. These self-reported minor variations may provide a greater insight into the nature of psychosis than acute symptoms themselves, which may be a transient "flare-up" of a long-standing underlying problem. It is predicted that akin to research conducted on psychotic symptoms, and in line with the continuum hypothesis, different schizotypal symptoms will be associated with different psychological processes.

In order to investigate the nature of schizotypal symptoms and social recovery from psychosis, this thesis compares a recovering early psychosis sample and a non-clinical student sample on a variety of variables. As all current measures of schizotypy utilise a personality trait approach, the development of a tool which also enables state schizotypy to be assessed is required. Therefore, the initial aim of this thesis is to modify an existing self-report measure of trait schizotypy for use to assess state schizotypal symptoms in both clinical and non-clinical populations. The following chapter reviews existing measures of schizotypy and tools for the assessment of low-level psychotic symptoms. The modification of a self-report measure of schizotypy to assess both state and trait schizotypy is then described, and psychometric data on the scale are reported. Following this, the prevalence of schizotypal symptoms in clinical and non-clinical samples is reported and compared.

Cognitive models of psychosis utilise a single symptom approach and stress the importance of separate but interacting aetiologies underlying different types of symptoms (Garety et al., 2001). Therefore, a further aim of this thesis is to investigate the associations between different types of schizotypal symptoms and a number of psychological and neuropsychological variables. A dissociation between the correlates of different types of symptoms is predicted and it is hypothesised that distinct underlying mechanisms are responsible for different kinds of symptoms. In the second study, associations between schizotypal symptoms and neuropsychological variables are examined in the clinical sample. It is predicted that where present, symptoms relating to anomalies of experience (e.g. low-level hallucinations) will be associated with specific neuropsychological and neurological variables, namely anomalies in visual processing. Conversely, it is hypothesised that low-level paranoia and social anxiety will not be associated with these variables. Thus, it is argued that there is a more organic and biological basis to anomalous symptoms. In the third study, associations between schizotypal symptoms and psychological and emotional variables are investigated in the non-clinical sample. It is predicted that social anxiety and paranoid schizotypal symptoms will be strongly associated with emotional and psychological variables, with anomalous schizotypal experiences being associated with these variables to a lesser extent. Following on from this, in the fourth study, relationships between schizotypal symptoms and trauma will be examined in the non-clinical sample. Hypothesised routes from trauma exposure to schizotypal symptomatology are discussed.

Following these studies examining the differential relationships between different schizotypal symptom types and emotional, psychological, and neuropsychological variables, the relationship between schizotypal symptoms and outcome is examined and a hypothesised dimensional model of recovery is discussed. The final chapter of this thesis will then experimentally test the hypothesised associations between schizotypal symptoms and social recovery. This investigation will be conducted using longitudinal data from a randomised controlled trial. The trial aims to examine the impact of a social recovery focused therapeutic intervention on schizotypal symptoms and time spent in structured activity.

CHAPTER TWO:
STUDY ONE: DEVELOPMENT AND PSYCHOMETRIC PROPERTIES OF THE SCHIZOTYPAL SYMPTOMS INVENTORY (SSI)

2.1 RATIONALE AND CONTEXT FOR THE STUDY

Traditional measures of the symptoms of psychosis (e.g. PANSS; Kay et al., 1987) are designed in relation to frank psychosis, and lack the sensitivity needed to detect subclinical psychotic phenomena. However, subclinical phenomena may be important in both the transition to, and recovery from, psychotic disorders. Early subclinical and anomalous experiences may later develop into full-blown psychotic symptoms, particularly if occurring very frequently, and thus it is important that they are measured and not missed (Hodges, Byrne, Grant, & Johnstone, 1989). Likewise, when they are recovering, many people with schizophrenia report a reversion to subthreshold psychotic symptoms. For example, a persecutory delusion may fade into an anxious feeling of being "noticed" or "standing out" in social situations. In the wrong conditions, these subclinical phenomena may once more escalate into a further episode of psychosis (Birchwood, Spencer et al., 2000).

Due to the overlap between schizotypal phenomena and subclinical psychotic symptoms (Bedwell & Donnelly, 2005), schizotypy measures could be used to assess the residual and prodromal symptoms of psychosis. However, a major problem is that existing measures (L. J. Chapman & Chapman, 1980; O. Mason et al., 1997; Venables, Wilkins, Mitchell, Raine, & Bailes, 1990) tend to assess schizotypy as a personality trait, rather than a mental state. While such measures can be used to highlight underlying vulnerability, they provide little information about current symptom profiles. Moreover, well-formulated theories now exist linking anxiety with the development of schizotypal phenomena (Birchwood, 2003; Fowler, 2000b), and there are therefore arguments for including measures of anxiety, particularly social anxiety, in an instrument designed to evaluate the ebb and flow of quasi-psychotic experience.

The specific aim of the first part of this chapter is to review existing measures for use in the assessment of schizotypal phenomena and low-level psychotic symptoms. Many of

the measures discussed have been used to assess the prevalence of schizotypal and low-level psychotic symptoms in the general population (e.g. Myin-Germeys, Krabbendam, & van Os, 2003). The suitability of these measures for assessing schizotypal symptoms in clinical samples will be considered. Following this review, the need for a new measure for use in this domain will be outlined and discussed.

2.2 REVIEW OF MEASURES TO ASSESS SCHIZOTYPY AND LOW-LEVEL PSYCHOTIC SYMPTOMS

This section will review four types of measures for the assessment of schizotypy and low-level psychotic symptoms. These include: measures designed to assess borderline conditions; measures designed to assess personality types related to psychosis; measures designed to assess schizotypy and schizotypal traits; and measures designed to assess the prodromal symptoms of psychosis.

2.2.1 Measures Designed to Assess Borderline Conditions

Tools designed to assess schizotypal personality originate from studies detecting borderline psychotic symptoms in the first-degree relatives of individuals diagnosed with psychosis (e.g. Kety, Rosenthal, Wender, Schulsinger, & Jacobsen, 1974). These findings led to the suggestion that psychosis exists on a continuum, ranging from normality to a clinically-definable disorder. As such, early schizotypy measures took the form of structured and semistructured interviews designed to highlight borderline phenomenology. Such measures are predominantly based on diagnostic criteria for Schizotypal Personality Disorder (SPD), as devised by Spitzer, Endicott, and Gibbon (1979).

Tools designed to assess borderline symptoms are outlined in more detail in Table 2.1. Such measures include the Diagnostic Interview for Borderlines (DIB; Kolb & Gunderson, 1980); the Symptom Schedule for the Diagnosis of Borderline Schizophrenia (Khouri, Haier, Rieder, & Rosenthal, 1980); and the Schedule for Schizotypal Personalities (SSP; M. Baron, Asnis, & Gruen, 1981). Perhaps the most well-known measure in this area is the Structured Interview for Schizotypy (SIS; Kendler, Liebermann, & Walsh, 1989). The SIS was developed from a large family

study of schizophrenia. It differs from other interviews in this domain as is not solely based on diagnostic criteria but includes coverage of other, potentially relevant signs and symptoms of at-risk mental state. The SIS has been shown by an independent pilot study to discriminate significantly between the relatives of individuals with schizophrenia and the relatives of controls, and thus demonstrates a high level of validity (Tsuang, Stone, Tarbox, & Faraone, 2002). Furthermore, the SIS has been used to identify "high-risk" individuals (e.g. P. Miller, Byrne et al., 2002). However, due to the fact that it is a semistructured interview, the SIS is reasonably lengthy and resource-heavy to administer.

A problem common to the majority of borderline assessment tools is that they assess personality disorders as opposed to personality types. Furthermore, rather than being necessarily specific to schizotypy, some measures assess all borderline personality disorders (e.g. DIB). Although there is evidence to suggest an overlap between personality disorders and psychosis, the extent of this overlap is unclear and forms part of a controversial debate which is still unresolved (Pope, Jonas, Hudson, Cohen, & Tohen, 1985). Therefore, interviews designed to assess borderline conditions could be argued to be somewhat limited in their usage in assessing the prodromal and/or residual signs of psychosis.

2.2.2 Measures Designed to Assess Personality Types Related to Psychosis

Studies have suggested that factors indicative of liability to the development of psychosis are broader than those included in SPD criteria (Tsuang et al., 2002). Broader, non-specific personality assessment measures have also been used to assess psychosis-like characteristics. These include general personality inventories, such as the Minnesota Multiphasic Personality Inventory (MMPI; Hathaway & McKinley, 1951), which has "paranoia" and "schizophrenia" subscales; and the Sixteen Personality Factor Questionnaire (16 PF; Cattell, Eber, & Tatsuoka, 1970) which has a "trust-suspiciousness" factor. However, these measures could be argued to lack adequate specificity when trying to identify individuals who may be at-risk (Muntaner, Garcia-Sevilla, Fernandez, & Torrubia, 1988).

A more specific psychosis-related personality measure is the Psychotism, or P-scale, designed by Eysenck and Eysenck (1976). Psychoticism is considered to be a dimension of normal personality which includes criminality, psychopathy, and manic-depressive disorder; with schizophrenia residing at the most extreme end. The P-scale was designed to assess this construct and the earliest form of the measure was included as part of the Eysenck Personality Questionnaire (EPQ; H. J. Eysenck & Eysenck, 1975). The initial P-scale contained several items with psychotic content (e.g. paranoid ideation and anhedonia) but also included items which tapped antisocial, impulsive and non-conformist traits. The inclusion of these items was influenced by research conducted by the authors suggesting that such traits were elevated in the relatives of individuals with psychosis. However, many studies have shown that anti-social and non-conforming individuals (who may or may not also experience psychotic symptoms) also have elevated scores on this scale (Farrell, 1992; Rahman, 1992). Several criticisms have been made against the measure in relation to this point, and also regarding the internal validity of the scale (e.g. Claridge, 1983). As a result of these criticisms, the P-scale was revised (S. B. Eysenck, Eysenck, & Barrett, 1985) but problems with the measure still remain. Zuckerman (1989) labelled the P-scale as a tool for assessing psychoticism as opposed to psychosis proneness. Furthermore, even in its revised form, the scale been shown to have weak predictive reliability in terms of diagnosing future psychosis (J. P. Chapman, Chapman, & Kwapil, 1994). Moreover, in factor analytic studies, rather than loading on psychotic-like symptom factors, the P-scale has a tendency to load on factors reflecting impulsive non-conformity, thus further questioning the validity of the scale for use in psychosis research (Bentall et al., 1989; Claridge et al., 1996; Raine & Allbutt, 1989).

General personality tools do not have adequate specificity to detect schizotypal symptoms. This has resulted in the development of measures devoted to the assessment of schizotypy, which will be discussed in the next section.

2.2.3 Measures Designed to Assess Schizotypal Personality Traits

These measures attempt to detect experiences occurring outside of clinically-defined psychosis, but which may highlight a predispositional vulnerability to the disorder.

They can be split into two types: those assessing schizotypy as a fully dimensional personality trait; and those assessing schizotypy as attenuated psychotic symptoms.

2.2.3.1 Measures designed to assess schizotypy as a fully dimensional trait

The conceptualisation of schizotypy as a fully dimensional personality trait can be explained using an extrapolation of Reich, James, and Morris' (1972) multiple-threshold liability model. This model suggests that SPD is a milder and more prevalent expression of the schizophrenia genotype. With this theory in mind, it could be argued that schizotypal traits are an even milder expression of schizophrenia genotype, with a lower liability than SPD, and a greater prevalence in the general population. A complete absence of schizotypal traits may occupy the lowest end of this dimension. Measures in this domain include those assessing individual schizotypal traits and others which assess schizotypy in a multidimensional manner. A summary of these measures is provided in Tables 2.2 and 2.3.

Measures assessing individual schizotypal traits. Most of the scales belonging to this group were created and validated using non-clinical populations. Items are based on phenomena thought to be linked to psychosis, but their content focuses on personal experience rather than diagnostic criteria. Each scale taps a different type of schizotypal phenomena, with many items used to assess each trait. The most commonly used scales in this domain include the Perceptual Aberration Scale (PER; L. J. Chapman, Chapman, & Raulin, 1978) which measures the tendency to perceptual distortion; and the Magical Ideation Scale (MIS; Eckblad & Chapman, 1983) which asks about superstitions and magical beliefs (e.g. experiences of precognition, mind-reading, etc). Other scales assess negative-type phenomena, such as loss of pleasure, as assessed by the Physical and Social Anhedonia Scales (PhA and SoA; L. J. Chapman, Chapman, & Raulin, 1976); and social isolation and social anxiety, as measured by the Revised Social Anhedonia Scale (Eckblad, Chapman, Chapman, & Mishlove, 1982) and the Social Fear Scale (Raulin & Wee, 1984). All of the scales demonstrate an impressive range of content, and have been shown to be reliable and valid (L. J. Chapman, Chapman, & Miller, 1982). However, the fact that the scales were validated using only non-clinical individuals does restrict their usage; although one study reports

successful use of the PhA scale in an acute psychosis population (Kontaxakis et al., 2006).

Other scales in this domain assess personality traits which are not obviously psychotic but which have been suggested to be characteristic of schizotypy (Meehl, 1962, 1990). These include the Intense Ambivalence Scale (IAS; Raulin, 1984), later revised to become the Schizotypal Ambivalence Scale (SAS; Kwapil, Mann, & Raulin, 2002); the Cognitive Slippage Scale (Miers & Raulin, 1985), designed to assess subtle thought disorder; and the Impulsive Nonconformity Scale (ImpNon; L. J. Chapman et al., 1984), designed to measure impulsive anti-social behaviour. Although these scales are not as well-used as other measures, they have been shown by their respective authors to have good psychometric properties and to have validity in assessing psychosis proneness.

The Chapman scales have been widely used in research into schizotypy and psychosis proneness; both in investigating the correlates of schizotypy, and in assessing the predictive reliability of schizotypy and the later development of psychosis (e.g. Gooding, Tallent, & Hegyi, 2001; Raine & Manders, 1988). However, some studies have questioned the stability of the scales in assessing psychosis proneness over time (Meyer & Hautzinger, 1999). Although the measures do predict onset of other schizotypal symptoms and psychotic-like experiences, their predictive reliability in diagnosing psychotic disorders has been shown to be low (J. P. Chapman et al., 1994; Kwapil, Raulin, & Midthun, 2000).

Although there is some value in using scales devoted to specific types of schizotypal traits, a multidimensional measure of schizotypy may be more accurate in reflecting an individual's symptom profile. This idea is reinforced by the suggestion that it is the range of schizotypal symptoms, rather than the symptoms themselves, which is indicative of an individual's risk of transition to psychosis (P. Miller, Byrne et al., 2002). As such, the use of multiple as opposed to single measures may preferable in the detection of schizotypic subjects (G. W. Barnes, Rhinewine, & Docherty, 2000). Measures assessing different aspects of schizotypy have different psychometric properties, and may also have different response formats (e.g. forced choice vs. Likert) and time scales (e.g. lifetime vs. recent prevalence). Therefore, their comparability and amalgamation is somewhat questionable. It is potentially more appropriate to use

measures which assess a range of schizotypal symptoms, as opposed to a range of measures which each assess an individual schizotypal symptom.

Multidimensional schizotypy measures. In order to ensure comprehensive measurement of schizotypal phenomena, Bentall, Claridge, and Slade (1989) developed the Combined Schizotypal Traits Questionnaire (CSTQ); a lengthy 420-item scale combining 18 of the previously discussed schizotypy assessment tools (e.g. PER, MIS, PhA, SoA). Although comprehensive, the CTSQ is not very practical for use in experimental research as administration is time-consuming and highly repetitive. A factor analysis of the CSTQ revealed four factors: "Unusual Experiences", "Cognitive Disorganisation", "Introvertive Anhedonia", and "Impulsive Nonconformity" (Bentall et al., 1989). New scales were developed using items which most accurately measured these four factors and as a result of this process, the Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE; O. Mason, Claridge, & Jackson, 1995) was developed. The O-LIFE addresses the problems of the CSTQ and provides a comprehensive, normally distributed measure of schizotypy which is suitable for use in the general population. The measure has good internal reliability and a comparable relationship of scores with age and gender to that found in psychosis samples. Despite these strengths, it is still quite a lengthy measure to administer and could be argued to retain the weaknesses of those scales combined in its creation. Nevertheless, the O-LIFE remains a widely used tool in schizotypy research (O. Mason & Claridge, 2006).

The Schizophrenism and Anhedonia Scale (Venables et al., 1990) is a further measure created by combining items from existing self-report schizotypy assessment tools. Developed in a non-clinical sample, the scale was designed to assess both positive (i.e. schizophrenism – cognitive-perceptual and attentional dysfunction) and negative (i.e. anhedonia – social dysfunction and anhedonia) aspects of schizotypy. Validation of the scale involved investigating the responses of high scorers on a variety of experimental tasks theoretically related to psychosis. These included reaction time measurement and skin conductance orienting (e.g. Hazlett, Dawson, Filion, Schell, & Nuechterlein, 1997). The results showed that associations between scores on the scale and performance on the tasks were in the same direction as those highlighted in a schizophrenic sample, and thus the validity of the scale was confirmed (Venables et al., 1990). Despite being fairly inclusive, the Schizophrenism and Anhedonia Scales do not explicitly cover items

relating to social anxiety, but rather social anhedonia. This is arguably problematic given the increasing evidence for the role of social anxiety in psychosis (Birchwood, 2003).

Summary. This section has discussed measures designed to assess schizotypy as a fully dimensional personality trait. These measures specifically assess psychosis-like personality traits, thus separating them from broader personality inventories. However, conceptualising schizotypy as a personality trait only allows an individual's underlying predisposition to psychosis to be assessed, and not their current risk of transition. More specifically, it can be assumed that all individuals at high-risk of psychosis would achieve similarly high scores on a schizotypal trait measure, and thus would appear as a homogenous group (Claridge & Beech, 1995). Heterogeneity in current experiences would not be detected by a trait measure alone.

As they are not based on diagnostic criteria, fully dimensional schizotypy measures support the continuum hypothesis and thus the notion that some individuals experience psychotic-like symptoms without associated distress (V. Bell et al., 2006). However, not being based on diagnostic criteria also reduces the clinical relevance of the scale. The aim of this thesis is to utilise a measure of schizotypy in the assessment of the prodromal and residual symptoms of psychosis. As such, items included in the measure should ideally bear some resemblance to clinically-defined psychotic symptoms and this is questionable for the above schizotypy assessment tools. The following section will review schizotypy measures which utilise diagnostic criteria in their construction, and conceptualise schizotypal phenomena as attenuated psychotic symptoms.

2.2.3.2 Measures assessing schizotypy as attenuated psychotic symptoms

These scales adopt a similar philosophy to measures based on diagnostic criteria (i.e. that symptoms represent a deviation from "normal" behaviour or experiences), but at the same time are grounded in the assumption that attenuated psychotic symptoms are distributed throughout the general population, only becoming a clinical problem when occurring above a certain level of severity and/or resulting in distress. Therefore, whilst not necessarily pathological, the presence of such symptoms may denote someone as being at-risk of developing a diagnosable disorder at a later stage. Many measures in

this domain assess single symptoms (e.g. hallucinations, delusional ideation) although others assess a range of symptoms. A summary of these measures is provided in Tables 2.4, 2.5, and 2.6.

Hallucinations. The Structured Interview for Assessing Perceptual Anomalies (SIAPA; Bunney et al., 1999) is an interview-based assessment tool designed to measure the frequency of sensory anomalies, or low-level hallucinations, across the five senses. Rather than assessing hallucinatory phenomena directly, the SIAPA focuses on changes in sensory intensity, attention and flooding. However, as a structured interview it is fairly resource-intensive to administer. The Launay-Slade Hallucination Scale (LSHS; Launay & Slade, 1981) is a self-report measure of hallucinatory predisposition and as such is easier to administer than the SIAPA. The 12-item questionnaire includes a combination of overtly pathological/clinical items as well as other items which represent a subclinical form of hallucinatory experience (e.g. intrusive thoughts and daydreams). The scale has been shown to adequately discriminate between clinical and non-clinical populations and also has validity in assessing hallucinatory experiences in non-clinical populations (Feelgood & Rantzen, 1994; Morrison et al., 2000).

A further scale assessing hallucinatory predisposition is the Cardiff Anomalous Perceptions Scale (CAPS; V. Bell et al., 2006). The CAPS is a 32-item self-report scale assessing lifetime frequency of anomalous experiences as well as associated distress and intrusiveness. Items included in the scale were taken from a variety of other measures, including those related to psychosis proneness, temporal lobe disturbance, and the clinical assessment of psychosis. Although designed to assess attenuated clinical symptoms, the scale uses neutral language and does not assume that people perceive their experiences as "unusual". The CAPS has been shown by the authors to adequately discriminate between clinical and non-clinical populations, and also has good test-retest and internal reliability.

Delusions. Measures devised to assess low-level delusional ideation include the Peters Delusion Inventory (PDI; Peters, Joseph, & Garety, 1999), a 40-item self-report assessment tool of delusional ideation for use in the general population. The PDI assesses lifetime prevalence of beliefs, interpretations and experiences using a 5-point

Likert scale to assess conviction, distress and frequency. Scale items are derived from the Present State Examination (PSE; Wing et al., 1974) and thus have a clinical basis. The scale has been shown by the authors to have good internal consistency and concurrent validity. Furthermore, it has been widely used in a range of studies assessing the psychosis continuum (e.g. Peters, Day, McKenna, & Orbach, 1999).

Whereas the PDI measures a range of delusional ideation, other measures have been developed to assess specific types of delusional thought. These measures have mostly focused on paranoia, and include the Paranoia Scale (Fenigstein & Vanable, 1992), a 20-item self-report scale based on items from the MMPI. Items refer mostly to public self-consciousness as opposed to persecution and are thus not overtly clinical in nature. However, the scale has been successfully applied to a schizophrenic population (Smári, Stefánsson, & Thorgilsson, 1994). Rawlings and Freeman (1996) developed a further measure to assess paranoia and suspiciousness in the general population. The Paranoia/Suspiciousness Questionnaire (PSQ) consists of questions modified from several established scales assessing paranoia and related concepts. The scale was shown by the authors to have good internal and test-retest reliability. However, due to its development in a non-clinical sample, the use of the PSQ in a psychiatric setting is somewhat questionable.

It has been argued that scales assessing paranoia do not contain adequate reference to persecutory phenomena and this prompted the development of the Paranoia Checklist (Freeman, Garety et al., 2005). The Paranoia Checklist was devised to investigate paranoid thoughts of a more clinical and distressing nature than those assessed by the Paranoia Scale. It provides a multidimensional assessment of paranoid ideation, with each of the 18 items being rated on a 5-point scale for frequency, degree of conviction, and distress. It has been shown by the authors to display good internal and convergent reliability, although it has not been validated in a clinical sample.

Measures assessing a range of symptoms. In terms of the wider concept of schizotypy and psychosis proneness, the measures discussed above each assess one domain of experience. This allows specific and detailed investigations into different types of symptoms but does not allow the co-occurrence of multiple symptom types to be examined. Measures which are more inclusive in their approach include the

Schizotypal Traits Questionnaire (STQ; Claridge & Broks, 1984); the King's Schizotypy Questionnaire (KSQ; L. A. Jones et al., 2000); the Scales for Rating Psychotic and Psychotic-like Experiences as Continua (L. J. Chapman & Chapman, 1980); and the Community Assessment of Psychic Experiences (CAPE; Stefanis et al., 2002). Items included in these measures are based on attenuated versions of symptoms outlined by diagnostic criteria for psychosis and SPD. Other measures in this group, such as the Schizophrenism Scale (Nielsen & Petersen, 1976), are based on attributes outlined by studies investigating early stage schizophrenia (e.g. J. P. Chapman, 1966) and thus tap aspects which are phenomenologically related to schizophrenia. The Rust Inventory of Schizotypal Cognitions (RISC; Rust, 1987; 1988) assesses a range of bizarre and eccentric thought patterns which may be indicative of increased risk of developing psychosis. Indeed, Miller, Lawrie, Byrne, Cosway, and Johnstone (2002) have suggested that high scores on the RISC are strongly associated with the presence of psychotic symptoms.

The only scale to assess all nine aspects of SPD, and thus arguably all of the attenuated symptoms of psychosis, is the Schizotypal Personality Questionnaire (SPQ; Raine, 1991). Although the SPQ is based on diagnostic criteria for SPD, it is not specific to diagnosing personality disorder and can also be used to assess attenuated symptoms of psychosis. The SPQ is a 74-item questionnaire with a dichotomous (yes/no) response format. It was developed using DSM-III-R criteria (American Psychiatric Association, 1987) and has been shown by the author to be an appropriately valid and reliable scale for use in both clinical and non-clinical populations for the identification of individuals with schizotypal traits. A factor analytic study conducted on the SPQ highlighted a three-factor structure encompassing "Cognitive-perceptual", "Interpersonal", and "Disorganised" schizotypy (Raine et al., 1994). This finding corresponds with other studies which have detected analogous factor structures in both schizotypal and psychotic phenomena (Bentall et al., 1989; Strauss et al., 1974). Moreover, findings from a study in which the SPQ was completed by first-episode schizophrenia patients and their first-degree relatives, suggest that the positive dimension of the questionnaire can be used to reflect the presence of a genetic vulnerability to schizophrenia (Vollema, Sitskoorn, Appels, & Kahn, 2002). The SPQ has also been used in numerous other studies examining the correlates of schizotypy (e.g. Dinn, Harris, Aycicegi, Greene, & Andover, 2002; Skosnik, Spatz-Glenn, & Park, 2001).

Summary. This section has discussed measures designed to assess schizotypal phenomena as attenuated symptoms of psychosis. Some of these measures assess individual symptoms (i.e. hallucinations or delusions), whereas others assess a range of psychotic-like phenomena simultaneously. A definite strength of these measures is that they are either based on diagnostic criteria for psychosis; or on concepts which are theoretically related to the disorder. As such, these measures view schizotypal experiences as analogous to (and on a continuum with) low-level psychotic experiences. Use of these measures would be advantageous in assessing the prodromal and residual symptoms of psychosis, which may not be detected by traditional psychiatric assessment tools. Despite these strengths, the majority of measures in this domain have been developed for use in non-clinical populations and are not suitable for use in clinical samples. The next section considers measures used to assess low-level psychotic symptoms in clinical populations.

2.2.4 Measures Designed to Assess Prodromal Psychotic Symptoms

Prodromal symptoms occur prior to the onset of clinically diagnosable psychosis (H. J. Jackson, McGorry, & Dudgeon, 1995). Measures designed to assess clinically-defined prodromal symptoms are relatively new in their development. Like other psychiatric assessment tools, they assess deviations from "normal" behaviour, but also take into account duration, severity and frequency of symptoms, as well any decline in general functioning.

Measures designed to assess prodromal symptoms of psychosis are broadly based on one of two approaches. The first is the Attenuated Positive Symptoms (APS) which is thought to characterise a late prodromal phase. APS measures include the Comprehensive Assessment of At-Risk Mental States (CAARMS; Yung et al., 2002) and the Structured Interview of Prodromal Syndromes (SIPS; McGlashan et al., 2003). Both the CAARMS and the SIPS are interviews which follow specific criteria in order to potentially define an individual as being at-risk of developing psychosis. The second approach to assessing prodromal symptoms is the Basic Symptoms (BS) approach. This is based on a detailed phenomenological way of describing disturbances occurring prior to the onset of psychosis, including low-level changes in perception, cognition,

language and motor function. The BS approach is thought to characterise an early prodromal phase. BS measures include the Bonn Scale for the Assessment of Basic Symptoms (BSABS; Gross, Huber, & Klosterkötter, 1987) and the Schizophrenia Prediction Instrument – Adult version (SPI-A; Klösterkotter, Schultze-Lutter, Wieneke, Picker, & Steinmeyer, 2001). Both of these scales are clinician-rated interviews designed to establish the presence or absence of basic symptoms. Other measures adopting a combination of the APS and BS approaches also exist as do numerous checklists for subthreshold psychotic symptoms (e.g. Heinimaa et al., 2003; Loewy, Bearden, Johnson, Raine, & Cannon, 2005). A more detailed description of each measure is summarised in Tables 2.7, 2.8, and 2.9.

Although APS and BS measures possess positive predictive value in determining the onset of psychotic disorder, and in highlighting those individuals who may be at-risk; there is wide variability in the approaches utilised. Some measures assess the presence or absence of particular phenomena (e.g. CAARMS, SIPS) whereas others investigate transformations in the structure or form of experience (e.g. EASE). In a recent review, Olsen and Rosenbaum (2006) outline the need for an investigation into the convergence and divergence of these various approaches. Many of the measures require clinician-administered interviews, making their administration time consuming and resource-heavy. Furthermore, the validity of many prodromal instruments is still under investigation and those which have been validated are mostly limited to clinical populations, resulting in difficulties in administering the measures to the general population. More research is required in order to isolate those symptoms which are particularly predictive of psychosis and which can be measured in both clinical and non-clinical populations. Moreover, these measures have been specifically designed to assess the prodromal symptoms of psychosis. As such, their ability to adequately measure residual psychotic phenomena is unknown.

Table 2.1 *Measures Designed to Assess Borderline Conditions*

Measure	Description	How items created	Reliability and Validity
Diagnostic Interview for Borderline (DIB; Kolb & Gunderson, 1980)	Semistructured interview schedule for assessing borderline patients. Assesses all borderline conditions – borderline schizophrenia and borderline personality	Authors abstracted major characteristics of borderline patients from interviews	Adequate inter-rater reliability reported by authors. Not a specific measure of schizotypal personality but rather all borderline personality disorders.
The Symptom Schedule for the Diagnosis of Borderline Schizophrenia (Khouri et al., 1980)	Structured interview containing eight items relating to perceptual, thought and behavioural changes	Based on schizotypal-like features found among relatives of schizophrenics in Danish adoption study (Kety et al., 1974)	Reported by authors to be a reliable measure of schizotypy
The Schedule for Schizotypal Personalities (SSP; M. Baron et al., 1981)	Structured interview designed to improve diagnostic reliability for borderline syndromes related specifically to schizophrenia. 10 scales: Illusions, Depersonalisation, Ideas of Reference, Suspiciousness, Magical Thinking, Inadequate Rapport, Odd Communication, Social Isolation, Social Anxiety, and Delusions/hallucinations, each consisting of 1-8 items rated on a 4-point scale of severity.	Based on DSM-III item set for SPD. Specific questions were derived from a review of the literature and a survey of clinical vignettes relevant to the concept of SPD.	In a reliability study conducted by the authors, diagnostic agreement on the presence or absence of SPD was found in 49 of 53 cases.
The Structured Interview for Schizotypy (SIS; Kendler et al., 1989)	Structured interview assessment with two parts: 1.Use of structured probes to elicit self-report information on symptoms such as magical ideation, social isolation, etc 2. Observation of behaviour and interviewer judgements on rapport, attention, etc	Items developed from a large family study of schizophrenia conducted by the authors	The SIS has been shown by three independent pilot studies to discriminate between relatives of individuals with schizophrenia and relatives of controls (Tsuang et al., 2002).

Note: SPD = Schizotypal Personality Disorder

Table 2.2 *Measures Designed to Assess Schizotypy as a Fully Dimensional Personality Trait – Individual Schizotypal Attributes*

Measure	Description	How items created	Reliability and Validity
Perceptual Aberration Scale (PER; L. J. Chapman et al., 1978)	35-item scale measuring cognitive perceptual psychotic-like experiences (i.e. bodily discontinuities and unusual sensory experiences) in the general population, e.g. "I have felt that something outside my body was a part of my body".	Items are based on experiences of somatic distortions and hallucinations, as reported in the clinical literature on schizophrenia and associated diagnoses.	Good internal consistency ($\alpha = .85$) College students scoring high on this measure exceeded control subjects on psychotic-like experiences (L. J. Chapman & Chapman, 1980) No association with scores on physical and social anhedonia scales – discriminant validity.
Magical Ideation Scale (MIS; Eckblad & Chapman, 1983)	30-item scale designed to measure unconventional beliefs (i.e. low-level delusions), such as thought transmission, psychokinetic effects, precognition and the transfer of psychic energies between people.	Covers a range of beliefs and experiences from first-rank symptoms of schizophrenia and ideas of reference to popular paranormal and conspiracy theory themes.	Good internal consistency ($\alpha = .80$). Good convergent validity: correlation of .70 with PER. Subjects with high scores on the MIS showed more psychotic like and schizotypal symptoms than control subjects (L. J. Chapman & Chapman, 1980).
Physical and Social Anhedonia Scales (PhA & SoA; L. J. Chapman et al., 1976)	PhA scale (40 items) reflects deficit in ability to experience physical pleasure (e.g. eating, touching, etc) SoA scale (48 items) reflects deficit in ability to experience interpersonal pleasure (e.g. being with people, talking, etc). Reflects "negative" aspects of schizotypy	Based on experiences/clinical literature of schizophrenia.	No association with scores on PAS or MIS, supporting different dimensions of positive and negative symptoms. Those with high scores on this measure were more socially withdrawn and had less sexual interest/activity.
Revised Social Anhedonia Scale (Eckblad et al., 1982)	40-item scale reflecting social isolation, social dysfunction and indifference e.g. "A car ride is much more enjoyable if someone is with me" (false)	Based on experiences/clinical literature of schizophrenia.	Good reliability and validity (Mishlove & Chapman, 1985)

Table 2.2 *Contd.*

Measure	Description	How items created	Reliability and Validity
Social Fear Scale (Raulin & Wee, 1984)	True/false scale developed to measure social fear in the general population	Items chosen which were considered to be specific to social fear – suggested by Meehl (1962; 1990) to be characteristic of schizotypy	Follow-up interviews confirmed the measurement of social fear. Osman, Jones, and Osman (1990) reported good internal and test-retest reliability.
Intense Ambivalence Scale (IAS; Raulin, 1984)	45-item true/false scale designed to measure intense ambivalence	Based on clinical literature on schizophrenia and relatives of those with schizophrenia.	Longitudinal study conducted by Kwapil et al. (2000) showed that elevated scores on the IAS predicted the development of psychotic illness at 10-year follow-up.
Schizotypal Ambivalence Scale (SAS; Kwapil et al., 2002)	19-item revision of the Intense Ambivalence Scale	Items taken from Intense Ambivalence Scale (Raulin, 1984)	Good internal consistency ($\alpha = .84$). Correlates moderately with other psychometric indices of schizotypy. High SAS scores associated with schizotypal, schizoid and paranoid symptoms, and poor functioning.
Cognitive Slippage Scale (Miers & Raulin, 1985)	True/false scale for use with the general population to assess subtle thought disorder.	Items taken from clinical literature on schizophrenia and thought disorder. Cognitive slippage outlined by Meehl (1962) as one of the four characteristics of schizotypy.	Study by Gooding et al. (2001) showed that high scorers on this scale had reduced cognitive performance – evidence of construct validity.
Impulsive Non-Conformity Scale (ImpNon; L. J. Chapman et al., 1984)	Scale constructed to measure impulsive non-conformity and anti-social behaviour.	Taken from examples of anti-social behaviour reported in the premorbid adjustment phase of psychosis	Correlates highly with Psychoticism scale ($r = .68$) suggesting convergent validity
Hypomanic Personality Scale (HypM; Eckblad & Chapman, 1986)	48-item true/false scale assessing impulsive and manic behaviour, e.g. "I often get so happy and energetic that I am almost giddy".	Items more representative of bipolar disorder or affective psychoses	Reported by authors: Good internal consistency ($\alpha = .87$) Good test-retest reliability ($r = .77$)

Table 2.3 *Measures Designed to Assess Schizotypy as a Fully Dimensional Personality Trait – Multidimensional Schizotypy Measures*

Measure	Description	How items created	Reliability and Validity
Combined Schizotypal Traits Questionnaire (CSTQ; Bentall et al., 1989)	Combination of a large body of scales to measure schizotypy in the general population – contains 420 items and true/false response format	Taken from STQ, Chapman scales, LSHS, Schizophrenism scale, Schizoidia scale, etc. Aim to capture multidimensionality of schizotypy	Psychometrics of 14 scales included in the CSTQ are well-documented by the author and viewed to be satisfactory (Claridge, 1997a).
Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE; O. Mason et al., 1995)	Made up of four scales each containing 24-30 items: Unusual Experiences (positive symptoms), Cognitive Disorganisation (attention, concentration and decision making), Introvertive Anhedonia (lack of enjoyment from social sources, dislike of intimacy), and Impulsive Non-conformity (violent, self-abuse) Yes/No response format	Reduction of CSTQ via factor analysis of scales. Items selected on the basis of factor loading size, endorsement rate and avoidance of repetition (unless question appeared highly relevant).	All scales have highly adequate internal consistency (α = .77-.89). Relationships with age and sex mirror those found in psychotic samples with psychotic symptoms.
Schizophrenism and Anhedonia Scales (Venables et al., 1990)	27-item yes/no scale developed for the measurement of the positive – (cognitive/perceptual/attentional function) and negative (social dysfunction and ahedonia) aspects of schizotypy 14 items measuring schizophrenism (SZ) 13 items measuring anhedonia (AH)	Initial questionnaire included 250 items taken from other scales – selections were made based on theory. Items were also chosen for having normal face value, i.e. non-pathologising	Good construct validity – has been shown by authors to be related to other measures of schizotypy and diagnoses of SPD

Table 2.4 *Measures Designed to Assess Schizotypy as Attenuated Psychotic Symptoms – Hallucinations*

Measure	Description	How items created	Reliability and Validity
The Structured Interview for Assessing Perceptual Anomalies (SIAPA; Bunney et al., 1999)	Interview-based assessment method designed to assess the frequency of sensory anomalies occurring across all five senses	Items assess perceptual anomalies as distinct from hallucinations but are based on schizophrenic experience.	Good inter-rater reliability demonstrated by the authors. Individuals with schizophrenia scored higher than controls across all five modalities.
Launay-Slade Hallucination Scale (LSHS; Bentall & Slade, 1985; Launay & Slade, 1981)	12-item forced choice (yes/no) questionnaire to measure hallucinatory disposition (includes combination of overt pathological items and other items which appear to represent a subclinical form of hallucinatory experience). Bentall and Slade added Likert scale for use in the general population.	30-item questionnaire initially constructed containing 7 clinical items, 20 subclinical items (vivid/intrusive thoughts, dreams, daydreams) and 3 filler items. This was reduced by removing items which failed to discriminate between clinical and non-clinical populations.	Discriminates between hallucinators and non-hallucinators in both patient and general population samples. Good test-retest reliability in non-clinical sample (r = .84).
Cardiff Anomalous Perceptions Scale (CAPS; V. Bell et al., 2006)	32-item self-report questionnaire using dimensional subscales to assess distress, intrusiveness, and frequency of anomalous experiences Initial yes/no response (lifetime) and then 5-point Likert scale to measure distress, intrusiveness and frequency. Aim was to construct a scale that would be selective for perceptual anomalies without being conceptually tied to the assumptions and language of previous clinical and psychometric scales.	Items were taken from measures related to psychosis proneness, the clinical assessment of psychosis, delusional and magical ideation, and hallucinatory experience. Particular focus on anomalous perceptual experience rather than more general aspects of schizotypy.	Completed by a clinical and non-clinical sample. 11.3% of the non-clinical sample scored above the mean of the clinical sample. Internal consistency good (α = .87) and test-retest reliability acceptable (r = .77). Internal consistency also stayed stable over time (α = .92). Convergent validity with LSHS, O-LIFE unusual experiences subscale and PDI-21. Small or non-significant correlations with other subscales of O-LIFE. Factor analysis revealed a three factor structure (temporal lobe, chemosensation, and clinical psychosis)

Table 2.5 *Measures Designed to Assess Schizotypy as Attenuated Psychotic Symptoms – Delusions*

Measure	Description	How items created	Reliability and Validity
Peters et al Delusion Inventory (PDI; Peters, Joseph et al., 1999)	40-item measure of delusional ideation which asks about individuals' beliefs, interpretations and experiences. Measures the total number of beliefs or experiences endorsed, but also the concurrent perceptions of distress, preoccupation and conviction. Measures lifetime prevalence, has initial yes/no response followed by Likert scale	Items derived from Present State Examination (Wing et al., 1974)	Good internal consistency and concurrent validity was confirmed with three scales measuring schizotypy, magical ideation and delusions. PDI scores up to 1 year later remained consistent Psychotic inpatients had significantly higher scores than controls
Paranoia Scale (Fenigstein & Vanable, 1992)	20-item self-report scale developed to measure paranoia in college students. Each item is rated on a 5-point scale from 1 (*not at all applicable*) to 5 (e*xtremely applicable*)	Items specific to paranoid ideation, e.g. "I sometimes feel as if I am being followed"	Good test-retest reliability over six months (r = .70). Good internal consistency (α = .80)
Paranoia/Suspiciousness Questionnaire (PSQ; Rawlings & Freeman, 1996)	47-item scale developed to measure paranoia and suspiciousness in a non-psychiatric sample Yes/No response format	Developed on a non-psychiatric sample using modified items from several established scales of paranoia and related concepts.	Factor analysis revealed five moderately correlated subscales. Full questionnaire and the subscales showed satisfactory internal consistency and test-retest reliability.
Paranoia Checklist (Freeman, Garety et al., 2005)	Devised to investigate paranoid thoughts of a more clinical nature than those assessed in the Paranoia Scale and to provide a multi-dimensional assessment of paranoid ideation. 18-item self report scale with each item rated on a five-point scale for frequency, degree of conviction, and distress.	Addition of more persecutory items to Fenigstein and Vanable's (1992) paranoia scale. Based on literature and assessments of psychotic symptoms.	Convergent validity with Paranoia Scale and good internal consistency (α = .90)

Table 2.6 *Measures Designed to Assess Schizotypy as Attenuated Psychotic Symptoms – Measures Assessing a Range of Symptoms*

Measure	Description	How items created	Reliability and Validity
The Schizotypal Traits Questionnaire (STQ; Claridge & Broks, 1984)	Consists of two scales STA – a 37-item scale assessing schizotypal personality STB – an 18-item scale assessing borderline personality Designed to specifically tap the DSM-III concepts of SPD and BPD	Items devised to cover cognitive, attentional and perceptual disturbances found in the self-reports of schizophrenic patients	STA has minimal correlation with Psychoticism. STB is significantly correlated with Psychoticism. Good reliability and validity reported by the authors.
The King's Schizotypy Questionnaire (KSQ; Williams, 1993) Unpublished dissertation – KSQ reported in paper by Jones et al. (2000)	63-item forced choice (yes/no) self-report measure of schizotypy	Items linked with DSM-III criteria for SPD with many items based on verbatim descriptions of personal experiences by the relatives of individuals with schizophrenia.	High internal consistency ($\alpha = .81$) Good test-retest reliability ($r = .73$) in a sample of 100 normal subjects Good validity correlation with STA ($r = .62$)
Scales for Rating Psychotic and Psychotic-like Experiences as Continua (L. J. Chapman & Chapman, 1980)	Modification of the Schedule for Affective Disorders and Schizophrenia-Lifetime version.(SADS-L; Endicott & Spitzer, 1978). Scores assigned between 1 and 11 on judgements of increasing deviancy.	Modification consisted of attenuating item content of SADS-L to reflect lower-level phenomena.	High internal consistency ($\alpha = .94$) Subjects who scored highly on magical ideation also scored highly on this scale.
Community Assessment of Psychic Experiences (CAPE; Stefanis et al., 2002)	42-item self-report questionnaire designed to assess positive, negative, and depressive experiences associated with psychosis in non-clinical populations. Items rated on a 4-point scale for frequency and distress. Assesses lifetime presence/general tendencies (i.e. Do you ever…)	Positive items derived from the PDI; negative/depressive items derived from the Scale for the Assessment of Negative Symptoms (SANS; Andreasen, 1981) and Subjective Experience of Negative Symptoms scale (SENS; Selten, Gernaat, Nolen, Wiersma, & van den Bosch, 1998)	All dimensions shown by authors to be stable, reliable, and valid in a large general population sample. Cross-validated with interview measures of schizotypy (Konings, Bak, Hanssen, van Os, & Krabbendam, 2006).

Note: SPD = Schizotypal Personality Disorder; BPD = Borderline Personality Disorder

Table 2.6 *Contd.*

Measure	Description	How items created	Reliability and Validity
Schizophrenism Scale (Nielsen & Petersen, 1976)	Taps cognitive/perceptual aspects of behaviour	Pool of items drawn from study reporting phenomenology of early stage schizophrenia (J. P. Chapman, 1966)	Good reliability and validity reported by authors. High scorers performed worse on neuropsychological tasks (Asarnow, Nuechterlein, & Marder, 1983) suggesting the scale does tap cognitive aspects.
Rust Inventory of Schizotypal Cognitions (RISC; Rust, 1987; Rust, 1988)	26 statements assessing schizotypal cognitions associated with the positive symptoms of acute schizophrenia. Scale aims to identify bizarre and eccentric thought patterns phenomenologically related to acute schizophrenia.	Based on symptomatology of schizophrenia	Good reliability and validity. Has been shown by authors to discriminate between individuals with acute schizophrenia and controls.
Schizotypal Personality Questionnaire (SPQ; Raine, 1991) Brief SPQ (SPQ-B; Raine & Benishay, 1995)	Designed with aim to develop a questionnaire closely modelled on DSM-III-R criteria that could provide an overall measure of schizotypal personality and attenuated psychotic symptoms. Contains nine subscales to assess each of the nine schizotypal features (ideas of reference, excessive social anxiety, odd beliefs/magical thinking, unusual perceptual experiences, odd/eccentric behaviour, no close friends, odd speech, constricted affect, suspiciousness). 74-item Yes/No response format, assesses lifetime prevalence. Shorter, 22-item instrument based on the SPQ consisting on the most reliable items.	Items generated from existing interview schedules for schizophrenia and schizotypal personality (e.g. PSE, SANS, SCID, SADS) and generation of new items to fill gaps in the item pool.	Good internal consistency ($\alpha = .90$ for total scale and .66-.81 for subscales). High test-retest reliability ($r = .82$). Good convergent validity with STA and schizophrenism scales and divergent validity with scales not tapping DSM-III-R schizotypal features. Kremen, Farone, Toomey, Seidman, and Tsuang (1998) showed that relatives of individuals with schizophrenia had higher scores on the cognitive perceptual factor of the SPQ than controls. Factor analysis of the scale resulted in a three-factor solution (Raine et al., 1994) which has been replicated (cognitive-perceptual, interpersonal, and disorganised).

Table 2.7 *Measures Designed to Assess Prodromal Symptoms – Attenuated Symptoms Measures*

Measure	Description	How items created	Reliability and Validity
Comprehensive Assessment of At-Risk Mental States (CAARMS; Yung et al., 2002)	Semistructured interview to diagnose at-risk mental state. Seven subscales (positive symptoms, cognitive change, emotional disturbance, negative symptoms, behavioural change, motor physical change, general psychopathology). Dimensions of intensity, frequency/duration, and fluctuation of symptoms are scored separately. A person is defined as being at-risk mental state when meeting one or more of the Ultra-High Risk (UHR) criteria based on positive symptoms of CAARMS (Yung et al., 2002).	Designed in conjunction with ultra-high risk criteria	A 6-month follow-up of 150 non-psychotic, help-seeking individuals demonstrated that meeting the CAARMS-defined UHR criteria significantly predicted psychosis (Yung et al., 2002). Good discriminant validity – 48 controls scored significantly lower than 49 individuals in a BPRS defined UHR state
Structured Interview for Prodromal Symptoms (SIPS) and Scale of Prodromal Symptoms (SOPS) (T. J. Miller et al., 1999)	Clinician-administered semistructured interview. The SIPS determines presence or absence of prodromal state and SOPS determines the severity. Consists of 5 positive symptom items, 6 negative symptom items, 4 disorganisation symptom items, and 4 general symptom items. Each item has a severity rating scale from 0 (*never/absent*) to 6 (*severe/extreme*). Interview contains probes for positive items, GAF, family interview and SPD checklist.	Based on PANSS (Kay, 1991) with modification of the positive symptom scales to provide more breadth of scoring within the lower, prepsychotic ranges of severity. Designed to rate the existence and severity of prodromal symptoms along dimensions between normalcy and lower levels of pathology as defined by conventional rating scales (e.g. BPRS, PANSS, SAPS, SANS, etc)	Factor structure shown to be similar to that of symptoms of schizophrenia (Hawkins et al., 2004), suggesting evidence for a psychosis continuum. Shown by authors to have good predictive validity.

Note: BPRS = Brief Psychiatric Rating Scale; SAPS = Scale for the Assessment of Positive Symptoms; SANS = Scale for the Assessment of Negative Symptoms

Table 2.8 *Measures Designed to Assess Prodromal Symptoms – Basic Symptoms Measures*

Measure	Description	How items created	Reliability and Validity
Bonn Scale for the Assessment of Basic Symptoms (BSABS; Gross et al., 1987)	Scale consists of six subscales of basic symptoms (two scales of dynamic deficits, cognitive disturbances, coenesthetic experiences, central vegetative experiences, and autoprotective behaviour). Each basic symptom is rated as either present or absent.	Created from research conducted by the authors into the basic symptoms of schizophrenia. These are subtle, subjectively experienced disturbances in the domains of perception, cognition, language, motor function, will, initiative, and level of energy and stress tolerance.	Specific disturbances of cognition, speech and perception have shown a significant predictive value in developing schizophrenia. Ten symptoms predicted schizophrenia with a probability of 71-91% (Klosterkötter, Hellmich, Steinmeyer, & Schultze-Lutter, 2001).
Schizophrenia Prediction Instrument – Adult version (SPI-A; Klosterkötter, Schultze-Lutter et al., 2001)	Shorter 40-item version of the Bonn Scale to assess basic symptoms.	Developed as a supplement to SIPS/CAARMS and PANSS.	Shown by authors to be predictive of later psychosis
Early Recognition Inventory (ERIraos; Häfner et al., 2004; Maurer, Hörrmann, Schmidt, Trendler, & Häfner, 2004)	Two-step inventory. 1. 17-item checklist, constructed as a screening instrument for application in primary care settings. 2. 110-item symptom list for incipient schizophrenia.	110-item symptom list compiled from other measures, including: Interview for the Retrospective Assessment of the Onset and Course of Schizophrenia and other Psychosis (IRAOS), BSABS, SIPS, CAARMS	Successfully used in a study to screen for individuals in prodromal states (Bechdolf et al., 2005)
Examination of Anomalous Experience (EASE; Parnas et al., 2005)	Semistructured symptom checklist, exploring phenomena regarded as important in the preonset phase which is also manifest in schizophrenia and schizotypy Interview consists of 57 items divided into five subscales (cognition and stream of consciousness, disorders of self-awareness and presence, bodily experience, transitivism, and existential re-orientation). Symptoms are rated in terms of frequency/severity and as to specific patterns	Items focus on experiential anomalies of self-awareness Many items overlap with BSABS but only items relating to self-experience are included. Other items are based on the authors own experience and phenomenological orientation.	Authors demonstrated good inter-rater reliability on the basis of video interviews.

Table 2.9 *Measures Designed to Assess Prodromal Symptoms – Screening Tools*

Measure	Description	How items created	Reliability and Validity
PRODscreen – a screen for prodromal symptoms of psychosis (Heinimaa et al., 2003)	Screen to detect elevated risk of psychosis. Interview or self-rating. Includes: 7 items for general functioning, 10 items for general symptoms and 12 items of more specific psychosis-like character. The main focus of the measure is attenuated positive symptoms.	Based on items from SIPS, IRAOS and BSABS	Authors demonstrated that the measure correctly defined a SIPS-defined prodromal state in 77% of cases from a mixed sample. Inclusion of qualitative data has shown to enhance the validity of the screening procedure.
Prodromal Questionnaire (PQ; Loewy et al., 2005)	Contains 92 true/false statements including subscales for positive, negative, disorganised and general symptoms.	Based on SIPS, SPQ and a few original items based on the authors' experience	Good concurrent validity of positive subscale with SIPS. Validated by authors in a large non-clinical population of students.
Youth Psychosis At-Risk Questionnaire (Y-PARQ; Ord & Myles-Worsley, 2004)	Consists of 92 yes/no items for self-rating Includes items on positive, affective and negative symptoms of prodromal schizophrenia	Based on CAARMS	Tested by authors on 648 high school students with elevated rates of familial schizophrenia. Positive predictive value of a CAARMS-defined at risk mental state of 82.4%.
SIPS Screen (T. J. Miller, Cicchetti, Markovich, McGlashan, & Woods, 2004)	Brief self-report screen consisting of 12 items covering positive symptoms only. Each item rated between 0 (*definitely disagree*) and 6 (*definitely agree*)	Covers same items as SIPS	SIPS screen was administered before SIPS interview on a sample of 36 subjects referred for prodromal evaluation. Screening instrument showed a sensitivity of .90 and indicated perfect specificity.
Basel Screening Instrument for Psychosis (BSIP; Gschwandtner et al., 2003; Riecher-Rössler et al., 2006)	46-item screening checklist to identify those at risk, developed for the FEPSY early detection study (Gschwandtner et al., 2003). Used in combination with the BPRS to rate psychotic phenomena.	Based on DSM-III-R prodromal symptoms and other risk factors as derived from the literature (social decline, drug abuse, previous psychiatric disorders and genetic risk)	Higher inter-rater reliability (Kappa = .87) Validated using longitudinal study assessing transition to psychosis. 32% of individuals classified at-risk by BSIP made transition within 2-5 years (Riecher-Rössler et al., 2008).

Note: BPRS = Brief Psychiatric Rating Scale

2.2.5 Summary of Review

This section has reviewed a wide range of measures for use in assessing schizotypy and low-level psychotic phenomena. The review was conducted in the context of the aim of this thesis: using a measure of schizotypal symptoms to assess the prodromal and residual symptoms of psychosis. Some measures are too broad in their scope to adequately address this aim (i.e. borderline personality measures); whereas others assess only specific types of psychotic-like phenomena and as such are too narrow (e.g. PDI, PAS). Moreover, many of the measures reviewed have been validated in non-clinical populations only, making their transferability to clinical groups questionable. Most tools also assess schizotypy as a fully dimensional personality trait; reflecting either lifetime prevalence of schizotypal symptoms, or underlying predisposition to the development of psychosis. As a result, state heterogeneity in psychotic-like experiences is not picked up by these measures. Assessment tools which do assess current low-level psychotic phenomena (i.e. prodromal symptom measures) often require resource-intensive structured interviews and were specifically developed for clinical groups, making them unsuitable for use in non-clinical samples.

As a result of this review, a need has been highlighted for the development of a self-report measure suitable for use in assessing current (i.e. mental state) levels of subclinical psychotic symptoms in both clinical and non-clinical populations. Such a measure could be used to detect both prodromal and residual symptoms of psychosis and also to investigate the associations of these symptoms with a range of other variables. The measure should aim to encompass the strengths of assessment tools described in the preceding review but also address the weaknesses. Therefore its content should be linked to diagnostic criteria, yet at the same time comprise adequate sensitivity for the detection of phenomena analogous to the symptoms of psychosis, but which occur outside of a psychiatric framework (i.e. prior to the onset of psychosis and in the recovery phase). Moreover, as the exact symptoms predictive of the development of psychosis or psychotic relapse are presently unclear (Gunderson, Siever, & Spaulding, 1983; Widiger, Frances, & Trull, 1987); the measure should cover a range of psychotic-like symptoms, as opposed to focusing on one symptom type in isolation.

The rest of this chapter reports on the development and psychometric properties of the Schizotypal Symptoms Inventory (SSI), a modified version of the Schizotypal Personality Questionnaire (SPQ; Raine, 1991). The SSI aims to assess schizotypal experiences in a more dimensional manner, taking account of the current frequency of symptoms, rather than their lifetime presence or absence. The SPQ was used as the basis for the new instrument because it is based on DSM-III-R criteria (American Psychiatric Association, 1987) and is impressively inclusive in the range of phenomena it assesses. In particular, it includes social anxiety, paranoia, and anomalous experiences dimensions. This means that the distributions of different psychotic-like symptom types can be examined using the same scale. The SPQ is also suitable for use in both clinical and non-clinical populations. The modifications made to the SPQ in this study allow schizotypal experiences to be assessed in a state- as opposed to trait-like manner, thus aligning schizotypal experiences more closely with clinical measures of symptomatology.

The distribution of psychotic-like experiences has been shown to be highly dependent on the instrument used to assess them (Johns & van Os, 2001). The SPQ has been shown to have an approximately normal distribution, suggesting that it takes a normalised approach towards the assessment of schizotypy (i.e. it measures normal variation in personality). This fits with the original design aim of the SPQ to assess schizotypal traits and tendencies, rather than attenuated psychotic symptoms (Raine, 1991); and also with findings that schizotypy measured by the SPQ may be an endophenotype of psychosis; i.e. a quantitative trait reflecting genetic liability to the disorder (Avramopoulos et al., 2002; Vollema et al., 2002). Conversely, measures which assess schizotypy as a more pathological construct, such as the Peters Delusion Inventory (PDI; Peters, Joseph et al., 1999), have been shown to have a skewed distribution, mirroring that of other psychopathologies, e.g. affective symptoms (Melzer, Tom, Brugha, Fryers, & Meltzer, 2002).

A state measure of schizotypal symptoms, such as the SSI, is arguably more clinical in its approach than a trait measure, such as the SPQ. In measuring state quasi-psychotic experiences, the SSI is likely to provide a more accurate assessment of current symptom profiles, and thus immediate risk of psychosis. Moreover, the SSI assesses recent frequency of schizotypal symptoms as well as their presence. This is important based on

suggestions from previous research that the experience of psychotic-like symptoms per se is not necessarily pathological, and that other dimensions of experience may be more predictive of risk (Lincoln, 2007). Increased frequency of experience is highlighted as a particular indicator of pathology, as it may lead to heightened emotional responses and increased distress (Birchwood, 1996). Given its more clinical focus, symptoms measured by the SSI may follow a more skewed distribution that that of schizotypal traits measured by the SPQ, which is normally distributed. This will be investigated in the current study.

2.3 STUDY AIMS AND RESEARCH QUESTIONS

The main aim of this study is to report the psychometric properties of the SSI, in both a clinical and non-clinical sample. In both groups, internal consistency, test-retest reliability and the underlying factor structure of the scale are reported. Furthermore, in order to investigate convergent validity of the modified scale, associations between the subscales of the SSI and assessments of psychotic symptoms are provided for the clinical sample. Moreover, to test the discriminative abilities of the SSI, schizotypy scores are compared in the clinical and non-clinical groups. It is hypothesised that the clinical sample will experience more schizotypal phenomena (i.e. have higher SSI scores), than the non-clinical sample; and that within the clinical sample, higher PANSS Positive scores will be associated with higher scores on the SSI.

Following on from these analyses, the most reliable items are taken from the SSI and used to create a shortened 20-item version of the measure. Psychometric analyses of the brief SSI are reported for the clinical and non-clinical samples. It is argued that a short version of the SSI may be useful for longitudinal research to assess changes in symptoms over time without asking individuals to repeatedly complete the full 74-item version.

Finally, in order to examine the ability of the scale to assess current low-level psychotic symptoms, rather than schizotypal traits and tendencies, distributions of item counts on the original SPQ and the new SSI are plotted using histograms and dot plots. The shape of these distributions is then examined and compared. It is predicted that schizotypal symptoms measured by the SSI will follow a skewed distribution (i.e. many people will

experience a few symptoms some of the time but only a few people will experience many symptoms a lot of the time). In contrast, it is hypothesised that the lifetime presence or absence assessment of schizotypy (as measured by the original SPQ questions) will be normally distributed.

2.4 METHODOLOGY

This section will provide a detailed description of the methodology used in this study.

2.4.1 Design

A non-clinical sample of university students and a clinical sample of patients diagnosed with early psychosis were surveyed in a cross-sectional design. A repeated measures design was used with a smaller subsample of participants to examine the test-retest reliability of the SSI. A between-subjects design was used to compare scores in the clinical and non-clinical populations. Within the clinical sample, a correlational design was used to investigate the hypothesised relationships between scores on the SSI and psychopathology measured by the PANSS.

2.4.2 Participants

2.4.2.1 Non-clinical sample

One thousand and one students were recruited for an anonymous internet survey from the University of East Anglia, Norwich; and King's College, London. A student sample was approached as their age group was thought to match that of an early psychosis sample and the typical age range at which individuals are thought to be most at risk of developing psychosis (Hodges et al., 1989). Convenience sampling, via a circular e-mail inviting individuals to participate, was used to recruit individuals into the study. A copy of the circular e-mail is included in Appendix A. The circular e-mail was distributed to a population of over 10,000 students and thus a conservative estimate of the response rate is approximately 10%.

There were no inclusion or exclusion criteria for this sample, other than individuals had to be registered students at either of the participating institutions. In addition to this, participants who incorrectly completed or missed more than 10% of items on the questionnaires (i.e. completion rate less than 90%) were excluded from the analysis. This left a total of 808 participants in the final analysis. Consequently, the percentage of SSI data that was prorated was minimal (0.54%).

2.4.2.2 Clinical sample

One hundred and twenty-six individuals with affective or non-affective psychosis (diagnoses of schizophrenia, schizoaffective disorder and bipolar disorder) also participated in the study. This sample consisted of 58 patients from the Norfolk Early Intervention Service (Norfolk and Waveney Mental Health Partnership); and 68 patients from the first cohort of participants recruited for the "Improving Social Recovery in Early Psychosis" (ISREP) Study – a randomised controlled trial of social recovery oriented cognitive-behavioural therapy (see Appendix B). All participants had been assessed with the Diagnostic Interview for Psychosis (DIP; Jablensky et al., 1999).

All participants had been diagnosed with psychosis in the last eight years but were not yet classified as belonging to a chronic and enduring psychosis population. Furthermore, all patients were not currently experiencing an acute episode of psychosis. Patients recruited from the Norfolk Early Intervention Service were assessed three months after entry into the service, by which time, their episode of psychosis had stabilised. Similarly, in order to take part in the ISREP study, patients must not have been experiencing any positive symptoms above moderate severity as defined by the PANSS.

2.4.2.3 Demographic characteristics of the sample

Demographic characteristics of the two samples are shown in Table 2.10. As can be seen, the mean ages of the clinical and non-clinical samples were similar. However, using an independent samples t-test, the clinical group was shown to be significantly older than the non-clinical group, $t(932) = 3.83$, $p < .001$. There is also a gender bias

towards females in the non-clinical sample and males in the clinical sample. In both samples, ethnicity was predominantly White and British.

Table 2.10

Demographic Characteristics of the Non-clinical and Clinical Samples

	Non-Clinical (*N* = 808)	Clinical (*N* = 126)
Mean Age (*SD*) in years	23.0 (6.6)	25.5 (6.2)
Gender (%):		
Male	192 (23.8)	88 (69.8)
Female	515 (63.7)	38 (30.2)
Did not disclose	101 (12.5)	-
Ethnicity (%):		
White	621 (76.9)	119 (94.4)
Asian	42 (5.2)	1 (0.8)
African	6 (0.7)	1 (0.8)
Afro-Caribbean	2 (0.3)	1 (0.8)
Other	34 (4.2)	4 (3.2)
Did not disclose	103 (12.7)	-
Diagnosis (%):		
Affective Psychosis	-	36 (28.6)
Non-affective Psychosis	-	90 (71.4)

2.4.2.4 Sample size and power analysis

Between-groups analysis. In order to compare differences in SSI scores between clinical and non-clinical populations, a power analysis calculation revealed that to achieve 90% power with a significance level of .05 and an estimated moderate critical effect size of .50, a minimum sample size of 85 participants per group was required (Cohen, 1988).

Correlational analysis. In order to examine the relationship between psychopathology and scores on the SSI within each group, a power analysis calculation revealed that to achieve 90% power with a significance level of .05 and an estimated small to moderate critical effect size of .40, a minimum sample size of 62 participants was required in each group (Cohen, 1988).

2.4.3 Ethical Considerations

The study was reviewed and approved by the ethics committees of all participating institutions, including the Institute of Psychiatry Research Ethics Committee, the University of East Anglia's Faculty of Health Ethics Committee, and Norfolk Local Research Ethics Committee. All letters of approval are located in Appendix C.

2.4.3.1 Informed consent

Non-clinical sample. An explanation of the nature of the research and what participation in the study would involve was included both in the circular e-mail inviting individuals to participate, and on the initial page of the website containing the questionnaires (see Appendix A). Participants were required to tick a box on the web page signifying that they had read and understood the instructions before they could progress to the questionnaires. Informed consent was assumed if participants visited the website and completed the questionnaires. If individuals decided not to take part after reading the instructions, they were free to leave the website. The instructions stated that participants did not have to answer any questions they felt uncomfortable with and could leave the website at any point, even if they had already started to complete the questionnaires.

Clinical sample. In the clinical sample, assessments were conducted as part of routine clinical assessments in the Norfolk Early Intervention Service; or as part of the baseline assessment process in the ISREP study. All patients were asked to sign a consent form stating that they were agreeable to their data being used for research purposes. They were also provided with information regarding the nature of the research and how their responses would be used. Examples of patient information sheets and consent forms are included in Appendix D. Data for any participant who did not wish

their responses to be used for research purposes was not included in the sample. Patients were given as much time as they wanted in order to make the decision as to whether they wanted to be involved in the research. A minimum of 72 hours was allowed between giving information about the research and taking informed consent. Any patient who was considered by a clinician as unable to give informed consent was not approached to take part in the research.

2.4.3.2 Confidentiality and anonymity

Non-clinical sample. No identifying details were recorded about the individuals participating in the internet survey. Circular e-mails were distributed by administrative staff at each institution and thus the researchers did not have access to individual e-mail addresses. Demographic details were requested but participants did not have to supply these if they did not wish to do so.

Following participation in the study, participants were assigned a code so that their responses to each questionnaire could be matched for analysis, whilst remaining anonymous. Following completion of the questionnaires, participant data was stored on the website until the end of the study. The web-page was developed with appropriate security measures by the Information Technology department at the Institute of Psychiatry. This ensured that no persons or organisations outside of the research team were able to gain access to the data collected. Storage of all data also complied with the terms of the Data Protection Act (1998).

Participants were informed at the beginning and end of their participation that if they would like feedback on the research or had any queries regarding their participation, this would be provided on an individual basis via e-mail with the researcher. This happened on several occasions but no problems were highlighted and contacts with the researcher mostly related to personal interest and individuals requesting more information about the study and its aims.

Clinical Sample. Each participant was allocated a code so that their responses to questionnaires and assessments could be matched without using their names. The names of participants were known only to the researcher and were not used

at any other point following the end of the individual's participation in the research. Participants were informed that all of their responses were confidential, unless something arose that resulted in the researcher becoming concerned about the personal safety of the participant or the safety of others. If any concerns did arise, the participant was always informed about who was to be contacted and what was going to happen. In the case of patients recruited from Norfolk Early Intervention Service, the assessments formed part of routine clinical practice and thus information was shared on a clinical basis. Participants were made aware of this at the beginning of their participation in the study.

2.4.4 Measures

2.4.4.1 Schizotypal Personality Questionnaire (SPQ; Raine, 1991)

The SPQ is a 74-item questionnaire with a dichotomous (yes/no) response format, designed to assess schizotypal traits in both clinical and non-clinical populations. The measure is based on DSM-III-R criteria for Schizotypal Personality Disorder (SPD) and is the only published scale reflecting all aspects of the disorder. The SPQ consists of nine subscales, each reflecting individual SPD criteria: Ideas of Reference (9 items), Excessive Social Anxiety (8 items), Magical Ideation (7 items), Unusual Perceptual Experience (9 items), Eccentric Behaviour (7 items), No Close Friends (9 items), Odd Speech (9 items), Constricted Affect (8 items), and Suspiciousness (8 items). The scale has been shown to fit into a three factor structure: Interpersonal Schizotypy, Cognitive-Perceptual Schizotypy, and Disorganised Schizotypy (Raine et al., 1994), mirroring the three types of symptoms seen in psychosis: negative, positive and disorganised (Strauss et al., 1974). Scores on the SPQ range from 0 to 74 with one point being given for every item endorsed.

The SPQ has been shown to have good psychometric properties. The author reports Cronbach's alpha internal consistency coefficients of .90 for the total scale and .71 to .78 (M = .74) for the individual subscales (Raine, 1991). Furthermore, two-month test-retest reliability is reported at .82. In addition to this, the SPQ has been shown to correlate at .81 with the STA subscale of the Schizotypal Traits Questionnaire (STQ; Claridge & Broks, 1984); and from .59 to .65 with the Schizophrenism Scale (Venables

et al., 1990). Both of these scales assess several of the DSM-III-R traits for schizotypal personality. Conversely, low correlations have been found between the SPQ and scales which assess constructs related to psychosis-proneness but which are not included in DSM criteria for schizotypal personality, e.g. .19 with Anhedonia (Venables et al., 1990) and .27 with Psychoticism (S. B. Eysenck et al., 1985). Thus the SPQ is shown to have good convergent and divergent validity. Moreover, 55% of individuals scoring in the top 10% of SPQ total scores have been reported by Raine (1991) to display a clinical diagnosis of SPD, as diagnosed by the SCID. Similar findings have been reported more recently by Kremen et al. (1998).

2.4.4.2 Modifications to the SPQ: creation of the Schizotypal Symptoms Inventory (SSI)

In its current form, the SPQ assesses a combination of lifetime occurrence of psychotic-like experiences (e.g. "Have you ever seen things invisible to other people?"); and the presence of schizotypal traits which may be argued to predispose someone to psychosis (e.g. "Do you often feel that other people have got it in for you?"). As such, the SPQ does not assess recent occurrence of schizotypal phenomena (i.e. mental state). In order to address this, the Schizotypal Symptoms Inventory (SSI) was created by altering items so that they enquired about symptom presence in the previous two weeks, rather than at any point in the respondent's lifetime. In addition, the original dichotomous response format of the SPQ was replaced with a 5-point Likert scale to assess the recent frequency of occurrence of each item. All of the 74 original questions and their wording were retained in order to retain the psychometric properties of the measure. Thus, individuals were asked how often, in the past two weeks, they had experienced each of the 74 schizotypal symptoms (0 = *not at all*, 1 = *occasionally*, 2 = *sometimes*, 3 = *often*, 4 = *all of the time*). Scores on the SSI range from 0 to 296. A copy of the SSI is provided in Appendix E.

2.4.4.3 Positive and Negative Syndrome Scale (PANSS; Kay et al., 1987)

The PANSS was used to assess the presence and severity of psychotic symptoms in the clinical sample, and to investigate the validity of the SSI as a self-report measure of low-level psychotic phenomena. The PANSS is a 30-item scale developed for the

assessment of phenomena associated with schizophrenia. It involves a semistructured psychiatric interview, used in combination with a detailed rating manual. Items are rated on a seven-point scale of increasing severity, from 1 (*absent*) to 7 (*extreme*). In the current study, symptoms were rated over the past 72 hours. Seven items are used to rate the positive symptoms of psychosis (e.g. delusional ideation, hallucinations, suspiciousness). A further seven items are used to rate the negative symptoms of psychosis (e.g. blunted affect, emotional withdrawal, poor rapport). There is also 16-item subscale assessing general symptomatology (e.g. anxiety, depression, motor retardation). High inter-rater reliability and test-retest reliability have been demonstrated for the scale by Kay et al. (1987) and these have also been replicated more recently (Peralta & Cuesta, 1994).

2.4.5 Procedure

2.4.5.1 Non-clinical sample

Participants were e-mailed the address of a website where they could complete the SSI online. The e-mail invited individuals to take part in a student project investigating how different aspects of emotion were associated with characteristics of personality. There was no mention of the term schizotypy as it was decided that this may be potentially stigmatising to some participants who may misinterpret the term and thus bias the sample. Completion of the questionnaires took approximately 30 minutes and there was no payment or other incentive offered for taking part in the research. The website was "open" for a period of two months following the initial e-mail. After completion of the questionnaires, participants were asked if they were agreeable to take part in a test-retest procedure for the SSI. Those who responded to this request were invited to complete the SSI two weeks after initial completion of the measure. One hundred and two participants responded to this request, thus enabling an assessment of the SSI's reliability over time to be carried out.

An internet survey design was chosen as it was thought that this provided a safe environment for participants to disclose anomalous experiences and unusual thoughts and feelings. The internet is becoming an increasingly popular method of conducting survey research, mostly because it offers many advantages over older surveying

techniques such as mail-outs and telephone interviews (Kaye & Johnson, 1999). Internet surveys reduce the time and cost normally associated with carrying out such research. They also remove the need for data entry as data is automatically saved as participants fill in the survey online. Research into internet-based surveying has shown that once individuals have consented to take part, the method is at least as effective as other modes of surveying. Birnbaum (2001) provides evidence that internet research reaches the same conclusions as laboratory-based research. Furthermore, it has been suggested that web surveys produce less item non-response than telephone surveys (Fricker, Galesic, Tourangeau, & Yan, 2005). Internet surveys may also be more desirable than face-to-face or telephone surveying methods, as they are potentially less intrusive and an individual may feel less pressurised into taking part.

2.4.5.2 Clinical sample

Measures for this study were completed as part of routine clinical assessments in the Early Intervention Service sample; and as part of the baseline assessment process for participants recruited for the ISREP study. The PANSS interviews were carried out by trained researchers who met regularly to ensure reliability and quality control. In the case of Early Intervention Service patients, interviews took place three months after entry into the service. This enabled stabilisation of the psychotic episode. In the case of the patients recruited from the ISREP study, assessments took place approximately one week after consent to enter into the study and before randomisation to either the control or treatment arm of the trial. Following the interview, patients self-completed the self-report questionnaires (i.e. SPQ, SSI). These were completed on a laptop using a specially designed database which stored responses as they were entered. This was done in order to make the response format of the SSI as identical as possible to that utilised by the non-clinical sample in the internet survey. The self-report questionnaires and the symptom interviews were completed within two weeks of one another.

2.4.6 Data Analysis Plan

2.4.6.1 Initial treatment of the data

Non-clinical sample. Raw data from the internet survey were screened and cleaned prior to analyses. The data set was screened for any missing data. For each participant, where answers to more than 10% of items in individual questionnaires were missing, data for that questionnaire was removed. For those participants with less than 10% of responses missing for each questionnaire, prorating was used to replace missing responses. This involved replacing missing variables with the participants' mean scores for the particular subscale in the particular questionnaire in which they occurred. This was considered a valid procedure to use as only a small number of missing variables were prorated (0.54%). Missing data analyses showed that missing data points were randomly distributed across the data set.

Clinical sample. Following entry, data were screened for any anomalous values and these were amended accordingly. Missing data were treated in the same way as for the non-clinical sample. After completion of the interviews and self-report questionnaires, researchers went through the raw data and pointed out any missing responses to the participant. This resulted in a low level of missing data (less than 0.50%).

2.4.6.2 Analyses of the data

All data were analysed using SPSS for Windows, version 14 (SPSS, 2005). In the first stage of the analysis, descriptive statistics and data distributions were calculated for all measures in both samples. Psychometric analyses were then conducted. Internal consistency for the total SSI and its subscales were measured for both samples using Cronbach's alpha internal consistency coefficients. Test-retest reliability was assessed for the total SSI and subscales in the clinical sample using Pearson's product moment correlations. Furthermore, a Principal Components Analysis with varimax rotation was

performed on the combined scale of 74 state schizotypy items for both groups in order to examine the internal factor structure of the scale.

Comparisons of mean scores in clinical and non-clinical samples on the total SSI and its subscales were conducted using a series of independent samples t-tests for normally distributed data. Where data were skewed, non-parametric statistics were used to compare differences between groups. Pearson's product moment correlations were also conducted to investigate the hypothesised relationships between psychotic symptomatology and scores on the SSI in the clinical sample. Where data were skewed, Spearman's Rho correlations were used. These relationships were also taken as a measure of the convergent validity of the scale.

In order to create a shortened version of the SSI consisting of the most reliable items, findings from the factor analyses conducted on the clinical and non-clinical sample data were used. Items loading on the same factors in both groups were selected and scores on each combined factor compared between groups in order to ensure adequate discrimination between clinical and non-clinical populations. The six items with the highest loadings on each factor between the two samples were chosen for the brief scale. Cronbach's alphas for each factor were then calculated for both samples. Where alphas were low, item-total correlations were conducted and items with the highest correlations were selected. Items thought to have particular clinical relevance were retained even if this was not supported by statistical analysis. Psychometric analyses outlined above were then repeated for the brief scale in both samples. Finally, distributions of symptom counts on the SPQ and SSI were plotted using histograms and dot plots. The shape of these distributions was compared.

2.5 RESULTS

This section reports the results of the statistical analyses outlined above. First, descriptive data is provided for both the clinical and non-clinical populations. This is followed by results of the psychometric analysis of the SSI, including internal consistency, test-retest reliability, convergent validity, and the factor analysis. The most reliable items are then used to create a shorter, easier to use, version of the SSI and the

psychometric analyses of this brief scale are reported. Finally, the distributions of symptom counts provided by the SPQ and SSI are compared.

2.5.1 Descriptive Data

2.5.1.1 Non-clinical sample

Table 2.11 provides descriptive data for the SPQ and the SSI in the non-clinical sample. SPQ scores are similar to non-clinical norms reported by other papers (e.g. Raine, 1991).

Table 2.11

Descriptive Data for the Non-Clinical Sample

	N	Min-Max	Median	Mean (*SD*)	Skewness (*SE*)
SPQ Total	808	0-68	27.50	28.59 (14.51)	0.43 (0.09)
SSI Total	808	0-202	33.00	42.23 (33.93)	1.32 (0.09)

2.5.1.2 Clinical sample

Table 2.12 provides descriptive data for the SPQ, the SSI, and the PANSS for the clinical sample. Individuals in the clinical sample displayed relatively low levels of psychotic symptomatology as defined by the PANSS, but appear to experience higher levels of schizotypal phenomena than the non-clinical sample.

Table 2.12

Descriptive Data for the Clinical Sample

	N	Min-Max	Median	Mean (*SD*)	Skewness (*SE*)
SPQ Total	126	0-74	40.50	39.60 (16.19)	-0.16 (0.22)
SSI Total	126	0-251	63.00	67.64 (49.03)	1.44 (0.22)
PANSS Positive	118	7-28	12.00	12.36 (4.33)	0.86 (0.22)
PANSS Negative	118	7-29	13.00	13.56 (4.21)	0.82 (0.22)
PANSS General	118	18-58	31.00	30.71 (7.33)	0.54 (0.22)

2.5.1.3 Normality of the distributions and transforms

In both samples, the distributions of scores were positively skewed for most variables, with the majority of participants scoring in the lower range. This can be seen by the levels of skewness shown for each measure in Tables 2.11 and 2.12. Data are thought to be particularly skewed when the skewness value exceeds +/- 1. Although this is not the case for all variables, Shapiro-Wilk tests showed the distributions for most variables to be significantly different from normal. The one exception to this is SPQ scores, which appear to be normally distributed, in line with Raine et al's (1994) findings. The data were resistant to transformation using both log and square root techniques. Therefore, where data is skewed, non-parametric statistics will be used to investigate relationships and differences between variables. Parallel parametric analyses did however reveal similar findings.

2.5.2 Comparison of SSI Scores in Clinical and Non-Clinical Samples

Independent samples t-tests were used to compare SPQ scores between the clinical and non-clinical samples. The clinical sample displayed significantly higher SPQ scores

than the non-clinical sample, $t(932) = 7.20$, $p < .001$. Independent samples Mann-Whitney U tests were used to compare SSI scores between the two samples. The clinical sample displayed significantly higher SSI scores than the non-clinical sample ($U = 33224.00$, $p < .001$). This suggests that, like the SPQ, the SSI can adequately discriminate between clinical and non-clinical populations.

2.5.3 Psychometric Analysis of the SSI

2.5.3.1 Internal consistency of the SSI

Cronbach's alpha coefficients for the SSI were .96 for the non-clinical sample and .97 for the clinical sample. Cronbach alphas should ideally be between .80 and .90 (Streiner & Norman, 2003). Coefficients for the SSI are therefore high, suggesting that there may be some item redundancy in the measure.

2.5.3.2 Test-retest reliability

To assess the stability of the scale over time, a sample of 103 participants in the non-clinical sample completed the SSI on two occasions, approximately two weeks apart. Mean test-retest response latency was 20.3 days ($SD = 7.7$ days). Test-retest reliability for the total scale was shown to be good, $r(103) = .87$, $p < .001$.

2.5.3.3 Convergent and construct validity of the SSI

Convergent validity of the SSI was examined in the clinical sample using Spearman's Rho correlations with scores on the SPQ. Total SPQ scores correlated with state schizotypy at $r(808) = .89$, $p < .001$ in the non-clinical sample, and $r(126) = .73$, $p < .001$ in the clinical sample. This suggests that the SSI is assessing the same construct as was intended to be measured by the original SPQ. In order to further investigate the validity of the SSI, associations with PANSS symptoms scores were investigated in the clinical sample. Total SSI scores were significantly correlated with PANSS positive symptoms, $r(118) = .60$, $p < .001$; and PANSS general psychopathology scores, $r(118) = .55$, $p < .001$. PANSS negative symptoms scores did not correlate with total SSI scores, $r(118)$

= .14, p = .14. This suggests that the predominant focus of the SSI is low-level positive psychotic phenomena.

2.5.4 Factor Structure of the SSI

In order to investigate the underlying factor structure of the SSI, Principal Components Analysis (PCA) with varimax rotation was performed on the combined scale of 74 state schizotypy items for both the clinical and non-clinical samples. The outcomes of these analyses are reported here. PCA is a data reduction technique often used in scale construction to reduce a larger set of observed variables to a smaller, more manageable number for use in subsequent analyses (Brown, 2006). Varimax rotation is an orthogonal rotation technique designed to maximise the independence of different factors (i.e. factors are constrained to be uncorrelated).

PCA in the non-clinical sample revealed the presence of 16 factors with eigenvalues exceeding one (Kaiser criterion), explaining a cumulative total of 61.13% of the variance. PCA in the clinical sample revealed the presence of 20 factors with eigenvalues exceeding one (Kaiser criterion), explaining a cumulative total of 76.28% of the variance. However, closer inspection of the scree plots (Cattell, 1966) indicated a break in both samples after the fourth component. See Figures 2.1 and 2.2 for scree plots for the non-clinical and clinical samples. Thus, a further PCA was conducted for both groups using a four factor solution. This solution accounted for 40% of the variance in the non-clinical sample and 43.4% of the variance in the non-clinical sample. Eigenvalues from the analysis are shown in Table 2.13.

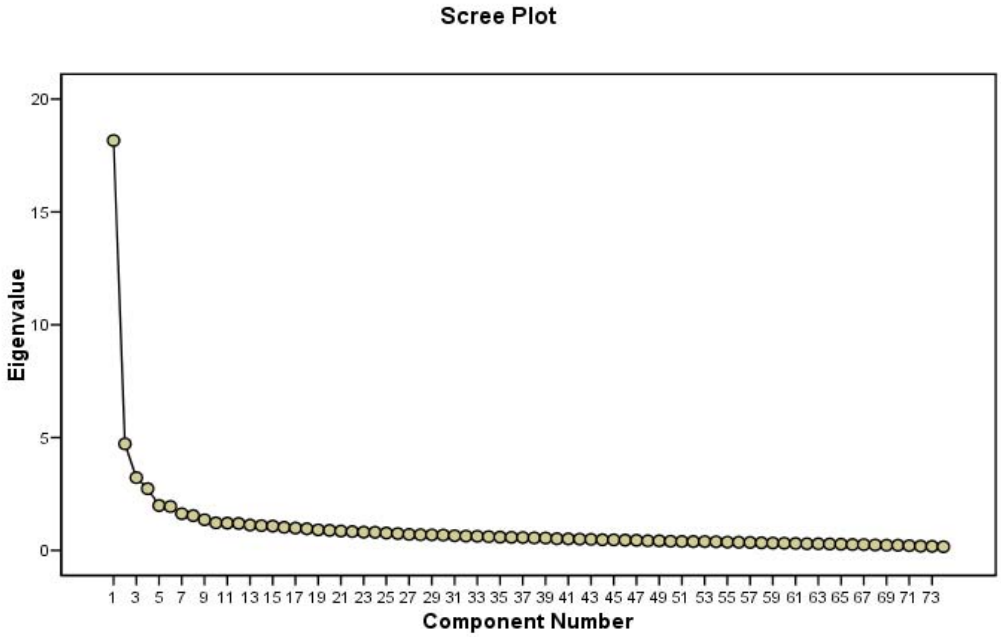


Figure 2.1

Scree plot for the non-clinical sample

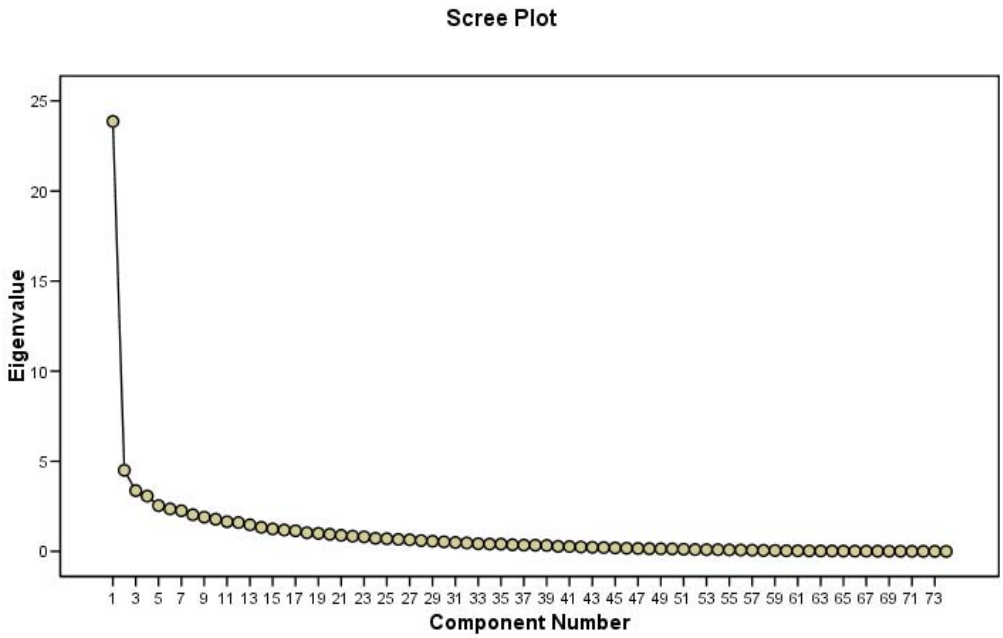


Figure 2.2

Scree plot for the clinical sample

Table 2.13

Dimensionality of the SSI Items: Initial Eigenvalues from a Principal Components Analysis

	Non-Clinical Sample (N = 808)			Clinical Sample (N = 126)		
Component	Total	% variance	Cumulative %	Total	% variance	Cumulative %
1 (Interpersonal)	18.2	24.5	24.5	22.1	29.9	29.9
2 (Disorganised)	4.7	6.4	30.9	3.07	4.1	34.0
3 (Paranoid)	3.2	5.4	36.3	4.1	5.6	39.6
4 (Anomalous)	2.7	3.7	40.0	2.8	3.8	43.4

The same four factors were highlighted in both the non-clinical and the clinical sample. Each factor will now be briefly discussed. Items were considered to "load" on a factor if it had a loading of .30 or above (Tabachnick & Fidell, 2001).

2.5.4.1 Factor 1

The first factor was labelled "Interpersonal Schizotypy" and could be argued to mirror Raine et al's (1994) Interpersonal factor in the original measure. It contains items concerning social anxiety and constricted affect and could be said to be analogous to some of the negative symptoms of psychosis, e.g. withdrawal, flattened affect, blunting, etc. This factor contained 25 items in the non-clinical sample, had an eigenvalue of 18.17, and accounted for 24.5% of the variance. In the clinical group, this factor contained 22 items, had an eigenvalue of 22.13, and accounted for 29.9% of the variance. Nineteen items loaded on this factor in both the clinical and the non-clinical samples and these are shown in Table 2.14.

Table 2.14

Factor Loadings for Factor 1 in Non-clinical and Clinical Samples

	Factor Loading	
	Non-Clinical	Clinical
I tend to keep in the background on social occasions. (SSI 57)	.72	.69
I feel very uneasy talking to people I do not know well. (SSI 71)	.75	.67
I am mostly quiet when with other people. (SSI 24)	.71	.65
I get anxious when meeting people for the first time. (SSI 29)	.63	.64
I have little interest in getting to know other people. (SSI 6)	.36	.63
Do you often feel nervous when you are in a group of unfamiliar people? (SSI 38)	.72	.62
I prefer to keep myself to myself. (SSI 15)	.62	.62
I get very nervous when I have to make polite conversation. (SSI 11)	.62	.59
I feel very uncomfortable in social situations involving unfamiliar people. (SSI 46)	.75	.59
I find it hard to be emotionally close to other people. (SSI 33)	.63	.58
I rarely laugh and smile. (SSI 26)	.48	.56
My non-verbal conversation is poor. (SSI 35)	.55	.55
I do not have an expressive and lively way of speaking. (SSI 68)	.52	.55
Do you feel that you are unable to get close to people? (SSI 66)	.69	.53
I tend to keep my feelings to myself. (SSI 73)	.62	.52
I find it hard to communicate with other people. (SSI 69)	.56	.50
I am poor at expressing my true feelings by the way I talk and look. (SSI 17)	.57	.50
I sometimes avoid going to places where there will be many people because I will get anxious. (SSI 2)	.57	.47
Have you found that it is best not to let other people know too much about you? (SSI 52)	.56	.46

2.5.4.2 Factor 2

The second factor was labelled "Disorganised Schizotypy" and mirrors Raine at al's (1994) Disorganised factor in the original measure. It contains items concerning oddities of behaviour and speech and could be said to be analogous to symptoms such as thought disorder in psychotic conditions. This factor contained 13 items in the non-clinical sample, had an eigenvalue of 4.72, and accounted for 6.4% of the variance. In the clinical sample, this factor contained ten items, had an eigenvalue of 3.07, and accounted for 4.1% of the variance. Nine items loaded on this factor in both the clinical and the non-clinical samples and these are shown in Table 2.15.

Table 2.15

Factor Loadings for Factor 2 in Non-clinical and Clinical Samples

	Factor Loading	
	Non-Clinical	Clinical
I sometimes jump quickly from one topic to another when speaking. (SSI 16)	.52	.76
I often ramble on too much when speaking. (SSI 34)	.56	.71
People sometimes comment on my unusual mannerisms and habits. (SSI 14)	.62	.68
Other people see me as slightly eccentric. (SSI 5)	.70	.67
Do you tend to wander off the topic when having a conversation? (SSI 58)	.59	.57
Sometimes other people think that I am a little strange. (SSI 23)	.71	.54
People occasionally comment that my speech is confusing. (SSI 72)	.62	.52
I sometimes use words in unusual ways. (SSI 50)	.60	.49
People sometimes find it hard to understand what I am saying. (SSI 7)	.52	.47

2.5.4.3 Factor 3

The third factor was labelled "Paranoid Schizotypy" and mirrors one half of Raine et al's (1994) Cognitive-Perceptual factor in the original measure. It contains items concerning suspiciousness and some ideas of reference. Some of the excessive social anxiety subscale also loaded on this factor, which could be said to be analogous to the paranoid symptoms of psychosis. This factor contained 13 items in the non-clinical sample, had an eigenvalue of 3.23, and accounted for 4.4% of the variance. In the clinical sample, this factor contained nine items, had an eigenvalue of 4.11, and accounted for 5.6% of the variance. Seven items loaded on this factor in both the clinical and the non-clinical samples and these are shown in Table 2.16.

Table 2.16

Factor Loadings for Factor 3 in Non-clinical and Clinical Samples

	Factor Loading	
	Non-Clinical	Clinical
Do you sometimes feel that people are talking about you? (SSI 63)	.76	.77
When you see people talking to each other, do you often wonder if they are talking about you? (SSI 53)	.62	.74
Do you sometimes feel that other people are watching you? (SSI 60)	.59	.65
I often feel that others have it in for me. (SSI 59)	.63	.62
When shopping, do you get the feeling that other people are taking notice of you? (SSI 45)	.57	.51
Do you often feel that other people have got it in for you? (SSI 18)	.64	.50
I am sure I am being talked about behind my back. (SSI 9)	.72	.50

2.5.4.4 Factor 4

The fourth factor was labelled "Anomalous Schizotypy" and mirrors the other half of Raine et al's (1994) Cognitive-Perceptual factor in the original measure. It contains items concerning magical ideation and unusual perceptual experiences and could be said to be analogous to some of the positive symptoms of psychosis, e.g. hallucinations and delusions. This factor contained 12 items in the non-clinical sample, had an eigenvalue of 2.74, and accounted for 3.7% of the variance. In the clinical sample, this factor contained 15 items, had an eigenvalue of 2.82, and accounted for 3.8% of the variance. Eleven items loaded on this factor in both the clinical and the non-clinical samples and these are shown in Table 2.17.

2.5.4.5 Other items

Other SSI items either did not load on any of the factors (i.e. loading less than .30) or cross-loaded between different factors (i.e. difference of less than .10 between loadings). They were therefore not considered to be assessing any specific type of symptom and were excluded from further stages of the analysis (Tabachnick & Fidell, 2001).

Table 2.17

Factor Loadings for Factor 4 in Non-clinical and Clinical Samples

	Factor Loading	
	Non-Clinical	Clinical
Have you ever felt that you are communicating with another person telepathically (by mind-reading)? (SSI 55)	.62	.67
Do you believe in telepathy? (SSI 12)	.68	.64
Do you believe in clairvoyancy (psychic forces, fortune telling, etc)? (SSI 30)	.65	.60
Have you ever had the sense that some person or force is around you, even though you cannot see anyone? (SSI 13)	.55	.55
Can other people feel your feelings when they are not there? (SSI 39)	.58	.55
When you look at a person, or yourself in a mirror, have you ever seen the face change right before your eyes? (SSI 22)	.32	.53
Have you had experiences with the supernatural? (SSI 3)	.50	.52
Have you ever noticed a common event or object that seemed to contain a special sign for you? (SSI 28)	.45	.49
Have you ever seen things invisible to other people? (SSI 40)	.56	.44
I often hear a voice speaking my thoughts aloud. (SSI 31)	.30	.44
Have you ever had experiences with astrology, seeing the future, UFOs, ESP, or a sixth sense? (SSI 47)	.64	.37

2.5.5 Descriptive Statistics of Factors

Descriptive data for each of the factors are provided in Table 2.18 for both the non-clinical and clinical samples.

Table 2.18

Descriptive Data for Factors of the SSI

	N	Min-Max	Median	Mean (*SD*)	Skewness (*SE*)
Interpersonal					
Non-Clinical	808	0-64	9.00	13.03 (13.22)	1.32 (0.09)
Clinical	126	0-76	23.00	24.75 (16.49)	0.67 (0.22)
Disorganised					
Non-Clinical	808	0-33	6.00	7.56 (6.56)	1.12 (0.09)
Clinical	126	0-34	6.00	7.98 (7.62)	1.23 (0.22)
Paranoid					
Non-Clinical	808	0-25	2.00	3.44 (4.17)	1.77 (0.09)
Clinical	126	0-28	5.00	6.90 (7.13)	1.20 (0.22)
Anomalous					
Non-Clinical	808	0-31	1.00	2.74 (4.16)	2.67 (0.09)
Clinical	126	0-38	2.00	5.04 (6.78)	2.13 (0.22)

As can be seen from the descriptive data, all distributions of the different factors are positively skewed and thus non-parametric statistics will be used in the following analyses.

2.5.6 Correlations between Different Factors

Spearman's Rho correlations between the different SSI factors are shown in Tables 2.19 and 2.20 for the non-clinical and clinical samples respectively.

Table 2.19

Correlations between SSI Factors in the Non-clinical Sample (N = 808)

	Disorganised	Paranoid	Anomalous
Interpersonal	.47***	.46***	.32***
Disorganised		.44***	.39***
Paranoid			.33***

*p <.05, **p <.01, ***p <.001

Table 2.20

Correlations between SSI Factors in the Clinical Sample (N = 126)

	Disorganised	Paranoid	Anomalous
Interpersonal	.43***	.49***	.36***
Disorganised		.50***	.45***
Paranoid			.57***

*p <.05, **p <.01, ***p <.001

All factors have low to moderate correlations with one another in both samples. This suggests that the factors are not completely independent from one another and somewhat overlap.

2.5.7 Creation of the Brief SSI

A brief version of the SSI was created using the most reliable items from the full scale, based on the factor analysis described above. The Cronbach's alphas for the full scale were high, suggesting that the measure is over-determined and highlighting item redundancy. Shortening the scale will remove redundancy and maximise both coherence and discrimination of items. In addition, most current measures of schizotypy are relatively long and thus a quick and easy-to-use tool for use in this domain is well needed.

2.5.7.1 Initial factor selection

In order to create a shortened version of the SSI, independent samples Mann-Whitney U tests were initially conducted to compare scores on each of the factors outlined above between the clinical and non-clinical samples. This was to ensure that the brief scale would adequately discriminate between clinical and non-clinical populations. As each factor contained a different number of items, factor scores were scaled prior to this analysis. Significant differences were found between Interpersonal (U = 27835.00, p <.001), Paranoid (U = 36473.50, p <.001), and Anomalous (U = 40724.50, p <.001) factor scores in the two samples, with the clinical sample scoring significantly higher than the non-clinical sample. However, there was no significant difference between the two samples on scores on the Disorganised factor (U = 50763.50, p = .96). These differences are further illustrated in Figure 2.3. It was therefore decided to remove Disorganised items from the brief version of the SSI, due to their lack of discriminative specificity. Thus, no items from the Odd Speech and Odd Behaviour subscales were included in the brief SSI.

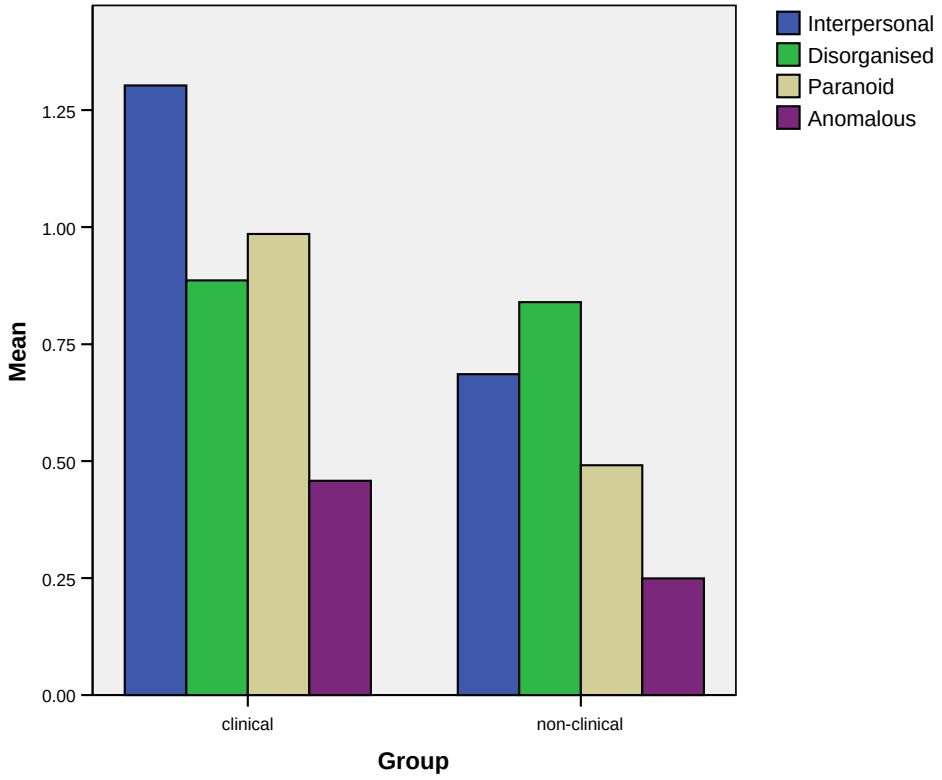


Figure 2.3

Comparison of mean SSI scores in clinical and non-clinical samples

2.5.7.2 Item Selection

Item-total correlations were calculated for all items loading on the remaining three factors in both samples. Item-total correlations for the Interpersonal Schizotypy factor ranged from .43 to .70 in the clinical sample and from .34 to .74 in the non-clinical sample. For the Paranoid Schizotypy factor, item-total correlations ranged from .48 to .80 in the clinical sample and from .50 to .76 in the non-clinical sample. Finally, for the Anomalous Schizotypy factor, item-total correlations ranged from .31 to .62 in the clinical sample and .30 to .55 in the non-clinical sample. A combination of the highest factor loadings and highest item-total correlations were used to select the final items for the Brief SSI. To maximise separation between subscales, items which loaded high on the factor of interest and low on other factors were selected.

This resulted in an 18-item scale made up of three subscales: a "Social Anxiety" scale consisting exclusively of items from the Excessive Social Anxiety subscale of the original SPQ; a "Paranoia" scale consisting of items from the Suspiciousness and Ideas of Reference subscales of the original SPQ; and an "Anomalous Experiences" scale consisting of items from the Odd Beliefs, Ideas of Reference and Unusual Perceptual Experiences subscales from the original SPQ. An additional two items (items 31 and 40) were added to the Anomalous Experiences subscale even though they were not the highest loadings for that factor. These items were retained as they were thought to be of important clinical relevance, reflecting low-level hallucinatory experience. Items included in the Brief SSI are shown in Table 2.21.

Table 2.21

Items Included in the Brief SSI

Scale	Item Number	Question
Social Anxiety	2	I sometimes avoid going to places where there will be many people because I will get anxious.
	11	I get very nervous when I have to make polite conversation.
	29	I get anxious when meeting people for the first time.
	38	Do you often feel nervous when you are in a group of unfamiliar people?
	46	I feel very uncomfortable in social situations involving unfamiliar people.
	71	I feel very uneasy talking to people I do not know well.
Paranoia	9	I am sure I am being talked about behind my back.
	18	Do you often feel that other people have it in for you?
	53	When you see people talking to each other, do you often wonder if they are talking about you?
	59	I often feel that others have it in for me.
	60	Do you sometimes feel that other people are watching you?
	63	Do you sometimes feel that other people are talking about you?
Anomalous	12	Do you believe in telepathy (mind-reading)?
	13	Have you ever had the sense that some person or force is around you, even though you could not see anyone?
	28	Have you ever noticed a common event or object that seemed to be a special sign for you?
	30	Do you believe in clairvoyancy (psychic forces, fortune telling, etc)?
	31	I often hear a voice speaking my thoughts aloud.
	39	Can other people feel your feelings when they are not there?
	40	Have you ever seen things invisible to other people?
	55	Have you ever felt that you are communicating with another person telepathically (by mind-reading)?

2.5.7.3 Description of the Brief SSI

The Brief SSI is a 20-item scale assessing state symptoms of schizotypy over three domains: Anomalous Experiences (8 items); Social Anxiety (6 items); and Paranoia (6 items). All items have the same 5-point Likert scale as the full scale in order to determine recent frequency of occurrence of each symptom, ranging from 0 (*not at all*) to 4 (*all of the time*). Therefore the total possible score on the scale as a whole is 80 (made up of 24 possible points each for the Social Anxiety and Paranoia subscales and 32 possible points for the Anomalous Experiences subscale). Items were ordered randomly to avoid clusters of questions asking about the same types of symptoms. A copy of the Brief SSI is provided in Appendix E.

2.5.8 Descriptive Data for the Brief SSI

Descriptive statistics and norms of schizotypy scores for both the total Brief SSI and each of the subscales in the clinical and non-clinical samples are shown in Table 2.22. All distributions were positively skewed and could not be corrected via transformation of the data. Thus non-parametric statistics were used in all analyses. All between-group differences were shown to be significant at the $p = .001$ level using independent samples Mann-Whitney U tests. All mean scores in the clinical group were equivalent to scores in the 75^{th} percentile or above in the non-clinical sample, suggesting that all types of schizotypal symptoms are more prevalent in clinical samples. However, the largest differences between the two populations were on the Social Anxiety and Paranoia subscales, with Anomalous Experiences acting as a weaker discriminator.

Table 2.22

Norms and Descriptive Data for Brief SSI

	Min-Max	Median	Mean (*SD*)	Skewness (*SE*)	*U*	*p*	25th centile	50th centile	75th centile	90th centile
SSI Brief Total										
Non-clinical (N = 808)	0-53	7.00	9.54 (9.22)	1.46 (0.09)	31589.00	<.001	3.00	7.00	14.00	23.00
Clinical (N = 126)	0-80	15.00	18.67 (15.70)	1.31 (0.22)			7.00	15.00	27.00	39.30
Social Anxiety										
Non-clinical (N = 808)	0-24	3.00	4.41 (5.14)	1.46 (0.09)	30961.00	<.001	0.00	3.00	7.00	12.00
Clinical (N = 126)	0-24	7.50	8.56 (6.65)	0.50 (0.22)			3.00	7.50	13.25	19.30
Paranoia										
Non-Clinical (N = 808)	0-21	2.00	2.85 (3.63)	1.88 (0.09)	36126.00	<.001	0.00	2.00	4.00	7.00
Clinical (N = 126)	0-24	4.00	5.99 (6.41)	1.24 (0.22)			1.00	4.00	9.00	16.60
Anomalous Experiences										
Non-Clinical (N = 808)	0-23	1.00	2.28 (3.43)	2.45 (0.09)	41525.50	.001	0.00	1.00	4.00	6.00
Clinical (N = 126)	0-32	2.00	4.12 (5.69)	2.35 (0.22)			0.00	2.00	6.00	11.30

2.5.9 Endorsement Frequencies

The average frequency of endorsement for each schizotypal symptom included in the Brief SSI is shown in Table 2.23. Endorsement was defined as a symptom having occurred often or all of the time in the last two weeks. Social Anxiety symptoms were the most common phenomena in the clinical sample, with 52.4% of the sample experiencing at least one symptom of social anxiety often or all of the time in the past two weeks. This is compared to 24.1% in the non-clinical sample. Thirty-five per cent of the clinical sample had experienced at least one symptom of paranoia often or all of the time in the past two weeks, compared with 13.5% of the non-clinical sample. Thirty-two per cent of the clinical sample had experienced at least one anomalous experience often or always in the past two weeks, compared with 18.7% of the non-clinical sample. Endorsement frequencies were compared between the clinical and non-clinical samples using chi-square tests. Social Anxiety and Paranoid symptoms were the strongest discriminators between clinical and non-clinical samples. Anomalous Experiences were the weakest discriminator.

Table 2.23

Average Endorsement of Brief SSI Items in Non-clinical and Clinical Samples

		Endorsement (%)		
		Non-Clinical	Clinical	χ^2
Social Anxiety	I sometimes avoid going to places where there will be many people because I will get anxious.	6.4	26.2	51.4*
	I get very nervous when I have to make polite conversation	8.8	19.8	14.4*
	I get anxious when meeting people for the first time	12.5	25.4	14.8*
	Do you often feel nervous when you are in a group of unfamiliar people?	13.1	31.0	26.4*
	I feel very uncomfortable in social situations involving unfamiliar people	8.3	24.6	30.9*
	I feel very uneasy talking to people I do not know well	8.4	27.0	38.6*
Paranoia	I am sure I am being talked about behind my back.	7.5	21.4	24.6*
	Do you often feel that other people have it in for you?	3.2	16.7	41.3*
	When you see people talking to each other, do you often wonder if they are talking about you?	4.0	13.5	19.9*
	I often feel that others have it in for me.	2.2	11.9	29.9*
	Do you sometimes feel that other people are watching you?	3.7	19.0	47.1*
	Do you sometimes feel that other people are talking about you?	3.3	16.7	39.7*
Anomalous Experiences	Do you believe in telepathy?	2.1	8.7	16.5*
	Have you had the sense that some person or force is around you, even though you could not see anyone?	6.9	11.9	3.8
	Have you noticed a common event or object that seemed to be a special sign for you?	2.6	4.8	1.8
	Do you believe in clairvoyancy?	3.5	7.1	4.3
	I often hear a voice speaking my thoughts aloud.	8.5	13.5	3.2
	Can other people feel your feelings when they are not there?	1.4	7.9	21.4*
	Have you seen things invisible to other people?	0.9	5.6	16.2*
	Have you felt that you were communicating with another person telepathically?	1.5	4.8	6.2

*p <.05 adjusted for multiple comparisons ($\alpha/20 = 0.0025$)

2.5.10 Psychometric Analysis of the Brief SSI

2.5.10.1 Internal consistency

Cronbach's alpha coefficients for the total brief scale were .87 for the non-clinical sample and .92 for the clinical sample. Coefficients for each of the subscales for the non-clinical and clinical samples respectively were: .89 (.89) for Social Anxiety; .85 (.90) for Paranoia; and .72 (.83) for Anomalous Experiences. These coefficients are more acceptable than those for the full scale and suggest that the Brief SSI has good internal validity but is not over-determined.

2.5.10.2 Test-retest reliability

Test-retest reliability for the shorter version of the scale was calculated using Pearson's correlations on the data used to calculate test-retest reliability for the longer version of the scale. A test-retest correlation of $r(103) = .86$, $p < .001$ was shown for the total brief scale and test-retest correlations ranged from .62 to .86 for the subscales ($p < .001$). Thus the scale shows good reliability over time.

2.5.10.3 Convergent and construct validity

Correlations between the brief and long versions of the SSI were $r(808) = .87$, $p < .001$ for the non-clinical sample and $r(126) = .90$, $p < .001$ for the clinical sample. This suggests that the shorter scale adequately reflects the content of the longer instrument. Correlations between the brief SSI and the original SPQ are shown in Tables 2.24 and 2.25 for the non-clinical and clinical samples respectively.

Table 2.24

Correlations between Brief SSI and SPQ Scores in the Non-clinical Sample (N = 808)

	Total SSI Brief Score	Social Anxiety	Paranoia	Anomalous Experiences
SPQ				
- Total	.78***	.63***	.63***	.50***
- Cognitive Perceptual	.70***	.39***	.66***	.60***
- Interpersonal	.75***	.75***	.58***	.29***
- Disorganised	.52***	.39***	.42***	.37***

*p <.05, **p <.01, ***p <.001

Table 2.25

Correlations between Brief SSI and SPQ Scores in the Clinical Sample (N = 126)

	Total SSI Brief Score	Social Anxiety	Paranoia	Anomalous Experiences
SPQ				
- Total	.67***	.59***	.58***	.50***
- Cognitive Perceptual	.65***	.45***	.60***	.58***
- Interpersonal	.62***	.64***	.53***	.38***
- Disorganised	.47***	.44***	.42***	.32***

*p <.05, **p <.01, ***p <.001

In order to further investigate the validity of the Brief SSI, scores on the measure were correlated with PANSS scores in the clinical sample. These correlations are shown in Table 2.26.

Table 2.26

Correlations between Brief SSI and PANSS Scores in the Clinical Sample (N = 118)

	Total SSI Brief Score	Social Anxiety	Paranoia	Anomalous Experiences
PANSS Positive	.58***	.33***	.56***	.67***
PANSS Negative	.11	.03	.18*	.09
PANSS General	.48***	.40***	.44***	.40***

*p <.05, **p <.01, ***p <.001

The correlations suggest a bias of the brief SSI towards positive symptoms. There were little to no correlations with measures of negative symptoms.

2.5.11 Distributions of Trait and State Symptom Counts

Distributions of trait and state schizotypy symptom counts (number of items endorsed) were plotted for the non-clinical and clinical samples using histograms and dot plots. Trait distributions were plotted on a histogram using total scores on the SPQ (i.e. number of "yes" responses). These are shown in Figures 2.4 and 2.5 and can be seen to broadly fit a normal distribution, in line with previous findings (e.g. Johns & van Os, 2001).

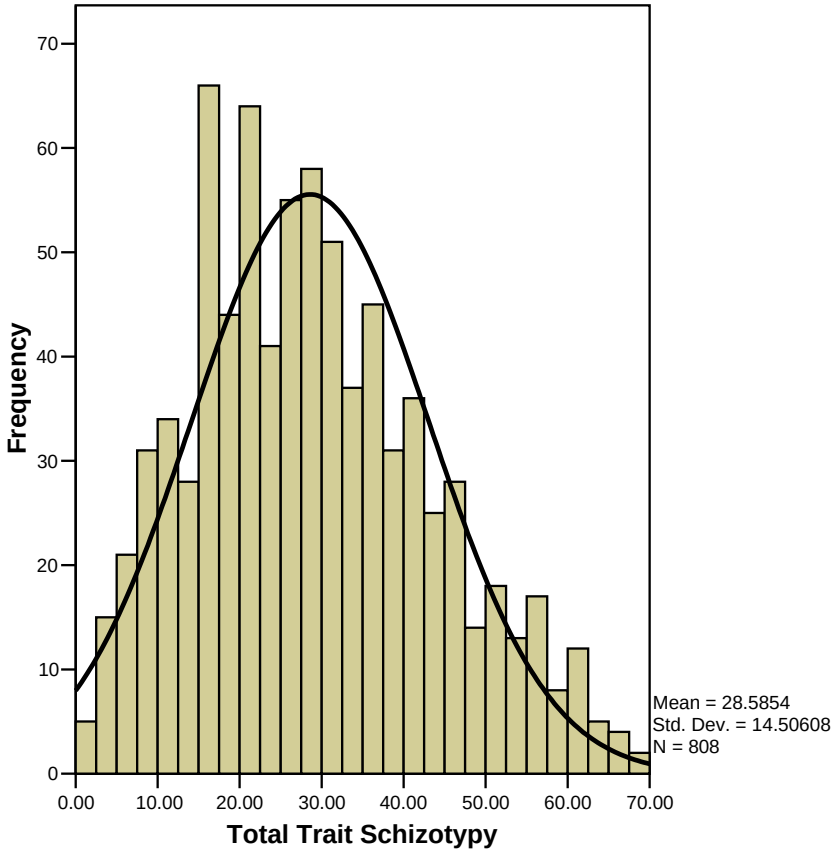


Figure 2.4

Frequency distribution of SPQ (trait schizotypy) scores in the non-clinical sample with fitted Gaussian curve

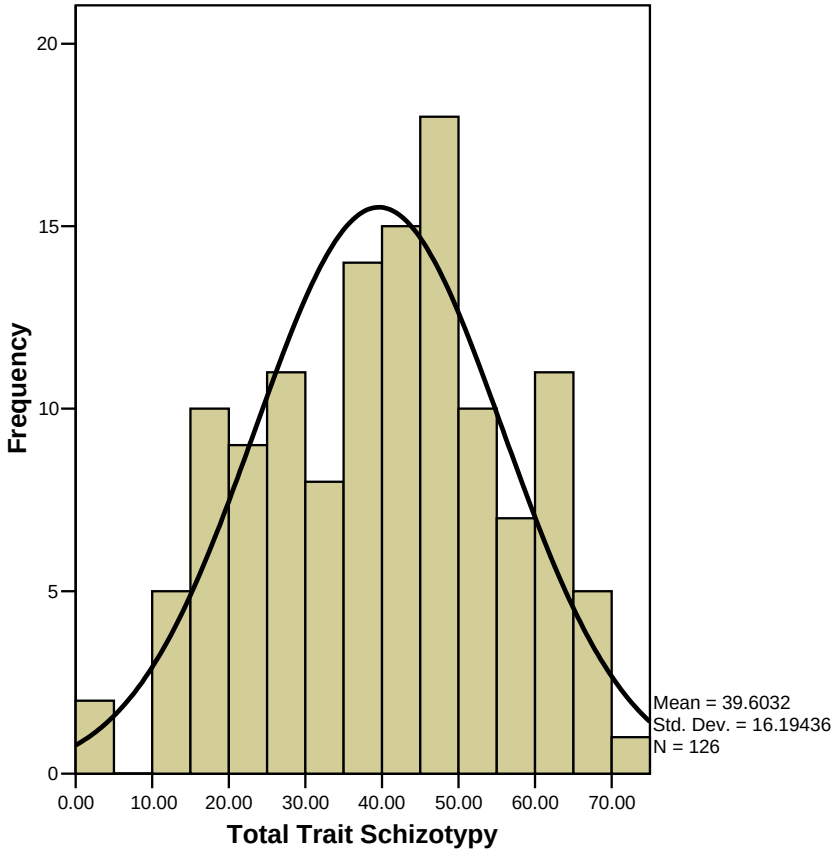


Figure 2.5

Frequency distribution of SPQ (trait schizotypy) scores in the clinical sample with fitted Gaussian curve

In order to investigate the distribution of state schizotypy scores, the number of brief SSI items endorsed by each participant for the total scale and each of the subscales was calculated for both the clinical and non-clinical samples. Endorsement was defined as a symptom having occurred often or all of the time in the last two weeks. This produced a symptom count for each participant which ranged from 0-20 for the total scale; 0-6 for Social Anxiety and Paranoia subscales; and 0-8 for the Anomalous Experiences subscale. These symptom counts were then plotted against sample frequency to obtain a symptom count distribution. Distributions of state symptom counts for the non-clinical and clinical samples are shown in Figures 2.6 to 2.9. These distributions are shown to be extremely skewed. Furthermore, Shapiro-Wilk tests demonstrated that distributions of state schizotypal symptoms were significantly different from normal in both the non-clinical (Total Scale: $w = 0.62$, $p < .001$; Social Anxiety: $w = 0.52$, $p < .001$; Paranoia: $w = 0.36$, $p < .001$; Anomalous: $w = 0.43$, $p < .001$) and clinical (Total Scale: $w = 0.77$, $p < .001$; Social Anxiety: $w = 0.76$, $p < .001$; Paranoia: $w = 0.62$, $p < .001$; Anomalous: $w = 0.55$, $p < .001$) samples.

The deviation from the normal distribution suggests that the SSI may be assessing phenomena which are less common than phenomena assessed by the SPQ (Lyoo, Youn, Ha, Park, & Kwon, 2003). Indeed, in the non-clinical sample, only 44% of individuals scoring above the median on the SPQ also scored above the 75th percentile on the SSI. Similarly, in the clinical sample, only 43% of individuals scoring above the mean on the SPQ also scored above the 75th percentile on the SSI. This suggests that there are a large group of individuals (56% and 57% in the non-clinical and clinical samples respectively) who score high on trait schizotypy but not on state schizotypy (bottom right corner on Figures 2.10 and 2.11). It is the group of individuals who score high on both state and trait schizotypy (top right corner on Figures 2.10 and 2.11) who may be most at risk. The SSI would identify these individuals whereas the SPQ would not.

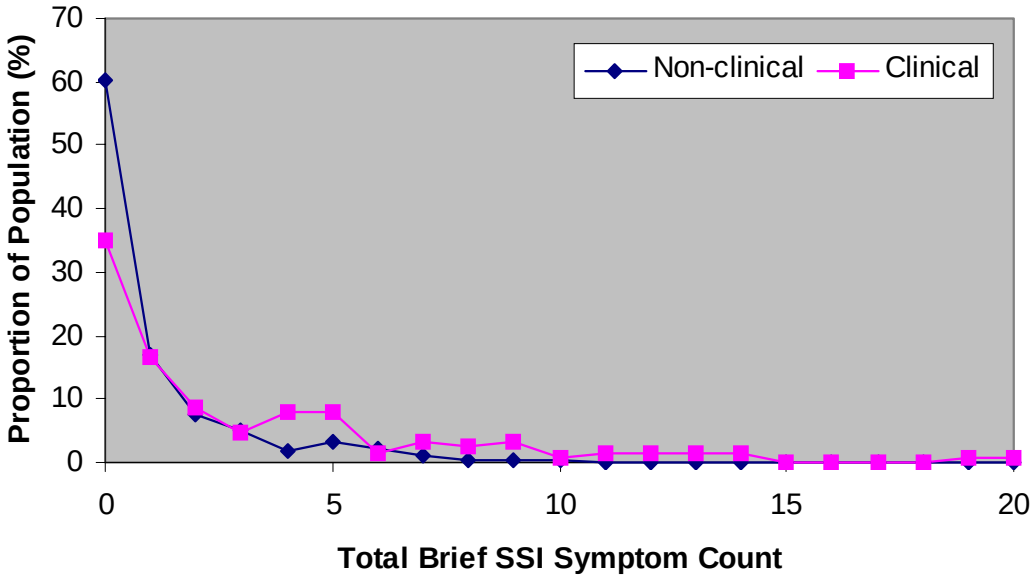


Figure 2.6

Distribution of total state schizotypal symptom counts (often/all of the time in the last two weeks) in the non-clinical and clinical samples

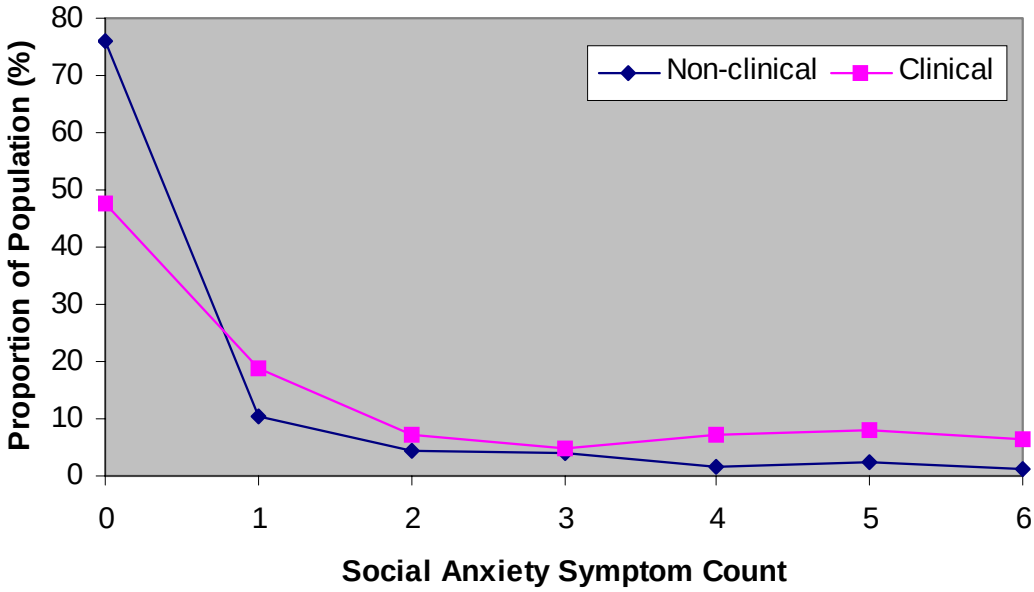


Figure 2.7

Distribution of state Social Anxiety schizotypal symptom counts (often/all of the time in the last two weeks) in the non-clinical and clinical samples

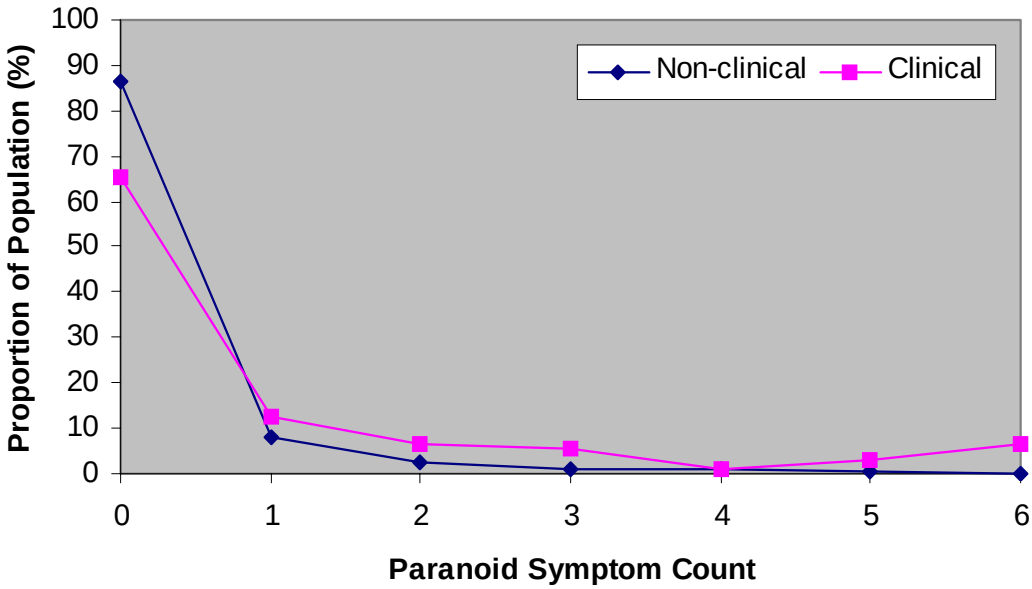


Figure 2.8

Distribution of state Paranoid schizotypal symptom counts (often/all of the time in the last two weeks) in the non-clinical and clinical samples

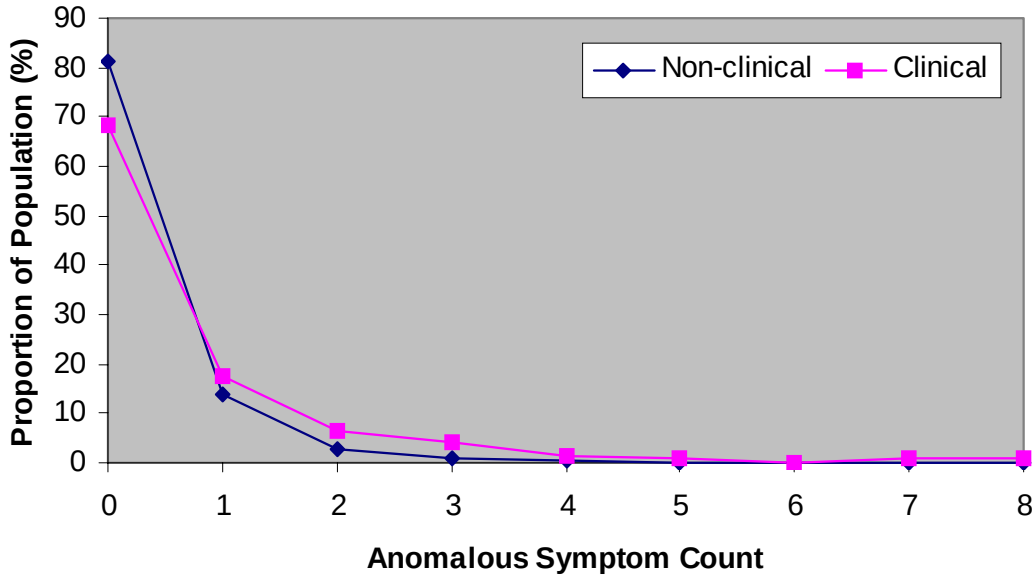
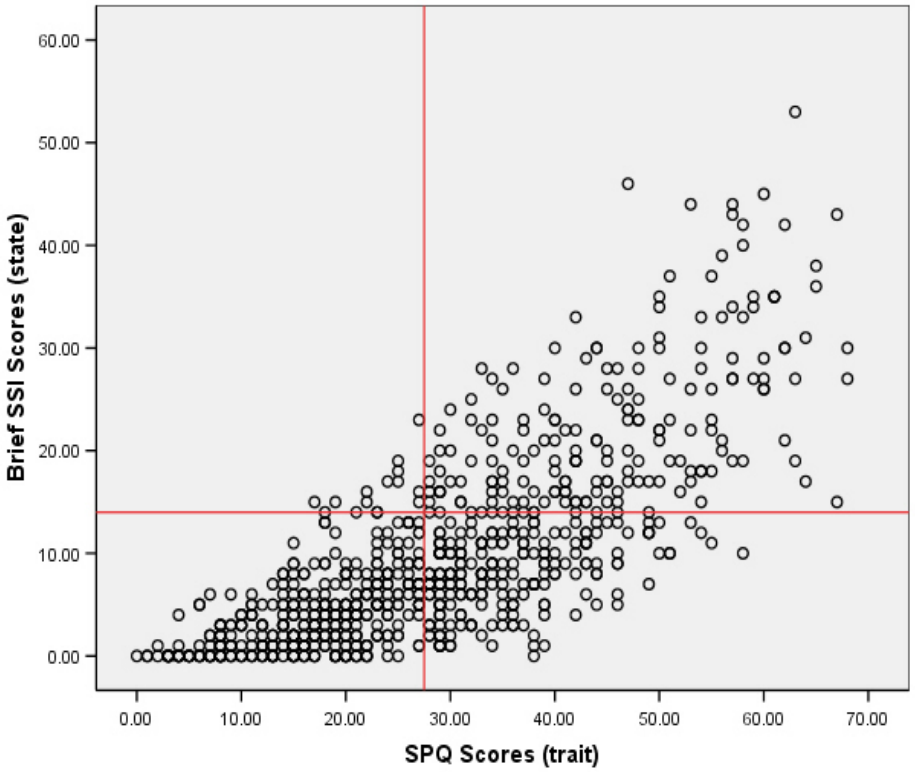
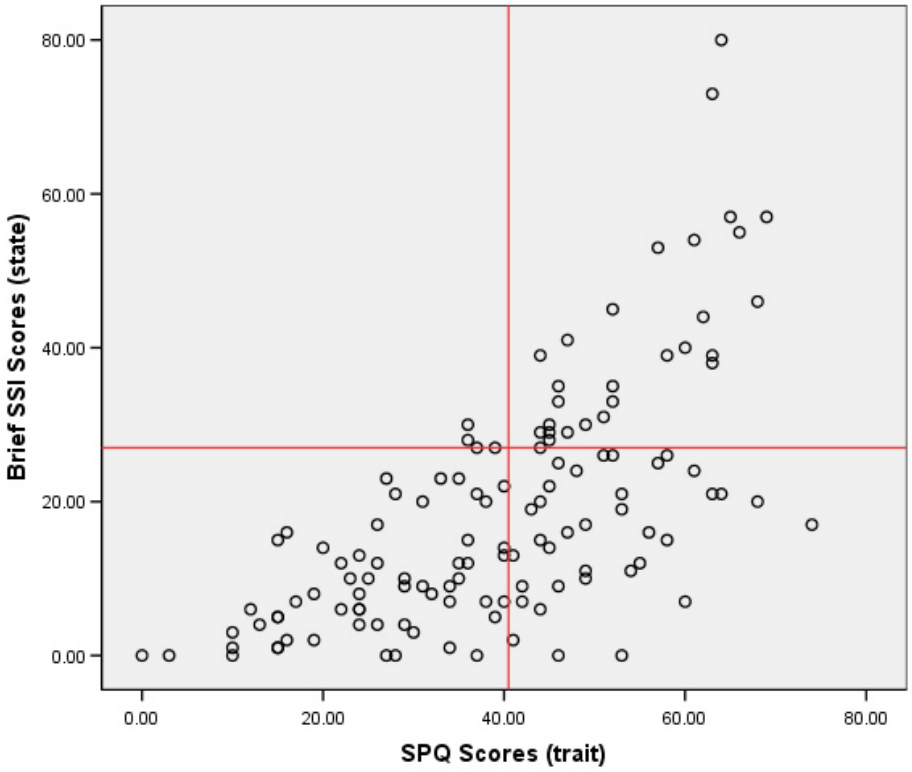


Figure 2.9

Distribution of state Anomalous schizotypal symptom counts (often/all of the time in the last two weeks) in the non-clinical and clinical samples





Note. Reference line on the X axis refers to median SPQ score. Reference line on the Y axis refers to 75th percentile SSI score.

Figure 2.10

Scatter plot of SPQ and Brief SSI scores in the non-clinical sample (N = 808)

Figure 2.11

Scatter plot of SPQ and Brief SSI scores in the clinical sample (N = 126)

2.6 DISCUSSION

This chapter has reported on the modification of a self-report measure, originally designed to assess schizotypal personality traits, so that it can be used to assess the current (i.e. state) presence and frequency of low-level psychotic symptomatology in both clinical and non-clinical populations. Such a scale could be used to detect the prodromal and residual symptoms of psychosis. This section will review the results of the current study. The findings will be discussed in relation to the current literature and the clinical implications will be examined. Following on from this, potential weaknesses of the study will be outlined, as will possibilities for future research.

2.6.1 Summary and Discussion of the Psychometric Properties of the SSI

The current study suggests that both long and brief versions of the SSI have robust psychometric properties. The analyses indicate good internal consistency (particularly in the brief version) and stability of the scale over time, in both clinical and non-clinical samples. Moreover, a Principal Components Analysis has highlighted a four-factor structure, resembling that of the SPQ (Raine et al., 1994), but where the Cognitive-Perceptual factor is divided into two separate but related components: Paranoia and Anomalous Experiences. This split may be important in differentiating between clinical and non-clinical populations.

In addition, the SSI has been found to adequately discriminate between clinical and non-clinical samples. It was hypothesised that the clinical sample would score more highly on the SSI than the non-clinical sample. A series of independent samples Mann-Whitney U tests showed that the clinical sample had higher scores on all of the factors, apart from the Disorganised factor which did not discriminate between the two samples. This was later removed to create the brief version of the SSI. Chi square tests revealed that social anxiety symptoms were the largest discriminator between clinical and non-clinical samples, suggesting that social anxiety may be an important clinical problem during the recovery stages of psychosis. Anomalous experiences were the weakest discriminator, suggesting that these may be the first symptoms to remit after a psychotic episode.

Analyses also suggest that the SSI is valid in assessing psychotic-like experiences, due to associations with the PANSS. It was hypothesised that within the clinical sample, higher scores on measures of psychopathology would be associated with higher scores on the SSI. Spearman's Rho correlations showed positive relationships between SSI scores and scores on the Positive and General subscales of the PANSS. However, little to no associations were shown between SSI scores and the Negative subscale of the PANSS. Similar findings were shown for the Brief SSI which was found to be associated with measures of positive psychotic symptoms but not with measures of negative psychotic symptoms. These findings suggest that the SSI is more sensitive and specific to the assessment of low-level positive psychotic symptoms, as opposed to negative or disorganised symptoms. It could be argued that negative and disorganised symptoms are better assessed in an objective as opposed to self-report manner (Andreasen, 1981). For example, an individual's social anxiety or paranoia may lead them to believe that they exhibit traits similar to those described by criteria for thought disorder (e.g. getting words mixed up, rambling, etc). However, in reality, and when measured objectively, these may not be present.

Based on its assessment of the presence and frequency of current schizotypal symptoms, it was hypothesised that the SSI would have a more clinical focus than the SPQ, which takes normality as its reference point. This hypothesis was supported by the differing distributions of symptom counts from the SPQ and the SSI. In both the clinical and non-clinical samples, distributions of symptom counts on the SPQ were shown to fit a normal Gaussian distribution, suggesting that schizotypy assessed by the measure is a quantitative trait reflecting normal variation in personality. Conversely, distributions of state schizotypal symptom counts using the SSI (i.e. the number of symptoms experienced often or always in the last two weeks) were extremely skewed and significantly different from normal. The half-normal distribution of SSI symptom counts mirrors that of other measures designed to assess schizotypal experiences as attenuated symptoms of psychosis (e.g. V. Bell et al., 2006; Peters, Joseph et al., 1999). Thus, the difference in distributions between the SPQ and the SSI from normal to half-normal could be taken as a form of validity that the SSI is assessing quasi-psychotic experiences, rather than personality traits (Johns & van Os, 2001).

A deviation from the normal distribution suggests that the SSI is assessing phenomena which are less common than schizotypal traits (Lyoo et al., 2003). Moreover, the finding that not all high-trait schizotypy individuals had high scores on state schizotypy could be taken as further evidence to suggest that the SSI provides a more accurate reflection of an individual's current symptom profile. Indeed, two individuals could score the same on a measure of trait schizotypy and yet have totally different state presentations. The SSI would allow discrimination of these two individuals, whereas the SPQ would not – they would both score in the right-hand tail of the Gaussian distribution.

2.6.2 Clinical Implications of Findings

The use of a state measure of schizotypy is extremely clinically relevant and has many advantages in that it allows the presence of current symptoms and their frequency to be assessed. This is in contrast to the assessment of general tendencies and lifetime experience as measured by trait schizotypy tools. Assessing dimensional concepts such as frequency is particularly important when thinking about transition to a psychotic episode. Indeed, a large proportion of people appear to experience a few schizotypal symptoms occasionally; but only a minority of people experience many of these symptoms frequently. It is this latter group of individuals who could be considered at greater risk of making the transition to a psychotic episode. Birchwood (1996) suggests that increased frequency of initial, low-level, anomalous or schizotypal experiences, may result in an extreme emotional reaction and drive a "search for meaning", potentially leading to the delusional systems characteristic of psychosis. In addition to this, the fact that low-level psychotic symptoms remain following recovery from acute psychosis is of important clinical relevance and could have implications in terms of defining early warning signs and relapse (A. Tait et al., 2002).

The SSI can be used to assess symptomatology in both clinical and non-clinical populations, thus enabling comparisons to be made between the two samples. Such a measure is relatively novel in its development as many other measures of schizotypy are validated using only community samples (J. P. Chapman, Chapman, & Kwapil, 1995). Conversely, most traditional measures of psychotic phenomena are only suitable for use with clinical populations and are often not sensitive enough to assess low-level

symptomatology (e.g. Kay et al., 1987). The SSI is could be argued to bridge this gap in assessment tools. Furthermore, although the SSI assesses symptom frequency over the past two weeks, this time scale could be changed in order to assess symptom change over longer or shorter periods dependent on the demands of the investigation, e.g. "Have you experienced X in the last month/week/24 hours?" The Brief SSI may be particularly useful in longitudinal studies and clinical practice in order to assess changes in symptoms over time. Most current measures of schizotypy are relatively long and thus a quick and easy-to-use tool for use in this domain is much needed.

2.6.3 Relevance to the Literature and Theoretical Significance of the SSI

As well as providing evidence for the reliability and validity of the SSI, the results from this study could be taken as support for the continuum hypothesis of psychosis (e.g. Johns & van Os, 2001). Indeed, the distributions of both schizotypal traits and state schizotypal symptoms in the non-clinical sample are continuous (rather than bimodal), suggesting that schizotypal phenomena may exist on a continuum. Moreover, although the clinical sample experienced significantly more schizotypal experiences, they were not uncommon in the non-clinical sample, with 20% of individuals surveyed having had an anomalous experience often or all of the time in the last two weeks. This is quite staggering when considering that the frequency of psychotic disorder is 0.5% (Robins & Regier, 1991) and supports other prevalence studies in this area (Johns, Nazroo, Bebbington, & Kuipers, 1998; Myin-Germeys et al., 2003; Ohayon, 2000; Olfson et al., 2002; van Os, Hanssen, Bijl, & Ravelli, 2000; Verdoux & van Os, 2002). In line with previous research, it appears that it is not the experience of psychotic-like symptoms per se which is unique to psychosis. Recent studies have suggested that it may be the interpretation of the experience which separates normality from pathology (Lincoln, 2007).

The presence of high levels of low-level schizotypal symptomatology in a clinical sample defined as having "recovered" from acute psychosis could be argued to support the rollback phenomenon. This is an idea which suggests that as an illness remits; many of the stages and symptoms that were seen during the early stages of its development are repeated, but in the reverse order (Detre & Jarecki, 1971; Fava, 1999). However, longitudinal research would need to be conducted over the entire course of psychosis in

order to fully examine this hypothesis (i.e. to investigate whether there similarities exist between prodromal and residual symptoms). Social anxiety appears to be particularly prevalent in individuals recovering from psychosis, with over half of the participants in the clinical sample reporting experiencing at least one symptom of social anxiety often or all of the time in the previous two weeks. This supports literature suggesting that social anxiety is an important clinical problem following psychosis (Birchwood et al., 2006).

2.6.4 Weaknesses of the Current Study

There are a number of considerations which should be borne in mind when interpreting the results of this study. First, it could be argued that the non-clinical sample was not epidemiologically representative due to the fact that they were university students (Prescott, 2002). However, in terms of a comparison sample for an early psychosis population, students are matched in terms of age, although not necessarily social status (Hodges et al., 1989). It could also be argued that the way in which participants in the non-clinical sample were recruited (i.e. via opportunity sampling) may have biased the sample, with individuals with a personal interest in or personal experience of this area choosing to take part (Freeman, Garety et al., 2005). Furthermore, it is unknown whether any of the participants had received treatment for a psychiatric disorder, and what the level of substance abuse was in the group. These weaknesses can only be overcome by the use of standardised and epidemiologically representative sampling methodology, as used in other prevalence studies (Johns & van Os, 2001). This was not feasible for the current study.

Despite the attractiveness of internet research, there are some concerns attached to its usage. Indeed, the generalisability of findings from such studies has been questioned due to the fact that participation is restricted to those who have access to computer networks (Best, Krueger, Hubbard, & Smith, 2001). However, as the current study was aiming to recruit participants from student populations, where internet access is extremely high, coverage bias was not thought to be a problem. Furthermore, using a web-based technique allowed the specific population under investigation to be targeted more easily, i.e. by using year group e-mailing lists. Web-based surveys have also been criticised for having lower response rates than other modes of data collection in this

domain (Couper, Blair, & Triplett, 1999; Fricker et al., 2005). However, this was overcome in the current study by sampling a large number of people. A strength of web-based methodology is that it is not significantly more resource-intensive to send an e-mail to 10,000 individuals than to 100 individuals.

A further weakness of the current study is that participants in the clinical sample were relatively recovered and as such may not have been experiencing symptomatology which may otherwise have discriminated between a clinical and non-clinical population, e.g. disorganised symptoms (McGlashan, 1987; Torgersen, 1985). However, it must be remembered that the aim of creating the SSI was to modify it to assess low-level psychotic symptomatology. Such phenomena are arguably best investigated in a recovered sample as ceiling effects may have been observed in a more acute sample where symptomatology is more severe.

It could be argued that although based on DSM-III-R diagnostic criteria, the SSI is somewhat biased towards positive psychotic phenomena and social anxiety. This is particularly true of the brief version of the scale where many of the negative symptom (e.g. Constricted Affect, No Close Friends) and disorganisation (e.g. Odd Behaviour, Odd Speech) subscales have been removed. However, when conducting comparisons of different symptom types between clinical and non-clinical samples, it was the positive-type (i.e. paranoia and anomalous experiences) and social anxiety items which were the best discriminators. Furthermore, negative and disorganised symptoms are arguably more accurately assessed using objective as opposed to self-report methods. There are numerous independent tools which could be used in addition to the SSI to assess such phenomena if this was deemed important (e.g. SANS; Andreasen, 1981).

2.6.5 Summary

In summary, the SSI has been shown to be a reliable and valid measure for assessing current low-level psychotic phenomena, in both clinical and non-clinical populations. Whilst assessment tools already exist to measure the presence of paranoia (e.g. Freeman, Garety et al., 2005), interpersonal anxiety (e.g. Liebowitz, 1987), and even hallucinatory phenomena (V. Bell et al., 2006; Launay & Slade, 1981) in non-clinical populations; the SSI assesses a range of schizotypal phenomena simultaneously and can

also be used to assess quasi-psychotic experiences in individuals in recovery from psychosis. Thus, the current study offers a significant contribution to research in this area, and provides a robust tool for use in later studies in this thesis. The studies which follow aim to elucidate the underlying mechanisms of different schizotypal symptom types and examine their role in recovery from psychosis.

CHAPTER THREE:
STUDY TWO: THE MCCOLLOUGH EFFECT AND POSITIVE SCHIZOTYPAL SYMPTOMS IN PSYCHOSIS

3.1 RATIONALE AND CONTEXT FOR THE STUDY

The previous chapter in this thesis highlighted the presence of schizotypal symptoms in a sample of individuals recovering from an acute episode of psychosis. This chapter will investigate associations between these symptoms, particularly those in the anomalous experiences domain, and the experience of a visual illusion paradigm. This section will first summarise the literature in relation to the occurrence of anomalous experiences in psychosis, before outlining the aims of the current study.

Low-level anomalous experiences and perceptual disturbances are viewed as being at the core of psychosis (M. Shepherd, 1987). Cognitive models suggest that it is the interpretation of these experiences which leads to delusion formation and the development of full-blown psychotic symptoms (Garety et al., 2001). It is hypothesised that these phenomena may be caused by a cognitive dysfunction. The cognitive dysfunction itself is not necessarily seen as the primary cause of psychosis, but rather a final common pathway through which environmental and genetic influences may operate (Hemsley, 2005a).

The nature of the cognitive dysfunction underlying anomalous experiences is a matter of some debate (Frith, 1979; Hemsley, 1987; Knight, 1984; Maher, 1983; Venables, 1984). However, key to all explanations is the notion that in the early stages of psychosis, there is some degree of failure in automatic processing. Hemsley (2005a) specifies this as a "weakening of the influence of spatial and temporal regularities on perception" (p. 979). This causes a disruption in processing by the intrusion of material that would normally remain below awareness. This theory, combined with the ideas of Gray (1982), resulted in the development of the Gray-Hemsley model (J. A. Gray, 1995, 1998a, 1998b; J. A. Gray, Feldon, Rawlins, Hemsley, & Smith, 1991; Hemsley, 1992, 1993, 1998). A central tenet of this model is the existence of a *comparator* which brings together the current state of an individual's perceptual world with a predicted state (J. A. Gray, 1993; Hemsley, 2005a). The predicted state is based on previous experience of

regularities occurring between different stimuli (J. A. Gray, 1995). If a *mismatch* occurs between the current perceptual state and the predicted state, attention is allocated to stimuli thought to be responsible for that mismatch. In psychosis, the Gray-Hemsley model suggests that repeated and inappropriate mismatch signals occur, which result in the inappropriate allocation of attention to details of the environment which would not normally reach awareness. This is hypothesised to result in the occurrence of anomalous experiences. This idea has been further developed by Corlett, Honey, and Fletcher (2007) who highlight the importance of increased *prediction errors* (i.e. a mismatch between expected and actual events) in the development of psychotic symptoms.

Evidence to support the Gray-Hemsley model comes from a variety of sources, including studies investigating latent inhibition in psychosis (Lubow & Gewirtz, 1995). Latent inhibition (LI) is a learning process whereby if individuals are primed with a stimulus which has no consequence, the later formation of conditioned associations with that stimulus is inhibited, i.e. the stimulus loses associability due to non-reinforced pre-exposure (Lubow & Moore, 1959). LI is thought to be adaptive as it prevents responses being made to irrelevant stimuli and thus allows such stimuli to be filtered out of awareness. In acute psychosis however, LI has been shown to be impaired: prior exposure to a non-reinforced stimulus does not inhibit later association formation with that stimulus (Baruch, Hemsley, & Gray, 1988). This supports the notion that psychosis may be characterised by an impaired ability to integrate the regularities of previously presented material with current sensory input. Similar findings have also been shown using other contextual processing tasks in both clinical and non-clinical samples (Hemsley, 2005a, 2005b; Steel, Hemsley, & Pickering, 2002). It should be noted that low LI is a phenomenon paired with acute psychosis only. When LI experiments have been conducted with individuals following remission of psychotic symptoms, performance has been shown to be similar to that of non-clinical individuals (Baruch et al., 1988). This suggests that low LI, and indeed the Gray-Hemsley model, may apply specifically to state fluctuations in low-level anomalous experiences and perceptual disturbances and not to the more general syndrome of schizophrenic illness. However, it is unknown whether the reinstatement of LI following the remission of acute psychosis is a result of antipsychotic medication, or due to factors intrinsic to the evolution of schizophrenic illness (Barak & Weiner, 2007; N. S. Gray, Pilowsky, Gray, & Kerwin, 1995).

A strength of using LI as an investigative tool is that it provides a task in which individuals with psychosis perform better than non-clinical individuals. Thus, the outcome cannot be attributed to a generalised cognitive deficit (L. J. Chapman & Chapman, 1973; Hemsley, 2005b). Rather, the awareness of redundant information that is hypothesised to occur in psychosis, suggests that there is an increase in cognitive activity. However, this is problematic due to issues of limited capacity. Thus, increased activity and lack of inhibition in psychosis are likely to reduce the efficiency of processing. This fits with the early ideas of Bleuler (1950) who described sufferers of schizophrenia as "flooded with an undifferentiated mass of incoming sensory data".

One difficulty with the Gray-Hemsley model acknowledged by the authors (Hemsley, 2005a) is that of "mapping constructs that have been generated to explain task performance, on to experiential phenomena" (p. 978). Indeed, although the LI paradigm supports the notion of impaired automatic processing in psychosis, it does not directly explain the occurrence of perceptual disturbance. One paradigm which could be employed with this aim in mind is the McCollough Effect (ME; McCollough, 1965). The ME is a visual illusion defined as an after-effect and involves the perception of colour when no colour is in fact present. After-effects are illusions which require a period of adaptation to produce them (Gregory, 1998). To produce an ME, an individual is alternately presented with a magenta vertical grating and a green horizontal grating for a few minutes. After this period of adaptation, an achromatic vertical grating appears green and an achromatic horizontal grating appears pink. The strength of this phantom colour is then assessed using a visual analogue scale, or by assessing the orientation over which the illusion can be perceived: the further away from 0°, the stronger the effect. Although no large scale population studies have been conducted, the ME is considered to be a phenomenon of "normal" visual perception. On average, over 90% of participants tested in experimental studies perceive the effect (Byth, McMahon, & King, 2000; Logue & Byth, 1993; McCollough, 1965).

Whilst the presence of the ME is considered "normal", individual differences have been shown to occur in ME strength and have been used to investigate potential underlying mechanisms responsible for the effect (e.g. Byth, Logue, Bell, Best, & King, 1992; Logue & Byth, 1993; Shute, 1979). The exact physiology and function of the ME is

unknown (Allan & Siegel, 1993; Humphrey, 1998; Skowbo, Timney, Gentry, & Morant, 1975). However, it is hypothesised that the ME is a low-level visual phenomenon with some top-down influence (J. Barnes et al., 1999), potentially occurring in area V1 (Humphrey & Goodale, 1998), and is reflective of the ability of the visual system to recalibrate itself in the face of perceptual anomalies (Dodwell & Humphrey, 1990, 1993). Adaptation level theory (Helson, 1964) suggests that one of the main challenges in visual perception is matching the internal representation of the world (i.e. what we expect to perceive based on past experience) to external properties (i.e. what we actually perceive). In making psychophysical judgements it is hypothesised that the observer sets up an implicit scale based on the statistical properties of the set of objects being judged. There is a neutral point or *adaptation level* in relation to which perceptual judgements are made (Helson, 1964). Some external properties are statistical in nature and reflect properties of the world that occur in the long run. For example, the normal correlation of colour and orientation is zero. It is deemed necessary to keep such elementary dimensions of perception separate in order to make different types of learning possible (Barlow, 1990). If such long-term perceptual rules are violated, for example during a period of adaptation, a discrepancy is detected. In response to this, through an *error correction device* (ECD; Andrews, 1964), the visual system recalibrates in order to reduce the discrepancy. Generally speaking, this results in an inverse transformation on its input.

During the ME adaptation phase, colour and orientation are highly correlated (i.e. green always occurs with the horizontal grating; and magenta always occurs with the vertical grating). This violates the normal zero correlation between orientation and colour. In response to this, the visual system recalibrates and de-correlates colour and orientation (Dodwell & Humphrey, 1990). As a result of this, the inverse colour to that which was paired with a particular orientation in the adaptation phase, is perceived upon presentation of the achromatic grating of that orientation. Thus, pink is perceived upon presentation of the achromatic horizontal grating, and green is perceived upon presentation of the achromatic vertical grating.

The idea of an ECD in perception has many similarities to that of Gray and Hemsley's comparator (J. A. Gray, 1982). Thus, it could be hypothesised that the ME would be altered in acute psychosis in the same way as LI has been shown to be affected.

Following adaptation and on presentation of the achromatic test stimulus, a large mismatch occurs between the current perceptual state and the predicted state. If individuals with psychosis have an increased sensitivity to mismatches and are more prone to prediction errors, they may experience a stronger ME than a non-clinical sample. A single case study conducted by Shute (1979) showed that an individual with schizophrenia exhibited a particularly strong ME during a florid psychotic episode. This effect was barely measurable after administration of antipsychotic medication. This is similar to the state dependent effect of LI in psychosis. Moreover, amphetamines have been shown to increase ME strength in a non-clinical population, suggesting that dopaminergic processes may play a role in the effect (Byth et al., 1992; Shute, 1979). However, no study has yet examined ME strength in a large sample of individuals with psychosis.

3.1.1 Aims of the Study

The aim of the current study is to examine the notion that the ME can be used to assess anomalies in automatic processing in individuals with psychosis. As previous studies have specifically linked such impairments with psychotic states, it is hypothesised that increased ME strength will be specifically associated with the presence and severity of low-level positive psychotic symptoms (i.e. anomalous experiences and paranoia), but not with other types of psychosis-related symptomatology (i.e. social anxiety, negative symptoms). Moreover, in line with the single case study by Shute (1979), it is hypothesised that antipsychotic medication will reduce ME strength. In addition to these hypotheses, the relationship between the ME and other aspects of neuropsychological functioning will be investigated. It is predicted that ME strength will be independent from more generic aspects of cognitive functioning.

3.2 RESEARCH HYPOTHESES

1. Increased ME strength will be specifically associated with the presence and severity of low-level positive psychotic symptoms (i.e. anomalous experiences and paranoia) but not with other types of psychosis-related symptomatology (i.e. social anxiety).

2. Participants receiving higher levels of antipsychotic medication will experience weaker MEs.

3. ME strength will be independent from more generic aspects of cognitive functioning.

3.3 METHODOLOGY

Similar methodology to that outlined in the previous chapter (section 2.4) was used in this study. A subsample of the participants from the clinical sample outlined in the previous study also took part in this study.

3.3.1 Design

A sample of patients in recovery from acute psychosis was surveyed in a cross-sectional design. All participants completed a range of self-report questionnaires and clinician-administered assessments. Participants also completed the McCollough Effect task and other neuropsychological assessments.

3.3.2 Participants

3.3.2.1 Sample description

A sample of 103 individuals with affective or non-affective psychosis were recruited from secondary mental health services in the East Anglia region of the UK and gave written consent to take part in the study (as outlined in Study 1, section 2.4). All participants had been diagnosed with psychosis in the last eight years but were not currently experiencing an acute episode. After assessment with the Diagnostic Interview for Psychosis (Castle et al., 2006), 70% of the sample met criteria for non-affective psychosis (predominantly schizophrenia), and 30% for affective psychosis (predominantly bipolar disorder). The mean age of the sample was 26.6 years (*SD* = 5.6). Seventy-three per cent were male and 27% were female.

3.3.2.2 Sample size and power analysis

In order to examine the relationship between ME strength and psychopathology within the group, a power analysis calculation revealed that to achieve 80% power with a significance level of .05 and an estimated small critical effect size of .30, a minimum sample size of 85 participants was required (Cohen, 1988). Therefore the study was adequately powered.

3.3.3 Measures and Materials

3.3.3.1 Schizotypal Symptoms Inventory (SSI)

As individuals in the sample were not currently experiencing an acute episode of psychosis, the brief Schizotypal Symptoms Inventory was administered to assess residual symptoms of psychosis. Moreover, as anomalies in automatic processing have been linked with low-level anomalous experiences and perceptual disturbances, rather than full-blown psychotic symptoms, it was thought that the SSI may be more sensitive

in detecting these phenomena than a more traditional psychosis assessment tool. The SSI has been described in detail in the previous chapter and as such a description will not be repeated here. Total and subscale scores were used in the analyses.

3.3.3.2 Scale for the Assessment of Positive Symptoms (SAPS; Andreasen, 1984)

The presence of any acute psychotic symptoms was assessed using the SAPS, a 35-item semistructured interview designed to assess the presence of positive psychotic symptoms. As well as the total scale score, a subscale score was calculated for each participant reflecting core psychotic symptoms (auditory hallucinations, voices commenting, voices conversing, somatic hallucinations, delusions of reference, delusions of being controlled, and delusions of mind reading). This subscale was taken from a factor analysis of the SAPS (Peralta & Cuesta, 1999) and thought to reflect the core symptoms of psychosis that might be attributable to impaired automatic processing.

3.3.3.3 Client Service Receipt Inventory (CSRI; Beecham & Knapp, 1992)

The CSRI is a research tool designed to collect information about the use of health and social care services. This was used to collect data from participants about current medication levels. Antipsychotic medications were converted into chlorpromazine equivalence so that they could be compared between individuals (Woods, 2003).

3.3.3.4 McCollough Effect paradigm (ME; McCollough, 1965)

The ME paradigm was presented on a computer screen. Participants were initially presented with adaptation stimuli for 4 minutes. These consisted of two gratings alternating at 5-second intervals with a 1-second interval between presentations. One grating was composed of vertical black stripes on a green background (0,255,0 on the RGB scale) and the other grating was composed of horizontal black stripes on a magenta background (255,0,255 on the RGB scale). See Figure 3.1 for examples of the adaptation stimuli. Following the adaptation phase, participants were shown an achromatic test stimulus, consisting of black and white horizontal and vertical gratings

(see Figure 3.2), and were asked to give a magnitude estimate of any phantom colour they perceived. This estimate was given using a visual analogue scale (VAS) with ratings from 0-100. Higher ratings correspond to stronger effects (i.e. 0 equal to no colour and 100 equal to colours observed in the adaptation period).

In addition to a VAS rating of effect strength, the test stimulus was presented over a range of orientations from 0-180° at 5° intervals for 5 seconds each. Previous research has shown that the ME is strongest at 0° and cannot usually be seen at 45° intervals (McCollough, 1965). However, the effect has been shown to reappear when the test stimulus is viewed at 90° and 180°. Thus, the orientation test in the current study provided four additional measures of effect strength (0-45°, 45-90°, 90-135°, and 135-180°). An average of these four measurements was then calculated to provide an orientation range between 0-45° over which the ME could be perceived. Higher orientation ranges were assumed to reflect stronger effects.

All stimuli in the paradigm subtended 8.8 degrees by 8.8 degrees of visual angle at a viewing distance of 60cm. Gratings in both the adaptation and test stimuli had a spatial frequency of 2.7 cycles per degree and were of high contrast. All stimuli were displayed in a central position on a 15" laptop computer screen using Microsoft PowerPoint.

3.3.3.5 Cambridge Neuropsychological Automated Test Battery (CANTAB; Sahakian & Owen, 1992)

In order to examine the ME in relation to other aspects of neuropsychological performance, participants completed selected tasks from the CANTAB. The Intra-extra Dimensional Set Shifting (IED) task is a test of rule acquisition and reversal and assesses executive function. Performance was measured by the number of errors participants made on the task, with increased errors reflecting poorer performance. The Paired Associate Learning (PAL) task assesses episodic memory and associative learning. Performance on the PAL task was measured by the number of trials it took for participants to learn associations, with an increased number of trials reflecting poorer performance. The Rapid Visual Information Processing (RVP) task assesses sustained attention. Performance on the RVP task is measured by a sensitivity score, ranging from 0 (*poor*) to 1 (*good*), and reflecting a participant's accuracy at detecting the target

sequence. Finally, the Spatial Working Memory (SWM) task assesses working memory abilities. Performance on the SWM task was measured by the number of errors participants made, with increased errors reflecting poorer performance.

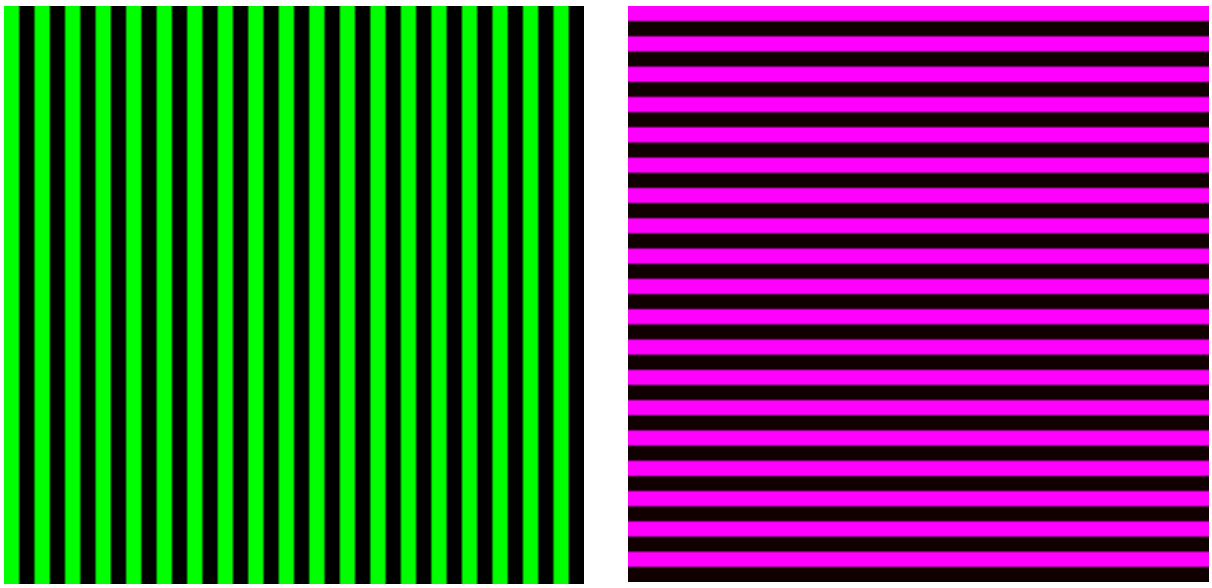


Figure 3.1

McCollough Effect adaptation stimuli

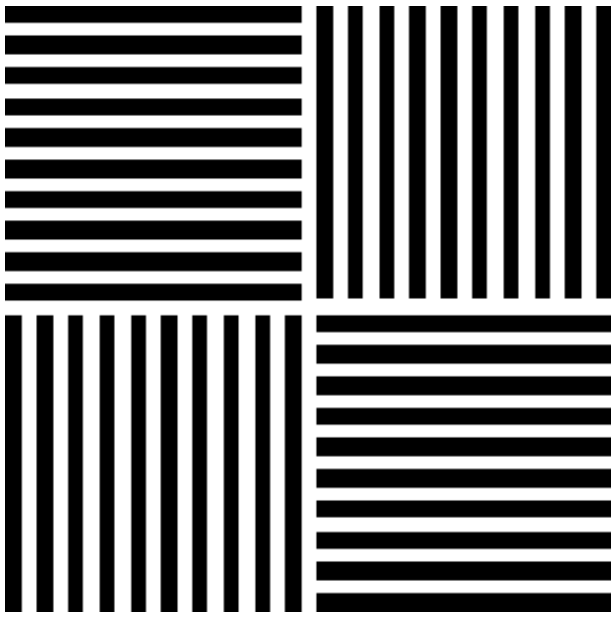


Figure 3.2

Test stimulus for the McCollough Effect task

3.3.4 Procedure

A similar procedure to that outlined in the previous study was used in this study. All measures were administered by trained researchers who met regularly to maintain reliability of procedures and ratings. Self-report measures were completed independently by participants. Symptom assessments were completed in a separate session to the ME and CANTAB tasks. Neuropsychological assessments were conducted in a quiet and controlled environment, predominantly in a clinical setting, to prevent distraction. Symptom and neuropsychological assessments were completed within two weeks of one another.

3.3.5 Data Analysis Plan

All data was analysed using SPSS for Windows, version 14 (SPSS, 2005). Data was screened and cleaned as outlined in the previous chapter (section 2.4.6.1). In the first stage of the analysis, descriptive statistics were calculated for all variables. Bivariate Pearson's product moment correlations were conducted to investigate relationships between ME strength, symptoms, medication levels, and neuropsychological performance. Differences between participants who perceived the effect and those who did not were investigated using independent samples t-tests.

3.4 RESULTS

3.4.1 Descriptive Data

Table 3.1 shows descriptive statistics for all variables in the group. The majority of variables had skewed distributions which were resistant to normalising transformation procedures. Therefore, parametric and non-parametric analyses were conducted. However, both types of analyses revealed the same findings and thus parametric analyses are reported.

Table 3.1

Descriptive Statistics for all Study Variables

	N	Min-Max	Mean (*SD*)	Skewness (*SE*)
Schizotypal Symptoms				
- Total	103	0-80	17.64 (15.41)	1.55 (0.24)
- Social Anxiety	103	0-24	8.09 (6.51)	0.61 (0.24)
- Paranoia	103	0-24	5.45 (6.23)	1.35 (0.24)
- Anomalous Experiences	103	0-32	4.11 (5.74)	2.39 (0.24)
SAPS				
- Total	103	0-48	14.02 (12.51)	0.74 (0.24)
- Core Psychotic Symptoms	103	0-25	4.99 (6.19)	1.44 (0.24)
Antipsychotic Medication Dose (mg)	77	0-726.67	216.26 (169.82)	0.54 (0.27)
McCollough Effect				
- Magenta estimate	103	0-100	7.68 (14.25)	3.51 (0.24)
- Green estimate	103	0-100	11.68 (17.17)	2.15 (0.24)
- Orientation range (°)	102	0-45	14.04 (13.72)	0.34 (0.24)
Intra-Extra Dimensional Set Shift (number of errors)	103	6-201	35.85 (39.52)	2.63 (0.24)
Paired Associate Learning (number of trials)	102	1-26	12.65 (5.38)	0.72 (0.24)
Spatial Working Memory (number of errors)	103	0-76	32.47 (20.75)	0.19 (0.24)
Rapid Visual Processing (accuracy)	96	0-0.97	0.83 (0.18)	-4.01 (0.25)

Note. Descriptive statistics for ME are for the whole sample, including participants who did not perceive the effect

3.4.2 Measuring the ME in a Sample with Psychosis

Magnitude estimation was significantly correlated with the orientation range over which the effect was perceived (magenta: $r(102) = .41$, $p < .001$; green: $r(102) = .51$, $p < .001$). This suggests that both measurements were tapping the same concept, i.e. ME strength. From this point forward, orientation range will be used as the measure of ME strength as this measure is unlikely to be affected by extreme responding in the same way as visual analogue scales. There was no significant difference in ME strength between individuals with affective and non-affective psychosis, $t(97) = 0.90$, $p = .37$.

3.4.3 ME and Medication Effects

The ME was experienced by 63% of individuals in the sample (mean orientation range = 22.37°, *SD* = 10.61°). Thus, 37% did not experience the effect at all. This is unusual when considering the fact that the effect is thought to be a phenomenon of normal visual perception. Current medication levels were known for a subsample of participants (n = 77). Those who could perceive the ME were receiving a significantly lower dose of antipsychotic medication than individuals who could not perceive the effect: 178mg vs. 279mg; $t(75) = 2.62$, $p = .01$. Moreover, there was a significant negative association between antipsychotic dose and ME strength, such that individuals receiving higher doses of antipsychotic medication perceived weaker MEs, $r(76) = -.28$, $p = .01$.

3.4.4 Relationships with Other Variables

ME strength was not associated with symptom scores on the SAPS (Total: $r(102) = .10$, $p = .32$; Core Psychotic Symptoms: $r(102) = .08$, $p = .44$). However, there was a correlation between ME strength and schizotypal symptoms. ME strength correlated with Anomalous Experiences, $r(102) = .23$, $p = .02$; and Paranoia $r(102) = .21$, $p = .03$; but not with Social Anxiety $r(102) = .14$, $p = .15$. Thus, those individuals reporting higher levels of Anomalous Experiences and Paranoia also displayed stronger MEs.

Bivariate correlations showed no association between ME strength and scores on other neuropsychological paradigms (IED: $r(102) = -.02$, $p = .82$; PAL: $r(101) = -.07$, $p = .48$;

SWM: $r(102) = -.02$, $p = .84$; RVP: $r(95) = .02$, $p = .88$). Thus the ME appears to be independent from other domains of neuropsychological functioning.

3.5 DISCUSSION

This chapter has examined the experience of the ME in a large group of individuals with psychotic illness. Previous clinical studies conducted in this area have focused on single cases (e.g. Shute, 1979) and thus this study is somewhat novel in its design. This section will review the results of the current study in relation to the research hypotheses outlined at the beginning of this chapter. The findings will be discussed in relation to the current literature and the clinical implications will be examined. Following on from this, potential weaknesses of the study will be outlined, as will possibilities for future research.

3.5.1 Overview of Findings

The ME was experienced by 63% of the sample and was significantly associated with the presence of anomalous and paranoid schizotypal symptoms. That is, individuals reporting more of these phenomena also experienced significantly stronger MEs. However, this association did not occur for symptoms of social anxiety, or for acute symptoms of psychosis as measured by the SAPS. This suggests that the relationship between ME strength and symptomatology may be specific to core low-level positive psychotic phenomena (e.g. perceptual disturbance and anomalous experience). Moreover, there appears to be an effect of medication on ME strength, with individuals taking higher levels of antipsychotic medication also displaying weaker effects. The group of 37% participants who could not perceive the ME were found to be taking significantly higher levels of medication than the 63% who could. In addition to these findings, ME strength was shown to be independent from other domains of cognitive functioning, suggesting that the findings of the current study cannot be attributed to general cognitive factors.

3.5.2 Relevance to the Literature

In light of the above findings it appears that the ME may be reflective of an anomaly in automatic processing, akin to that described in the Gray-Hemsley model, and which is specific to state low-level positive psychotic symptomatology. The increased strength of the ME in individuals who report more positive schizotypal symptoms suggests that individuals with psychosis may display an increased sensitivity to discrepancies between what is actually perceived and what is expected based on past experience. Moreover, unlike tasks which have previously been used to test the Gray-Hemsley model (e.g. latent inhibition); the ME is more directly relevant to the presence of perceptual aberration.

The finding that MEs are not present in individuals taking high levels of antipsychotic medication suggests that the effect may be blocked in some way. The exact process by which this occurs can only be speculated. However, it is known that there is heightened dopamine transmission in individuals with psychosis (Davis et al., 1991; Seeman, 1987; Seeman & Kapur, 2000). Excess dopamine in the ventral striatum is thought to be responsible for anomalous experiences due to its role in the aberrant attribution of salience, and in turn the direction of attention, to irrelevant stimuli (Kapur, 2003). Antipsychotic medication is hypothesised to be effective in reducing psychotic symptoms due to a blockade of this dopamine transmission (Kapur & Mamo, 2004). However, experimental studies have shown that in normal circumstances, dopamine is released in the face of a discrepancy between what is predicted to occur in the environment and what actually occurs (Berridge & Robinson, 1998; Schultz, 1999). It is hypothesised that this allows attention to be directed to new stimuli to aid learning. In this study it is suggested that the ME is the result of a process which attempts to respond to a discrepancy between predicted and actual perception (Dodwell & Humphrey, 1990). A lack of dopamine transmission in individuals taking high levels of antipsychotic medication may therefore disrupt the process by which the ME occurs, thus resulting in no effect being experienced. Conversely, via the same process, an excess of dopamine may result in a stronger effect being experienced.

Other studies investigating perceptual processing in psychosis have suggested that clinical populations are less susceptible to visual illusions as a result of weak contextual suppression (Dakin, Carlin, & Hemsley, 2005). Dakin et al. showed that individuals with schizophrenia were more accurate in a contrast discrimination task than controls, and less prone to the "contrast-contrast" illusion – a paradigm where a target appears lower in contrast when presented within a high-contrast surround than in isolation (Chubb, Sperling, & Solomon, 1989). With this in mind, it could be argued that ME strength would be weaker in individuals with psychosis, due to reduced influence of the adaptation phase (i.e. context) on the perception of colour in the test stimulus. However, if the contrast detection task is viewed in terms of individuals with psychosis being more sensitive than controls to mismatch, and thus better able to detect subtle differences in contrast; then it seems reasonable and consistent that they would also experience stronger MEs. Moreover, in line with Dakin et al. (2005), the finding that individuals with increased levels of psychotic-like phenomena "over-perform" on the ME paradigm may help to reveal underlying mechanisms of the disorder. This is in contrast to tasks on which individuals with psychosis show a deficit, whereby performance is usually explained in the context of a generalised impairment. In future studies it would be interesting to compare ME strength with performance on other visual illusion paradigms and tasks relating to contextual processing.

3.5.3 Weaknesses of the Current Study

There are a number of limitations to the current study which should be taken into account when interpreting the findings. First, the study lacks a non-clinical control group. As such it is unknown whether MEs experienced by individuals with psychosis are stronger than those experienced in the general population; or whether the lack of ME in 37% of participants was merely a result of the way in which the paradigm was administered in this study. However, in a study of ME strength in a non-clinical student sample using similar methodology, over 90% of participants perceived the effect (Thompson & Hodgekins, 2004). An analogous pattern of association between ME strength and schizotypal symptoms was also found in this non-clinical (and thus medication naïve) sample, with weaker MEs occurring in low-schizotypy individuals (Thompson & Hodgekins, 2004).

Second, although associations were found between schizotypal symptoms and ME strength in the current study, no associations were found between psychotic symptoms as rated by the SAPS. However, it must be remembered that this group were not currently experiencing an acute episode of psychosis and thus levels of positive psychotic symptoms were relatively low. It could also be argued that the SSI is a more sensitive measure of low-level psychotic symptoms, assessing phenomena similar to those which have previously been associated with anomalies in automatic processing. Thus it is more likely that associations between ME strength and symptoms would be found when using this scale, as opposed to the SAPS. In order to assess the relationship between ME strength and full-blown psychotic symptoms, this study would need to be replicated in a sample with acute psychosis.

It could be argued that there are problems with using prescribed medication dosage to examine associations between medication use and other variables, given the high level of mediation non- or partial-compliance in psychotic disorders (e.g. Coldham et al., 2002). In order to gain more accurate estimates of medication status, plasma levels of antipsychotic medication would ideally have been taken. However, this was beyond the scope of the current study. Moreover, the relationship between antipsychotic medication and ME strength may be confounded by other variables, including illness severity and duration. Thus, it may be the case that a subcategory of chronic psychosis patients (who also take higher levels of medication) have a reduced likelihood of perceiving the ME than less chronic individuals. Further research is therefore needed to unpick the nature of these relationships. Indeed, as this study is correlational in design, conclusions cannot be made with respect to causality, or the direction of relationships between ME perception, antipsychotic medication, and schizotypal symptoms. Future research in this area should focus on investigating fluctuations in ME strength over the course of a psychotic episode, both before and after the administration of antipsychotic medication. Based on the current findings, it would be predicted that perception of the ME would be strongest during an acute episode when anomalous experiences were at their most florid, and then gradually decline following the remission of these experiences, potentially after the administration of antipsychotic medication.

3.5.4 Summary

Despite the above limitations, the findings of this study have some interesting clinical implications and add a promising dimension to the search for a basic cognitive dysfunction underlying the positive symptoms of psychosis. The results also support those of other studies which have demonstrated anomalies in both low-level and top-down visual processes in psychosis (Giersch & Rhein, 2008; Vohs et al., 2008). Moreover, the findings concur with suggestions that such anomalies may be specific to clinical state (Johannesen, Bodkins, O'Donnell, Shekhar, & Hetrick, 2008). In addition to previous research, the current study also outlines how aberrant perception may be linked to the symptoms of psychosis. However, it must be remembered that a basic cognitive dysfunction resulting in anomalous experiences is only part of the story. Interpretation is key to the development of such experiences into later psychotic illness. This is likely to be a largely psychological process. In order to develop effective therapeutic approaches, future research must focus on examining how these two processes interact. The association between psychotic-like experiences and emotional and psychological variables will be the focus of the next chapter of this thesis.

CHAPTER FOUR:
STUDY THREE: RELATIONSHIPS BETWEEN SCHIZOTYPAL SYMPTOMS, EMOTION, AND SCHEMA VARIABLES IN A NON-CLINICAL SAMPLE

4.1 RATIONALE AND CONTEXT OF THE STUDY

4.1.1 Overview

Thus far, this thesis has demonstrated that schizotypal symptoms occur both in non-clinical populations and in clinical populations recovering from psychosis. This study examines the relationship between these symptoms and emotional and psychological variables in a non-clinical sample.

Findings from the previous chapter support theories suggesting that anomalous experiences may be the product of an underlying cognitive disturbance (J. A. Gray et al., 1991; Hemsley, 1998). The cognitive model of psychosis argues that emotional and psychological variables are particularly important in the appraisal of anomalous experiences arising from such a disturbance, and thus in the development and maintenance of psychotic symptoms (Garety et al., 2001). In line with the continuum hypothesis, it is likely that schizotypal experiences will also be accompanied by a range of emotional and psychological factors influencing their development and maintenance. However, there may be differential relationships between individual schizotypal symptom types and emotional and psychological variables. Indeed, interpersonal phenomena (e.g. social anxiety and paranoia) could be argued to have a more emotional and psychological basis than anomalous experiences, which have been argued to be more organic in nature.

Although numerous studies have used a single-symptom approach (e.g. Freeman, Garety et al., 2005) to investigate the factors associated with specific psychotic phenomena (e.g. paranoia), little research has been conducted to investigate the differential associations between discrete symptom types and potential associated variables. The SSI is advantageous in this respect as it assesses a range of low-level psychotic symptoms simultaneously. Moreover, investigating the relationships between

schizotypal symptoms and emotional and psychological variables in a non-clinical population may inform more specifically upon the factors and processes underlying the development of such phenomena, without the confounding effects of the disease process (Raine & Lencz, 1995). Furthermore, if relationships between schizotypal symptoms and emotional and psychological variables in the non-clinical sample mirror those found in clinical samples, this could be taken as evidence to support the psychosis continuum.

This chapter first reviews the literature regarding associations between positive psychotic symptoms and emotional and psychological processes, before investigating the relationships between different types of schizotypal symptoms and emotional and psychological variables in a non-clinical population.

4.1.2 Psychosis and Emotional and Psychological Processes

Birchwood (2003) argues that emotional dysfunction is pervasive in psychosis, and may occur pre- and post-onset, as well as during the psychotic episode itself. Several routes to emotional dysfunction in psychosis have been postulated. Birchwood (2003) proposes three overlapping pathways: first, that emotional dysfunction is intrinsic to psychosis; second that emotional disorders are a psychological reaction to psychosis; and third, that emotional disorders arise from shared risk factors with psychosis. It is likely that all of these pathways are valid, accounting for different levels of emotional disturbance at different stages of psychotic illness. However, the finding that emotional disturbance occurs prior to the onset of psychosis (Häfner, Löffler, Maurer, Hambrecht, & an der Heiden, 1999; McGlashan, 1996), suggests that it may have a direct impact on the development of psychotic symptoms. Moreover, an emotional response to psychotic symptoms may create a vicious cycle, thus maintaining the presence of psychotic phenomena (Fowler, Freeman, Steel et al., 2006).

In line with the cognitive model of psychosis, different aspects of emotion and self and other evaluation may be associated with different types of psychotic symptoms. Evidence for this will now be reviewed.

4.1.2.1 Anxiety

Increased levels of anxiety have been found to be common before the onset of psychotic symptoms (Tien & Eaton, 1992) and in the prodromal stages of the disorder (Birchwood et al., 1992; McGlashan, 1996). Anxiety has also been shown to be predictive of transition to psychotic illness. A study by Weiser et al. (2001) showed that adolescents with neurotic disorders were more likely to develop psychosis than those without neurotic disorders. These findings have since been replicated by other researchers (P. Jones, Rodgers, Murray, & Marmot, 1994; P. Miller, Byrne et al., 2002). Following the onset of psychosis, anxiety has been highlighted as a common co-morbid feature of acute psychotic episodes, with studies suggesting rates of around 40-50% (Argyle, 1990; Cosoff & Hafner, 1998; Turnbull & Bebbington, 2001). Moreover, Voges and Addington (2005) found that 31% of a first-episode psychosis sample met criteria for social phobia.

Anxiety is thought to play an important role in both the development and maintenance of psychotic symptoms. The notion that anxiety may be partially responsible for the occurrence of hallucinations and delusions is an idea supported by J. A. Gray et al. (1991), whose neuropsychological model implicates arousal as a central process in the onset of psychosis. Increased arousal prior to symptom formation is thought to direct attention towards irrelevant stimuli and trigger a search for meaning as to why such stimuli feel personally meaningful. The importance of arousal also fits with the stress-vulnerability model of psychosis (Nuechterlein & Dawson, 1984) and findings that symptom occurrence and psychotic relapse are often preceded by stressful life events and corresponding increases in arousal (Bebbington et al., 1993; Malla, Cortese, Shaw, & Ginsberg, 1990). In addition, studies have demonstrated that individuals with psychosis have greater stress sensitivities (Myin-Germeys, Delespaul, & van Os, 2005).

Emotional processes are not thought to be directly responsible for causing hallucinations (Freeman & Garety, 2003). However, it is argued that anxiety may provide a trigger for the hypothesised dysfunction in cognitive processes which is thought to lead to the occurrence of hallucinations (Morrison, 1998; Slade, 1976; Tien & Eaton, 1992). The exact physiological process by which this occurs has yet to be

clearly defined but it has been suggested that anxiety may result in failures in self-monitoring (Frith, 1992). Morrison (2001) further elaborates on the maintaining role of anxiety on hallucinatory experience, suggesting that hallucinations occur as a result of intrusions into awareness, governed by anxiety. The misinterpretation of these intrusions as external threats produces a further emotional response which leads to more intrusions, creating a vicious cycle. In addition, it is thought that anxiety may have an impact on the content and interpretation of auditory hallucinations, perhaps resulting in a threatening theme, e.g. "we're after you" (Fowler, 2000a; Garety et al., 2001; Morrison, 2001). Thus, it appears that there is a role for anxiety in the development, content and maintenance of hallucinations.

As well as hallucinations, Freeman and colleagues suggest that anxiety may be important in the development and content of delusions, particularly paranoia and persecutory beliefs (Freeman, 2007; Freeman & Garety, 2003). Anxiety and interpersonal sensitivity – a specific type of anxiety concerning social interaction – have been shown to predict the presence of paranoia in both clinical and non-clinical populations (Freeman, Dunn et al., 2005; Freeman, Garety et al., 2005), with paranoid thoughts (e.g. "people are out to get me") often building upon common interpersonal worries and anxieties (e.g. "people are looking at me"). It is hypothesised that delusions, particularly those of a persecutory nature, are threat beliefs maintained by similar psychological processes traditionally associated with emotional disorders (Freeman et al., 2002). Indeed, similar processes are thought to underlie both anxiety and paranoia, including the anticipation of threat or danger, and the use of safety behaviours to prevent the feared event from taking place (Freeman, 2007; Salkovskis, 1991). The majority of individuals with persecutory delusions have also been shown to have a worry thinking style, even about matters unrelated to paranoia (Freeman & Garety, 1999). In addition to this, increased emotional arousal in response to paranoia is hypothesised to induce an attentional bias, resulting in individuals becoming hyperaware of their surroundings. This increases vigilance for potentially threatening stimuli, which may in turn maintain persecutory beliefs. Evidence in support of this view comes from studies highlighting an attentional bias towards threat stimuli in individuals with psychosis (M. J. Green, Williams, & Davidson, 2003).

The evidence reviewed suggests that anxiety may play an important and sometimes causal role in the development and maintenance of psychotic symptoms, namely hallucinations and delusions. Individuals with psychosis may be more vulnerable to fluctuations in anxiety in response to daily hassles and major life events, which could in turn trigger symptom formation and influence its content and accompanying interpretation. It has been argued that anxiety is simply a consequence of psychotic symptoms (J. P. Chapman, 1966). Indeed, anxiety problems often remain even after the remission of acute psychosis (Voges & Addington, 2005). However, whilst it is accepted that psychotic symptoms are accompanied by increases in anxiety, the finding of emotional disturbance prior to the onset of full symptoms suggests that the relationship is circular (Freeman & Garety, 2003). That is, changes in arousal trigger the onset of symptoms which then have an additional impact on anxiety, in turn increasing vulnerability for further symptom development.

4.1.2.2 Depression

As with anxiety, increased levels of depression are a common feature of psychosis, both before and after onset. A retrospective study by Häfner and colleagues demonstrated that 73% of patients with first-episode psychosis had experienced a prodromal phase lasting several years, which had begun with the occurrence of depressive and negative symptoms (Häfner et al., 1999). Furthermore, Bustamante, Maurer, Löffler, and Häfner (1994) suggest that 81% of the depression occurring in the early stages of psychosis begins an average of 4.3 years prior to diagnosis. Following onset, depression has been shown to be frequently co-morbid with schizophrenia (Siris, 1995), with around 45% of individuals experiencing depressed mood (Leff, Tress, & Edwards, 1988). A more recent study estimates an even higher prevalence of depression in psychosis at between 22-75%, depending on which assessment tools and criteria are used (Koreen et al., 1993).

In the same way as anxiety, it is thought that depression may impact upon the content of hallucinations and the way in which they are experienced and interpreted. Most of the research in this domain tends to focus on auditory hallucinations, with suggestions that depressed mood may lead to critical content of voices, e.g. “you’re useless” (Fowler, 2000b; Freeman & Garety, 2003; Morrison, 2001). Indeed, Smith et al. (2006) showed

that individuals who were more depressed, experienced voices with more negative content and were also more distressed by them. Thus, there is a suggestion that the content of hallucinations may reflect emotional concerns. However, causality is difficult to establish here as it is equally likely that distressing voices evoke an increased emotional response. For example, being called useless by a voice is likely to result in low mood. Chadwick and Birchwood (1994) conducted a study to investigate how beliefs about voices, as well as content, influenced mood. They found that emotional distress was most closely tied to beliefs about the voices, with increased depression being associated with beliefs that voices were malevolent. In 30% of cases, these beliefs occurred even if the voices were not negative in content. This suggests that mood may not just be a response to the occurrence of hallucinations, but rather play a more important role in their interpretation.

Depression is also argued to impact upon delusion formation by providing the individual with a feeling of social exclusion and a sense of being a target for others (Freeman & Garety, 2003). Aspects of depression, such as guilt, may also feature in the content of delusional beliefs. For example, a depressed individual may believe that they deserve to be persecuted because they are a bad person. This has been defined as "bad me" paranoia (Trower & Chadwick, 1995). Moreover, in a cross-sectional study by C. Green et al. (2006), individuals with paranoid beliefs who reported feeling subordinate and powerless in the face of persecution, were shown to suffer from higher levels of depression than individuals who felt able to exert some control over their situation. Causality in such studies is however still an issue. Depression may result in feelings of powerlessness and inferiority, thus influencing delusion content, or may also occur as an emotional response to delusional beliefs, particularly if they are negative in nature. Akin to findings for hallucinations, individuals who are more distressed by their delusions also experience higher levels of depression (Smith et al., 2006). As with anxiety, the relationship between depression and psychotic phenomena is thus likely to be circular.

Whilst depression appears to influence the content and interpretation of psychotic symptoms, it has been argued that anxiety may play a more central role than low mood in symptom development (Norman, Malla, Cortese, & Diaz, 1998). Indeed, some studies have found stronger links between depression and negative symptoms (e.g.

amotivation) rather than positive psychotic phenomena (Norman & Malla, 1991). Despite this, it has been hypothesised that depression in psychosis may induce a cognitive bias towards the processing of negatively connotated information, thus influencing and maintaining hallucinations and delusions (Guillem, Pampoulova, Stip, Lalonde, & Todorov, 2005). Moreover, it may be specific negative evaluative beliefs associated with depression, rather than low mood per se, which are important in the development of psychotic symptoms. These will be considered in the next section.

4.1.2.3 Self-esteem and schematic beliefs

Closely linked to depression, low self-esteem and negative self evaluation have been shown to be common and pervasive in psychosis, as well as in non-clinical samples who report psychotic-like symptoms (Bowins & Shugar, 1998; Combs & Penn, 2004; Freeman et al., 1998; Silverstone, 1991). In addition to a low baseline level of self-esteem, individuals with psychosis are also hypothesised to experience increased fluctuations in self-esteem over time (Thewissen, Bentall, Lecomte, van Os, & Myin-Germeys, 2008; Thewissen et al., 2007). This may be reflective of general emotional instability, or may be associated with fluctuations in symptoms. Low self-esteem in psychosis has been linked to increased illness severity and poorer outcome (Freeman et al., 1998). Moreover, whilst implicated in the development and maintenance of psychotic symptoms (Bentall et al., 1994; Garety et al., 2001; Smith et al., 2006), there is a consistent finding of low self-esteem in patients at varying phases of psychotic illness, even after symptom recovery (Gureje et al., 2004), suggesting that it may also be a reaction to the disorder itself. Indeed, the process of developing psychosis has significant implications for the development of negative self-concept (Birchwood & Iqbal, 1998; Laithwaite et al., 2007).

There are numerous problems in assessing self-esteem in psychosis, including how the construct should be defined and the selection of appropriate measurement tools. Whilst it is generally accepted that global self-esteem is reduced in psychosis, exactly which aspects of low self-esteem influence symptoms, if at all, is a matter under debate. Bentall and colleagues argue that psychotic symptoms, particularly persecutory delusions, develop in individuals with low self-esteem to prevent negative thoughts and feelings about the self from reaching consciousness (Bentall, Corcoran, Howard,

Blackwood, & Kinderman, 2001; Bentall et al., 1994). That is, individuals with psychosis have a tendency to make external personal attributions (i.e. blame other people) for negative events in order to protect themselves against underlying low self-esteem. Evidence to support this view comes from findings of externalising attribution biases in paranoid individuals (Krstev, Jackson, & Maude, 1999; Lyon, Kaney, & Bentall, 1994); and from studies highlighting reductions in self-esteem which immediately precede increases in paranoia (Thewissen et al., 2008). Moreover, intact self-esteem has been observed in some individuals with paranoia, even in cases with high depression (Candido & Romney, 1990). However, in the majority of cases, low self-esteem is directly related to paranoia and other positive symptoms of psychosis (Freeman & Garety, 2003). This would not be expected if paranoia served as an effective defence. Thus, an alternative view is that low self-esteem in psychosis occurs as a result of normal emotional processes and has a central, non-defensive role in the development of symptoms (Fowler, 2000a; Garety et al., 2001).

More recent research has focused specifically on the role of extreme negative self and other evaluation in psychosis, as opposed to global self-esteem. Indeed, increased negative self evaluation has been strongly associated with the presence of positive symptoms of psychosis (Barrowclough et al., 2003). Moreover, Freeman and Garety (2003) propose that psychotic symptoms are a direct reflection of pre-existing (and predominantly negative) beliefs about the self, world, and others. With this in mind, Fowler, Freeman, Smith et al. (2006) have developed a tool with the specific aim of assessing core schematic beliefs (both negative and positive) about self and others. The Brief Core Schema Scales (BCSS) allow a distinction to be made between extreme negative self evaluations (e.g. “I am bad”), hypothesised to be characteristic of psychosis; and generic self-esteem assessments which tend to measure the absence of positive evaluations of self (e.g. “Other people are more successful than me”).

Using the BCSS, individuals with psychosis report more extreme negative beliefs about self and others when compared to a non-clinical sample (Fowler, Freeman, Smith et al., 2006). These beliefs are hypothesised to develop via adaptation and social learning, potentially in response to early adverse experiences (Garety et al., 2001). The triggering of negative schematic beliefs (e.g. “I am vulnerable”, “others are dangerous”) may lead to the experience of psychotic symptoms due to their influence on the appraisal of

anomalous experiences or ambiguous social situations. Indeed, an individual who believes that they are weak and vulnerable to attack, and that others are hostile and dangerous, may be more likely to interpret a glance from a passer-by as a threat signal. Equally, negative schematic beliefs about the self may be reflected in the content of auditory hallucinations (Freeman & Garety, 2003). Thus, it is the accessing of these schemas, and not just emotional disturbance alone, that is proposed to be associated with psychotic symptoms (Garety et al., 2001). Smith et al. (2006) demonstrated that the occurrence of persecutory delusions was associated with negative evaluative beliefs about the self and others, even when controlling for current mood and self-esteem. Auditory hallucinations on the other hand, were found to be associated with higher levels of depression and lower self-esteem, but not with negative evaluative beliefs about self or others. However, more distressing hallucinations, and those with more negative content, were associated with increased negative beliefs about self.

The role of schemas in the development and maintenance of psychotic symptoms could be argued to date back to the work of Laing (1960; 1961). In *Self and Others*, Laing (1961) refers to the notion of an individual with psychosis feeling as though other people are against them: "…self attributes to others the intention to oust self from his position in the world, to displace and replace him" (p. 132). This is similar to what Fowler, Freeman, Smith et al. (2006) refer to as negative beliefs about others. Laing also refers to an individual with psychosis as feeling vulnerable and having an insecure position in the world: "what tortures him is his harrowing suspicion that he is of no importance to anyone" (p. 136). This is similar to what Fowler, Freeman, Smith et al. (2006) refer to as negative beliefs about self. A combination of beliefs that the external world is hostile and the self is weak and vulnerable creates a dangerous social position, which Laing (1961) describes as being "false and untenable" (p. 125). Such a position, combined with increased anxiety, may lead to the development of persecutory delusions and appears to be characteristic of individuals with psychosis. This is in line with research by Smith et al. (2006) outlined above.

Whilst closely related to the more generic constructs of low self-esteem and low mood, it appears that negative beliefs about self and others may have a specific role in the formation, content, and maintenance of psychotic symptoms. These schemas seem particularly important in the formation of delusions and paranoia, providing the

foundations for delusional beliefs to be built upon. They appear to play less of a role in the development of hallucinatory experiences, although may have an impact on the content and interpretation of hallucinations, as well as associated distress. Moreover, the presence of psychotic symptoms is likely to feed back into and confirm pre-existing schematic beliefs about self, thus creating a vicious cycle (Smith et al., 2006).

4.1.2.4 Summary

Research into the role of emotion and psychological processes in psychosis suggests that anxiety, depression, and evaluative beliefs about self and others have important and potentially differential roles in positive symptom formation and maintenance. Whilst the content of both delusions and hallucinations may often be a direct representation of the emotional state of the individual, the role for emotion and schematic beliefs in the initial development of psychotic symptoms is perhaps most clear for delusions, particularly those of a paranoid and persecutory nature. Indeed, negative beliefs about self and others have been shown to be important predictors of persecutory beliefs (Smith et al., 2006). This is in contrast to hallucinations, where emotion and psychological processes are thought to be more of a maintenance (as opposed to causal) factor. However, anxiety appears to be a core component in the development of all psychotic symptoms (Tien & Eaton, 1992), both influencing delusion formation and providing a trigger for mechanisms underlying hallucinatory experiences.

4.1.3 Aims of the Study

The introduction to this chapter has emphasised the importance of emotion and schematic beliefs about self and others in both influencing the development and shaping the content of psychotic symptoms. The aim of the current study is to examine differential associations between dimensions of positive schizotypal symptoms and emotional and psychological variables in a non-clinical population. Although previous studies have examined these associations in analogue populations (e.g. Freeman, Garety et al., 2005; Gracie et al., 2007), the current study has a specific focus on current low-level psychotic symptoms rather than more generic predispositions to psychosis. It will also examine differential associations between different symptom types simultaneously, rather than focusing on one symptom type in isolation.

4.2 RESEARCH HYPOTHESES

1. Social anxiety schizotypal symptoms will be associated with a lack of positive beliefs about self (i.e. low self-esteem), anxiety, and interpersonal sensitivity.

2. Paranoid schizotypal symptoms will be associated with negative beliefs about self and others, anxiety, and interpersonal sensitivity.

3. Anomalous schizotypal symptoms will be associated with anxiety but not schematic beliefs about self and others, or interpersonal sensitivity.

4.3 METHODOLOGY

The methodology for this study was the same as that outlined for the non-clinical sample in Study 1 (section 2.4). The same participants and procedures were used.

4.3.1 Design

A non-clinical sample of university students were surveyed in a cross-sectional design. All participants completed the Schizotypal Symptoms Inventory (SSI) and other measures via an internet survey, as described in Study 1 (section 2.4). A within-subjects correlational design was used to investigate the hypothesised relationships between schizotypal symptoms, emotion, and schema variables.

4.3.2 Participants

The same group of 808 non-clinical participants who were described in Study 1 (section 2.4.2.1) also took part in this study. The sample will not therefore be described again here.

4.3.2.1 Sample size and power analysis

Correlations. In order to examine the relationship between emotion, schema, and scores on the SSI, a power analysis calculation revealed that to achieve 90% power with a significance level of .05 and an estimated small to moderate critical effect size of .40, a minimum sample size of 62 participants was required (Cohen, 1988). Therefore the study was adequately powered.

Multiple regression. In order to examine whether emotion and schema predict SSI scores, a multiple regression analysis with seven predictors was conducted for each dimension of the SSI. A power analysis calculation revealed that to achieve 90% power with a significance level of .05, and an estimated small to moderate critical effect size of .15, a minimum sample size of 129 participants was required (Cohen, Cohen, West, & Aiken, 2003). Therefore the study was adequately powered.

4.3.3 Measures

4.3.3.1 Schizotypal Symptoms Inventory (SSI)

The SSI was completed to provide an index of current low-level psychotic symptomatology. The SSI has been described in previous chapters of this thesis and as such will not be repeated here. In this study, the brief version of the SSI was used. Total and subscale scores (i.e. Social Anxiety, Paranoia, Anomalous Experiences) were used in analyses.

4.3.3.2 Depression Anxiety Stress Scales (DASS; Lovibond & Lovibond, 1995)

The depression and anxiety scales of the DASS were used to assess current (i.e. past week) levels of emotional distress in the sample. The depression and anxiety scales of the DASS each consist of 14 items rated on a 4-point Likert scale from 0 (*did not apply to me at all*) to 3 (*applied to me very much*). Scores on the two scales therefore range from 0 to 42, with higher scores indicating higher levels of emotional distress. Lovibond and Lovibond (1995) report results of a comparison of the DASS with the Beck Anxiety

Inventory (BAI; Beck & Steer, 1987) and the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) in a non-clinical sample (N = 717). The DASS anxiety scale correlated at .81 with the BAI; and the DASS depression scale correlated at .74 with the BDI. The authors report Cronbach's alpha internal consistency coefficients of .91 and .81 for the DASS depression and anxiety scales respectively. A principal components analysis showed a good distinction between the depression and anxiety factors of the DASS. The scale has also recently been shown to be reliable and valid in a large UK non-clinical population (Crawford & Henry, 2003). The depression and anxiety scales of the DASS were chosen for use in the internet survey instead of the BDI and BAI as they are more appropriate for use in non-clinical populations than the latter, which are more clinical in nature. Furthermore, unlike the BDI and BAI, the DASS scales are not bound by copyright and are thus freely available for use in online research.

4.3.3.3 Brief Core Schema Scales (BCSS; Fowler, Freeman, Smith et al., 2006)

The BCSS consists of 24 items concerning beliefs about the self and others that are assessed on a 5-point rating scale (0-4). The scale was designed to create a quick and easy-to-use assessment of the type of extreme positive and negative evaluations of self and others that have been observed clinically to be typical of people with psychosis. Four scores are obtained: negative self (6 items, e.g. "I am bad"), positive self (6 items, e.g. "I am talented"), negative others (6 items, e.g. "Other people are hostile") and positive others (6 items, e.g. "Other people are accepting"). Individuals are initially asked to indicate in a dichotomous format whether they hold each belief. If they answer "Yes" to holding the belief, they are then asked to indicate their degree of belief conviction by choosing a number from 1 (*believe it slightly*) to 4 (*believe it totally*).

The BCSS has been shown to have good psychometric properties. A Principal Components Analysis of the items suggested an underlying dimensional structure reflecting independence between the different dimensions of self and other evaluation. Furthermore, Fowler, Freeman, Smith et al. (2006) report good internal consistency, with Cronbach's alpha internal consistency coefficients for the subscales ranging from .78 to .88. Moderate to strong associations were shown between the BCSS and the Rosenberg self-esteem questionnaire (Rosenberg, 1965). The BCSS was chosen for use

in this study as schema is argued to be important in the development of psychotic symptoms. For example, viewing the self as vulnerable and other people as bad may result in a tendency towards paranoid thinking and potential delusion formation. The BCSS was specifically designed for use in psychosis and differs from other measures of schema which tend to be more reflective of self-esteem, e.g. the Young Schema Questionnaire (Young, 1998).

4.3.3.4 Interpersonal Sensitivity Measure (IPSM; Boyce & Parker, 1989)

The IPSM is a 36-item self-report measure assessing excessive sensitivity to the interpersonal behaviour of others, to social feedback and to perceived or actual negative evaluation by others. The 36 items are completed on a 4-point Likert scale ranging from 1 (*very unlike me*) to 4 (*very like me*). The measure provides a total score and five subscale scores: Interpersonal Awareness (8 items), Need for Approval (8 items), Separation Anxiety (8 items), Timidity (8 items), and Fragile Inner Self (5 items). The IPSM has good psychometric properties, as reported by the authors (Boyce & Parker, 1989). Correlations between the five factors were shown to be low indicating their relative independence. Cronbach's alpha internal consistency coefficients in a non-clinical population are reported as .86 for the total scale and .55 to .76 for the individual subscales. Six-week test-retest reliability was reported as .70 for the total scale and .55 to .70 for the subscales.

The IPSM was selected for use in this study as the scale has been implicated in the assessment of the underlying mechanisms of social anxiety (Harb, Heimberg, Fresco, Schneier, & Liebowitz, 2002), and has also been liked with paranoia (Freeman, Dunn et al., 2005). Therefore, interpersonal sensitivity may also be involved in the development of schizotypal symptoms. This will be investigated in the current study. For the purpose of the current study, total IPSM scores were used in the analyses.

4.3.4 Procedure

The same procedure as outlined in Study 1 (section 2.4.5.1) was adopted for this study and as such will not be repeated here. The non-clinical sample completed the measures via an internet survey.

4.3.5 Data Analysis Plan

Initial screening and cleaning of the data has been described in Study 1 (section 2.4.6.1) and will not be repeated here. All data was analysed using SPSS for Windows, version 14 (SPSS, 2005).

In the first stage of the analysis, descriptive statistics were calculated for all variables. Bivariate correlations were conducted to investigate relationships between schizotypy and emotional and psychological variables. Simultaneous linear multiple regression analyses were then conducted to further determine which variables best predicted different types of schizotypy. Separate regression analyses were conducted for each subscale of the SSI. Schizotypy scores served as the dependent variable. Anxiety, depression, BCSS subscales, and IPSM scores served as the independent variables.

4.4 RESULTS

4.4.1 Descriptive Data

Table 4.1 provides descriptive data on all variables for the whole sample. As can be seen from the descriptive statistics, data on most variables are positively skewed. This could not be corrected by transforming the data. Therefore, Spearman's Rho (i.e. non-parametric) correlations were conducted to investigate associations between variables. Standard multiple regression techniques were used despite the skewness of the data.

Table 4.1

Descriptive Statistics for all Study Variables

	N	Min-Max	Mean (*SD*)	Skewness (*SE*)
Schizotypal Symptoms				
- Social Anxiety	808	0-24	4.41 (5.14)	1.46 (0.09)
- Paranoia	808	0-21	2.85 (3.63)	1.88 (0.09)
- Anomalous Experiences	808	0-23	2.28 (3.43)	2.45 (0.09)
- Total	808	0-53	9.54 (9.22)	1.46 (0.09)
DASS				
- Depression	779	0-42	10.07 (10.12)	1.39 (0.09)
- Anxiety	779	0-37	5.94 (6.55)	1.74 (0.09)
Brief Core Schema Scales				
- Negative Self	769	0-24	4.29 (4.31)	1.51 (0.09)
- Positive Self	769	1-24	13.02 (4.84)	-0.19 (0.09)
- Negative Other	769	0-24	4.89 (4.18)	1.04 (0.09)
- Positive Other	769	3-24	12.48 (3.83)	-0.06 (0.09)
Interpersonal Sensitivity	753	53-144	93.85 (15.49)	0.06 (0.09)

4.4.2 Relationships between Schizotypal Symptoms and Emotional and Psychological Variables

4.4.2.1 Bivariate correlations

Spearman's Rho correlations between schizotypy scores and emotional and psychological variables are shown in Table 4.2. With reference to Cohen (1988), in both groups, associations between schizotypy and emotional and psychological variables appear to be stronger for social anxiety and paranoid schizotypal symptoms than for anomalous experiences. This is supported using analyses cited by Meng, Rosenthal, and Rubin (1992) to compare correlated correlations. The association between anxiety and schizotypal symptoms was shown to be significantly smaller for anomalous experiences than that for social anxiety ($Z = 4.07$, $p < .001$) or paranoid schizotypal symptoms ($Z = 3.45$, $p < .001$). The latter two symptom types did not however differ in terms of the strength of their relationship with anxiety ($Z = 0.91$, $p = .18$). Similarly, the association between depression and schizotypal symptoms was shown to be significantly smaller for anomalous experiences than for social anxiety ($Z = 3.68$, $p < .001$) or paranoid schizotypal symptoms ($Z = 3.88$, $p < .001$). However, the strength of association between social anxiety and depression and paranoia and depression was the same. For interpersonal sensitivity, the relationship with schizotypy was significantly stronger for social anxiety when compared with both paranoia ($Z = 3.64$, $p < .001$) and anomalous experiences ($Z = 8.41$, $p < .001$). For negative other schema, the relationship with schizotypy was significantly stronger for paranoia when compared with both social anxiety ($Z = 2.52$, $p = 0.01$) and anomalous experiences ($Z = 5.74$, $p < .001$). The relationship between negative self schema and schizotypy was comparable for social anxiety and paranoia, but significantly smaller for anomalous experiences ($Z = 6.10$, $p < .001$; $Z = 6.44$, $p < .001$).

Table 4.2

Correlations between Schizotypal Symptoms and Anxiety, Depression, Schema, and Interpersonal Sensitivity

	Social Anxiety Schizotypy	Paranoid Schizotypy	Anomalous Schizotypy	Total Brief Schizotypy
Anxiety (*N* = 779)	.46***	.43***	.30***	.54***
Depression (*N* = 779)	.39***	.39***	.24***	.46***
Schema (*N* = 769)				
- Negative Self	.44***	.44***	.19***	.49***
- Positive Self	-.37***	-.25***	-.06	-.33***
- Negative Other	.27***	.36***	.13***	.34***
- Positive Other	-.27***	-.31***	-.13**	-.32***
Interpersonal Sensitivity (*N* = 753)	.52***	.40***	.18***	.52**

*p <.05, **p <.01, ***p <.001

4.4.2.2 Regression analyses

In order to further investigate the associations between schizotypal symptoms and emotional and psychological variables, simultaneous multiple regression analyses were conducted.

Social Anxiety schizotypal symptoms. A simultaneous multiple regression was undertaken with the Social Anxiety subscale of the Brief SSI as the dependent variable, and depression, anxiety, schema (BCSS), and interpersonal sensitivity as the explanatory variables. The results are shown in Table 4.3.

Table 4.3

Summary of Simultaneous Regression Analysis for Variables Predicting Social Anxiety Schizotypal Symptoms (N = 746)

Variable	*B*	*SE B*	β	sr^2 (unique)
Constant	-4.31	1.36		
Anxiety	0.16	0.03	.20***	.02
Depression	-0.01	0.02	-.01	.00004
Negative Self Schema	0.11	0.06	.09†	.003
Positive Self Schema	-0.14	0.04	-.13**	.01
Negative Other Schema	0.04	0.04	.03	.0007
Positive Other Schema	-0.02	0.05	-.01	.0001
Interpersonal Sensitivity	0.10	0.01	.30***	.06

Note. R^2 =.34 , p <.001 (unique variability = .09, shared variability = .25); Adjusted R^2 = .33
*p <.05, **p <.01, ***p <.001, $^{\dagger}p$ <.10

Interpersonal sensitivity was the best predictor of scores on the Social Anxiety subscale of the SSI, followed by anxiety and positive self schema. There was a trend for negative self schema as a predictor of Social Anxiety. Depression and the other BCSS scales contributed no unique variance to Social Anxiety scores, and apparent bivariate

associations between these variables and Social Anxiety scores were made redundant or mediated by other variables.

Paranoid schizotypal symptoms. A simultaneous multiple regression was undertaken with the Paranoia subscale of the Brief SSI as the dependent variable, and depression, anxiety, schema (BCSS), and interpersonal sensitivity as the explanatory variables. The results are shown in Table 4.4.

Table 4.4

Summary of Simultaneous Regression Analysis for Variables Predicting Paranoid Schizotypal Symptoms (N = 746)

Variable	*B*	*SE B*	β	sr^2 (unique)
Constant	-1.74	0.93		
Anxiety	0.08	0.02	.15***	.01
Depression	0.02	0.02	.07	.002
Negative Self Schema	0.15	0.04	.17***	.01
Positive Self Schema	0.02	0.03	.03	.0004
Negative Other Schema	0.18	0.03	.21***	.03
Positive Other Schema	-0.08	0.03	-.08*	.005
Interpersonal Sensitivity	0.03	0.01	.14***	.01

Note. $R^2 = .36$, $p < .001$ (unique variability = .07, shared variability =.29); Adjusted $R^2 = .35$
*$p < .05$, **$p < .01$, ***$p < .001$, $^{\dagger}p < .10$

Negative other schema was the best predictor of scores on the Paranoia subscale of the SSI, followed by negative self schema, anxiety, interpersonal sensitivity, and positive other schema. Depression and positive self schema contributed no unique variance to Paranoia scores, and apparent bivariate associations between these variables and Paranoia scores were made redundant or mediated by other variables.

Anomalous Experiences. A simultaneous multiple regression was undertaken with the Anomalous Experiences subscale of the Brief SSI as the dependent variable, and depression, anxiety, schema (BCSS), and interpersonal sensitivity as the explanatory variables. The results are shown in Table 4.5.

Table 4.5

Summary of Simultaneous Regression Analysis for Variables Predicting Anomalous Experiences (N = 746)

Variable	*B*	*SE B*	β	sr^2 (unique)
Constant	-0.31	1.00		
Anxiety	0.15	0.02	.30***	.05
Depression	0.03	0.02	.09	.003
Negative Self Schema	-0.01	0.05	-.01	.00004
Positive Self Schema	0.05	0.03	.07	.002
Negative Other Schema	0.03	0.03	.04	.001
Positive Other Schema	0.01	0.04	.02	.0002
Interpersonal Sensitivity	0.01	0.01	.02	.0003

Note. $R^2 = .13$, $p < .001$ (unique variability = .06, shared variability = .07); Adjusted $R^2 = .12$
*$p < .05$, **$p < .01$, ***$p < .001$, $^{\dagger}p < .10$

Anxiety was the only predictor of scores on the Anomalous Experiences subscale of the SSI. Depression, the BCSS scales, and interpersonal sensitivity, contributed no unique variance to Anomalous Experiences scores and apparent bivariate associations between these variables and Anomalous Experiences were made redundant or mediated by other variables.

4.5 DISCUSSION

This chapter has examined the differential associations between schizotypal symptom types and emotional and psychological variables in a non-clinical sample. This section will review the findings of the current study. The results will initially be considered in relation to each of the research hypotheses outlined at the beginning of this chapter. They will then be discussed in relation to the current literature and the clinical implications of the findings will be examined. Following on from this, potential weaknesses of the study will be outlined, as will possibilities for future research.

4.5.1 Evaluation of the Findings in Relation to Research Hypotheses

It was hypothesised that associations between schizotypal symptoms and levels of emotional and psychological disturbance would be stronger for social anxiety and paranoia, than for anomalous experiences. This was supported by the data. Using both Cohen's (1988) criteria and Meng et al's (1992) method of comparing correlated correlations, associations between anomalous experiences and emotional and psychological variables were found to be significantly smaller than associations between emotional and psychological variables and other schizotypal symptom types. Regression analyses were then conducted to examine differential predictors of each schizotypal symptom type. The results of these analyses will now be discussed.

4.5.1.1 Hypothesis 1

It was hypothesised that social anxiety schizotypal symptoms would be associated with reduced positive beliefs about self, interpersonal sensitivity, and anxiety. This hypothesis was supported by the data. Scores on the Social Anxiety subscale of the SSI were found to be predicted by increased interpersonal sensitivity, increased anxiety, and decreased positive beliefs about self (which could also be considered as low self-esteem). There was also a trend suggesting increased negative beliefs about self as a predictor of Social Anxiety scores. Thus, it appears that social anxiety is related to the way in which an individual feels about themselves, and may also be linked to concerns

about how others view them. These findings support the underlying mechanisms of social anxiety as postulated in the literature. Indeed, previous studies have suggested that socially anxious individuals have both reduced self-esteem and a tendency to anticipate negative social feedback (Clark & Wells, 1995; De Jong, 2002).

4.5.1.2 Hypothesis 2

It was hypothesised that paranoid schizotypal symptoms would be associated with negative beliefs about self and others, interpersonal sensitivity and anxiety. These hypotheses were also supported by the data. Scores on the Paranoia subscale of the SSI were found to be predicted by increased negative beliefs about others, increased negative beliefs about self, increased anxiety, increased interpersonal sensitivity, and decreased positive beliefs about others. Thus, it appears that paranoia is related to the way an individual feels about themselves, as well as the way in which they perceive other people. These findings fit with the notion that paranoid beliefs may develop from a combination of beliefs about the self as vulnerable and others as hostile (Fowler, Freeman, Smith et al., 2006; Laing, 1961). Anxiety also appears to be an important predictor, potentially by increasing arousal and producing a threat response. This supports the idea that paranoid beliefs are maintained by the same psychological processes that have traditionally been associated with emotional disorders (Freeman, 2007). Moreover, the finding that interpersonal sensitivity predicts paranoia provides support for the hypothesis that paranoid beliefs may build upon common interpersonal worries (Freeman, Garety et al., 2005).

4.5.1.3 Hypothesis 3

It was hypothesised that anomalous experiences would be associated with anxiety, but not with schematic beliefs about self or others. This hypothesis was supported by the data. Indeed, bivariate correlations showed weak associations between scores on the Anomalous Experiences subscale of the SSI and emotional and psychological variables, apart from anxiety where the relationship was stronger. Moreover, regression analyses suggested that the only predictor of anomalous experiences was increased anxiety (specifically physiological anxiety as measured by the DASS as opposed to interpersonal sensitivity). The regression model including all emotional and

psychological variables accounted for only 12% of the variance in Anomalous Experiences scores. This is in contrast to models for Social Anxiety and Paranoia which accounted for 33% and 35% of the variance respectively. The finding that anxiety was the only predictor of anomalous experiences fits with research suggesting that increases in anxiety may be involved in the underlying physiology of such phenomena (Morrison, 1998; Slade, 1976). Furthermore, studies investigating hallucination proneness in the general population have found that individuals with higher levels of anxiety display stronger predispositions to hallucinatory experience (Ohayon, 2000).

4.5.1.4 Summary of findings

The findings of this study support the notion that psychotic symptoms are the product of numerous interacting processes (Freeman & Garety, 2003). In particular, the current study has highlighted a role for emotion and psychological variables in the occurrence of psychotic-like phenomena. Such variables appear to have a differential influence on individual symptom types, being particularly important in the development of social anxiety and paranoia. Social anxiety has been shown to be related to the way in which an individual feels about themselves, as well as concerns about how other people view them. Negative beliefs that others are hostile, combined with negative beliefs about self have been implicated as being predictive of paranoia. Conversely, anomalous experiences appear to be influenced to a lesser extent by emotion and psychological variables. This supports the idea that anomalous experiences may be predominantly caused by cognitive dysfunction, rather than being psychologically determined. Despite this, anxiety has been shown to be a significant predictor of all schizotypal symptoms, including anomalous experiences.

4.5.2 Relevance to the Literature

All of the findings in this study support arguments in the literature, as outlined in the introductory section of this chapter. This will now be discussed in more detail.

Anxiety has previously been linked to all types of positive psychotic symptoms (e.g. Freeman & Garety, 2003). The finding that anxiety was a significant predictor of scores on the Social Anxiety, Paranoia, and Anomalous Experience subscales of the SSI in the

current study supports such previous research. Anxiety has been hypothesised to increase arousal and hypervigilance, thus in turn leading to the development of both social anxiety and paranoid ideation (Freeman & Garety, 2003; Nuechterlein & Dawson, 1984). Similarly, anxiety has been implicated as a trigger for the underlying physiology of hallucinatory experiences (Frith, 1992; J. A. Gray et al., 1991; Morrison, 2001). The same mechanisms may explain why anxiety was found to significantly predict schizotypal symptoms in this study. In addition to anxiety, interpersonal sensitivity was also shown to be predictive of social anxiety and paranoia. Interpersonal sensitivity is a specific type of anxiety related to social interaction and the way in which we perceive others to evaluate our behaviour (Boyce & Parker, 1989). The finding that this construct predicts paranoia replicates findings from previous research and supports the idea that paranoid beliefs may stem from common interpersonal worries (Freeman, Dunn et al., 2005; Gilbert, Boxall, Cheung, & Irons, 2005).

Depression was not found to be a significant predictor of any type of schizotypal symptom in the current study. However, bivariate correlations did show an association between depression and all types of schizotypal symptoms. Therefore, rather than being involved in the development of psychotic symptoms, depressed mood may be an emotional response to the occurrence of psychosis and psychotic-like experiences. This is an idea that has been put forward in the literature (e.g. Birchwood, Iqbal et al., 2000). Alternatively, it may be the case that it is specific negative evaluative beliefs associated with depression, as opposed to low mood per se, which are important in the development of psychotic symptoms (Barrowclough et al., 2003). This would explain why in the regression analyses, depression was not associated with schizotypal symptoms when controlling for negative schematic beliefs. Negative schematic beliefs about self and others and not depression were found to be predictive of social anxiety and paranoia, but not anomalous experiences.

A distinction should be made between extreme negative evaluative beliefs about self and low self-esteem, which is perhaps more clearly defined as a lack of positive beliefs about self (Fowler, Freeman, Smith et al., 2006). Low self-esteem has previously been implicated in the development of paranoia, with the suggestion that the function of paranoid thoughts is to prevent negative thoughts and feelings about the self from reaching consciousness (Bentall et al., 2001; Bentall et al., 1994). However, results

from the current study do not support this hypothesis. Although global self-esteem was not assessed in the current study, the positive self subscale of the BCSS (e.g. "I am successful") has been shown to be highly correlated with measures of self-esteem (Fowler, Freeman, Smith et al., 2006). Increased paranoia was associated with reduced scores on this dimension of the BCSS, suggesting that self-esteem is decreased in paranoid individuals. This would not be expected if paranoia served as an effective defence. The findings therefore suggest that low self-esteem may arise as a result of normal emotional processes. Moreover, it was specifically extreme negative beliefs about self and others that were found to predict Paranoia scores, rather than a lack of positive self beliefs. Low self-esteem (i.e. reduced positive beliefs about self) was however found to be predictive of increased Social Anxiety scores, which may be a precursor to paranoia.

4.5.3 Clinical Implications of Findings

In line with the continuum hypothesis of psychosis, investigating relationships between emotional dysfunction and schizotypal symptoms in a non-clinical population enables the potential underlying mechanisms of positive psychotic symptoms to be investigated, without the confounding effects of the psychotic disease process (Raine, Lencz, & Mednick, 1995). Moreover, the finding that relationships between schizotypal symptoms and emotional and psychological disturbance mirror those occurring for psychotic symptoms in clinical samples (Fowler, Freeman, Smith et al., 2006; Freeman & Garety, 2003) can be taken as further support for the continuum hypothesis (Claridge, 1997b).

The findings of the current study combined with those of previous research suggest that there is a need for therapeutic approaches for psychosis to focus on the role of emotion. Assessing and targeting emotional disturbance in the early stages of psychosis may lessen the severity, or even prevent the onset of a psychotic episode (Morrison et al., 2002). Furthermore, as previously outlined in the literature, the content of psychotic symptoms may highlight the emotional status of the individual (Freeman & Garety, 2003). Thus, by reducing emotional distress, psychotic symptoms may also be reduced. Understanding emotional and psychological factors that may have led to the development of psychotic symptoms may also help to inform formulation and enhance

understanding of the potential processes maintaining such beliefs and experiences. Moreover, techniques modified from treatments for emotional disorders (e.g. Clark & Fairburn, 1997) may be useful in the treatment of psychosis (Freeman & Garety, 2003). Emotional disturbance should therefore be assessed and treated in its own right, rather than being assumed to be an epiphenomenon of psychotic symptoms.

It must also be remembered that emotional disturbance may be a response to psychotic symptoms as well as a precursor (Birchwood, 2003; Morrison et al., 2003; Tarrier, Khan, Cater, & Picken, 2007). As such, emotional and psychological factors are likely to have a profound impact on the recovery of individuals who have experienced a psychotic episode. Indeed, even when symptoms have remitted, emotional disturbance has been shown to remain (Birchwood, Iqbal et al., 2000; Chadwick, 1997; Voges & Addington, 2005). It is important that this is not ignored as it may have implications for psychotic relapse, i.e. by providing a trigger for symptom recurrence (Subotnik & Nuechterlein, 1988; Tarrier, Barrowclough, & Bamrah, 1991); and also for long-term functional recovery (Birchwood et al., 2006; Pallanti et al., 2004).

4.5.4 Weaknesses of the Current Study

The current study is cross-sectional and thus it is difficult to know whether the emotional and psychological distress reported is involved in the development of schizotypal symptoms, or whether it occurs as a response to the symptoms themselves. It is likely that both of these hypotheses are correct and that the relationship between emotion and psychosis is reciprocal (Freeman & Garety, 2003). However, although the findings have highlighted a relationship between emotion, psychological variables and schizotypy, the direction of this relationship and the mechanism by which it occurs can at present only be postulated. There is also an issue concerning multicollinearity amongst measures used in this study, in that scores on most measures were inter-correlated. This is a common feature of psychological research (Miles & Shevlin, 2001). However, clear hypotheses were put forward as to the proposed direction of relationships between variables, before any analyses were conducted. This is not to say that other models would not fit the data equally well, or indeed better, and this needs to be considered when interpreting the findings.

The sample used in this study was recruited from a student population using opportunistic sampling methods. Thus, it is questionable as to whether the group is representative of the wider population. Moreover, it is unknown if any of the sample have experienced, or are currently experiencing, a psychotic episode or any other mental health problem. The same problems of using internet-based survey methodology, as outlined in previous chapters (section 2.6.4), also apply here. This research has been conducted using an analogue population under the assumptions of the continuum hypothesis of psychosis. These assumptions suggest that the same mechanisms which underlie psychotic symptoms are also likely to be operating in schizotypal symptoms. This may however not be the case and as such the applicability of the findings to a clinical population remains to be determined. Nevertheless, this same criticism could be argued to apply to all psychosis research conducted on non-clinical samples. Moreover, the findings of the current study replicate those from similar studies conducted using clinical samples and fit with current theory (e.g. Fowler, Freeman, Smith et al., 2006).

Despite some methodological weaknesses, the findings of the current study provide an estimate of the type of relationships which may occur between psychotic-like symptoms and emotional and psychological variables. These can be used to shape the hypotheses and design of future studies investigating these relationships in clinical populations and using longitudinal data. The current study is also very large in comparison to others in this field, which is a clear strength. Moreover, although potentially not representative, participants were matched in age to a first-episode psychosis population, thus aiding the applicability of findings to a clinical group.

4.5.5 Summary

In conclusion, this study has replicated findings of previous research arguing for a role for emotion and schema in the development of psychotic symptoms. However, the emotional route to psychosis may be more defined for social anxiety and paranoia as opposed to anomalous experiences. Moreover, there may be differential associations between individual symptoms and emotional and psychological variables. It must also be remembered that emotional and psychological disturbance may be a reaction to psychotic-like experiences, as well as being involved in their development. With this in mind it is likely that emotional and psychological factors will influence recovery from

psychosis. This will be investigated in more detail in a later chapter of this thesis. One way of further investigating the role of emotion and evaluative self and other beliefs in the formation and maintenance of psychotic symptoms, is to examine the way in which traumatic experiences influence the presence of psychotic-like phenomena. This will be the focus of the next chapter.

CHAPTER FIVE:
STUDY FOUR: RELATIONSHIPS BETWEEN SCHIZOTYPAL SYMPTOMS AND TRAUMA EXPOSURE IN A NON-CLINICAL SAMPLE

5.1 RATIONALE AND CONTEXT OF THE STUDY

5.1.1 Overview

The previous chapter in this thesis examined differential associations between schizotypal symptoms and emotion and schema variables. Paranoia and social anxiety were found to be most strongly associated with these variables, whereas anomalous experiences were predicted solely by anxiety. A further way of investigating the role of emotion and evaluative self and other beliefs in psychosis is to examine the way in which trauma exposure influences the presence of psychotic-like phenomena. This chapter first reviews the literature regarding associations between psychotic symptoms and trauma history, before investigating the impact of traumatic life events on schizotypal symptoms in a non-clinical sample.

5.1.2 Trauma and Psychosis

A history of trauma and victimisation has been shown to be relatively common in individuals with psychosis (e.g. Carmen, Rieker, & Mills, 1984; Goff, Brotman, Kindlon, Waites, & Amico, 1991; Masters, 1995). However, the link between psychosis and trauma is somewhat controversial, with reported rates of trauma being dependent on the assessment tools used and the criteria applied (Mueser, Rosenberg, Goodman, & Trumbetta, 2002). Despite this, most findings suggest that rates of trauma may be elevated in populations with severe mental illness (Goodman, Rosenberg, Mueser, & Drake, 1997). Similarly, rates of post-traumatic stress disorder (PTSD) also appear to be high in this group, suggesting either the presence of more severe trauma or an increased sensitivity to trauma (i.e. a heightened response) in individuals with psychosis (Mueser et al., 1998). In addition to retrospective studies with individuals diagnosed with psychosis, prospective studies have shown that individuals with a history of trauma are at increased risk of developing psychosis at a later date (Janssen et al., 2004; Spataro,

Mullen, Burgess, Wells, & Moss, 2004; Spauwen et al., 2006). Furthermore, investigations with non-clinical samples suggest increased psychosis proneness in individuals who have been exposed to a traumatic event (Gracie et al., 2007; Read, Goodman, Morrison, Ross, & Aderhold, 2004). Psychotic-like symptoms have also been shown to be prevalent in individuals diagnosed with PTSD, further suggesting a link between trauma and psychosis (R. W. Butler, Mueser, Sprock, & Braff, 1996).

Psychosis occurring in the context of a trauma history has been shown to be characterised by more severe psychotic symptoms, particularly in relation to hallucinations, and a worse outcome across a variety of domains (Carmen et al., 1984; Greenfield, Strakowski, Tohen, Batson, & Kolbrener, 1994; Mueser, Rosenberg et al., 2002; Ross, Anderson, & Clark, 1994). Moreover, trauma has been linked to psychosis in a dose-response way, with more severe traumas being associated with more severe symptom presentations (Read, van Os, Morrison, & Ross, 2005; Schenkel, Spaulding, DiLillo, & Silverstein, 2005). In particular, specific links have been made between interpersonal traumas, such as child sex abuse (CSA) and victimisation, and the positive symptoms of psychosis, rather than other types of trauma such as road traffic accidents (Hammersley et al., 2003; Read, Agar, Argyle, & Aderhold, 2003; Ross et al., 1994). Indeed, a study by Bebbington et al. (2004) suggested that individuals with psychosis were 15.5 times more likely to have suffered sexual abuse than individuals without any mental disorder.

Explanations of the association between trauma and psychosis are numerous but can be broadly split into two categories: those suggesting a direct link between trauma and psychosis, and those suggesting an indirect link between trauma and psychosis (Fowler, Freeman, Smith et al., 2006; Morrison et al., 2003). These will now be discussed.

5.1.2.1 Direct route from trauma to psychosis

Direct approaches argue that psychosis and PTSD may share similar developmental processes, with the traumatic event acting as a trigger for the onset of psychosis. This view fits with traditional theories such as the stress-vulnerability model, suggesting that trauma may result in an augmentation in arousal, which in turn influences symptom formation (Zubin & Spring, 1977). In addition, it has been argued that exposure to

interpersonal trauma in childhood may result in over-reactivity and dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, thus increasing stress sensitivity and vulnerability to psychotic symptoms (Read, Perry, Moskowitz, & Connolly, 2001). Although this may be true to a certain extent, it is likely that traumatisation is linked with the emergence of psychotic symptoms in a more specific way. Indeed, most symptoms of psychosis have a content that can be meaningfully related to past and personally significant experiences (Fowler, Garety, & Kuipers, 1998). It is thought that an alternative direct link between traumatic experience and psychotic symptoms may relate to the notion of re-experiencing; where the content of hallucinations and delusions reflect the events of the traumatic experience itself. For example, in the case of CSA, auditory hallucinations may manifest as the voice of the abuser (Read & Argyle, 1999). Where this occurs, these phenomena are appraised as relating to a current threat, as opposed to rumination about a past experience, and thus an emotional response is generated. This is similar to processes thought to occur in PTSD (Brewin & Holmes, 2003; Ehlers & Clark, 2000). Moreover, Fowler and colleagues suggest that the re-experiencing-type symptoms of psychosis may be influenced by problems in the contextual processing of trauma memories (Fowler, Freeman, Steel et al., 2006; Steel, Fowler, & Holmes, 2005). This anomaly in processing results in memories of the event being stored in a manner which leaves them vulnerable to being triggered involuntarily, and thus experienced as intrusions into awareness. In line with this view, experimental studies using analogue and clinical populations suggest that vulnerability to psychosis (i.e. high trait schizotypy) may be associated with an information processing style similar to that outlined above (i.e. a weakened ability to integrate information within a temporal and spatial context) and thus may also act as a vulnerability factor in the development of intrusions following exposure to a traumatic event (Holmes & Steel, 2004; Marzillier & Steel, 2007; Steel, Mahmood, & Holmes, 2008).

5.1.2.2 Indirect route from trauma to psychosis

Although the re-experiencing route may account for psychotic phenomena in a substantive minority of cases, many other cases exist where there is no obvious link between the traumatic life event and the psychotic symptomatology that is reported. This is reflected in a study by Hardy et al. (2005) where a direct link between hallucination content and past trauma was shown in only 13% of cases, with 42% of

cases showing no association at all. In cases without a direct link it is hypothesised that traumatic events may influence the development of psychosis in a more indirect and idiosyncratic way, by inducing emotional disturbance and synthesising negative beliefs about self and others (Fowler, Freeman, Steel et al., 2006; Morrison et al., 2003). Indeed, trauma exposure is associated with a wide variety of undesirable outcomes and can have severe implications upon how an individual views themselves and the external world. Previous studies in non-psychotic populations have found an association between a history of interpersonal trauma (i.e. bullying, assault, sexual abuse) and negative changes in evaluative beliefs about self and others (Foa, Ehlers, Clark, Tolin, & Orsillo, 1999). Moreover, trauma history in non-psychotic individuals has been shown to be related to increased emotional disturbance (Mueser, Rosenberg et al., 2002) and increased interpersonal sensitivity (Figueroa, Silk, Huth, & Lohr, 1997). As outlined above, emotion and schematic beliefs about self and others are important in the interpretation of initial anomalous experiences occurring in the early stages of psychosis (Garety et al., 2001). Thus it seems likely that these same processes will influence psychotic symptoms occurring in the context of trauma history, where emotional disturbance is likely to be increased. In line with this view, a cognitive model of paranoia suggests that persecutory delusions are threat beliefs, potentially emerging in response to the experience of interpersonal stress and trauma (Freeman et al., 2002). In this model, trauma is hypothesised to give rise to threatening appraisals of others (i.e. that others are dangerous and untrustworthy), increasing the likelihood of paranoid interpretations of ambiguous situations or stimuli. Depression, anxiety, and low self-esteem arising as a result of trauma exposure may also influence the content of psychotic symptoms and associated distress, as outlined in the previous chapter (e.g. Freeman & Garety, 2003).

5.1.2.3 Summary

It is more than likely that both direct and indirect routes linking trauma and psychosis account for the development of psychotic symptoms. However, it may be that they each offer differential explanations for individual symptom types (Fowler, Freeman, Steel et al., 2006). Evidence for a direct link between trauma and psychosis has tended to focus on auditory hallucinations, often in the context of CSA (Read et al., 2001). Conversely, exposure to interpersonal trauma (i.e. bullying, sexual abuse, physical victimisation) has

been hypothesised to influence other types of psychotic symptoms (e.g. paranoia and persecutory delusions) by increasing levels of anxiety and depression, and synthesising negative schematic beliefs about self and others (Fowler, Freeman, Steel et al., 2006; Garety et al., 2001; Morrison et al., 2003). Non-interpersonal trauma is not hypothesised to influence the development of psychosis (Mueser et al., 1998). Associations between trauma exposure and state schizotypal symptoms will be investigated in the current study using a non-clinical sample.

5.1.3 Aims of the Study

The introduction to this chapter has emphasised the importance of trauma exposure on the development and maintenance of psychotic symptoms. Exposure to a traumatic life event, particularly of an interpersonal nature, may impact upon symptom formation by increasing stress sensitivity; inducing hallucinations via a re-experiencing route; and/or by increasing emotional disturbance and synthesising extreme negative evaluative beliefs about self and others. The aim of the current study is to examine the influence of trauma history on the presence of schizotypal symptoms and their association with emotion and schema.

5.2 RESEARCH HYPOTHESES

1. Interpersonal trauma (i.e. bullying, physical victimisation, and sexual abuse) will be associated with increased schizotypal symptoms and emotional distress, whereas non-interpersonal trauma (i.e. road traffic accidents, witnessing an accident, natural disaster) will not.

2. The pathway between interpersonal trauma and increased schizotypal symptoms may be different for different symptom types:

    a. Exposure to interpersonal trauma may be specifically and directly associated with anomalous experiences (direct route)

    b. The relationship between interpersonal trauma and paranoia and social anxiety may be mediated by increased negative schematic beliefs about self and others (indirect route)

5.3 METHODOLOGY

The methodology for this study was the same as that outlined for the non-clinical sample in Studies 1 (section 2.4) and 3 (section 3.3). The same participants and procedures were used.

5.3.1 Design

A non-clinical sample of university students were surveyed in a cross-sectional design. All participants completed the Schizotypal Symptoms Inventory (SSI), a trauma screen, and other measures via an internet survey, as described in Study 1 (section 2.4). A between-subjects design was used to investigate group differences between individuals who had and had not been exposed to a traumatic event. A within-subjects design was used to examine the relationships between trauma exposure, emotion, schema, and schizotypal symptoms.

5.3.2 Participants

The same group of non-clinical participants who were outlined in Study 1 also took part in this study (section 2.4.2.1). Sixty-one participants in the sample did not complete the trauma screen and therefore were not included in the current study. The demographic characteristics of the 747 participants who completed the trauma screen were not significantly different from those who did not (mean age = 23.1 years, *SD* = 6.7; 64% female, 24% male, 12% did not disclose gender).

5.3.2.1 Sample size and power analysis

Between-groups analysis. In order to compare differences in SSI scores between those who had and had not been exposed to a traumatic event, a power analysis calculation revealed that to achieve 90% power with a significance level of .05 and an estimated moderate critical effect size of .50, a minimum sample size of 85 participants per group was required (Cohen, 1988).

Correlations. In order to examine the relationship between trauma exposure, emotion, and scores on the SSI, a power analysis calculation revealed that to achieve 90% power with a significance level of .05 and an estimated small to moderate critical effect size of .40, a minimum sample size of 62 participants was required (Cohen, 1988).

Multiple regression. In order to examine whether trauma exposure was a predictor of SSI scores (either directly or indirectly), a multiple regression analysis with eight predictors was conducted for each dimension of the SSI. A power analysis calculation revealed that to achieve 90% power with a significance level of .05, and an estimated small to moderate critical effect size of .15, a minimum sample size of 136 participants was required (Cohen et al., 2003).

5.3.3 Measures

5.3.3.1 Schizotypal Symptoms Inventory (SSI)

As in previous chapters, the SSI was completed to provide an index of current low-level psychotic symptomatology. The SSI has been described in previous chapters and as such will not be repeated here. In this study, the brief version of the SSI was used. Total and subscale scores (i.e. Social Anxiety, Paranoia, Anomalous Experiences) were used in analyses.

5.3.3.2 Trauma History Screen

Participants were asked to complete a trauma history screen (see Appendix E). This consisted of the Trauma History Questionnaire (THQ; B. L. Green, 1996) and eight items selected from the Traumatic Life Events Questionnaire (TLEQ; Kubany et al., 2000). The items enquire about experience of road traffic accidents, bullying, sexual abuse (at three different time points), and physical abuse (with and without a weapon). An additional general item was also included so that participants could report any other types of trauma they had experienced but which had not been specifically asked about (e.g. natural disaster, witnessing an accident). Items from the THQ and TLEQ measures have been shown to have good validity and reliability and to be suitable for research purposes (Norris & Hamblen, 2004). In order to reduce potential distress, specific details about traumas were not requested. Instead, participants were asked to state whether or not they had experienced the particular trauma being asked about. If a particular trauma was endorsed, the participant was asked to give an indication of how severe the incident was (i.e. "Did you think you might be killed or seriously injured?") and how they felt about it now (i.e. "Do you still think about it?"; "Does it still affect you now?"). This information was used to decide whether the event met DSM-IV-TR A1 stressor criteria (American Psychiatric Association, 2000a). Traumas were split into interpersonal (bullying, physical victimisation, sexual abuse) and non-interpersonal (road traffic accident, witnessing an accident, natural disaster) subtypes. In addition, each type of interpersonal trauma was analysed separately.

5.3.3.3 Other measures

In order to examine relationships between trauma, emotion, schema, and schizotypal symptoms; the depression and anxiety scales of the DASS (Lovibond & Lovibond, 1995); the Brief Core Schema Scales (BCSS; Fowler, Freeman, Smith et al., 2006); and the Interpersonal Sensitivity Measure (IPSM; Boyce & Parker, 1989) were also administered. As these measures were described in detail in the previous chapter (section 4.3.3), they will not be discussed again here.

5.3.4 Procedure

The same procedure as outlined in Study 1 (section 2.4.5.1) was adopted for this study and will not be described again here. The non-clinical sample completed the measures via an internet survey.

5.3.5 Data Analysis Plan

All data was analysed using SPSS for Windows, version 14 (SPSS, 2005). Initial screening and cleaning of the data has been described in Study 1 (section 2.4.6.1). In the first stage of the analysis, descriptive statistics were calculated for all variables. Responses to the trauma history screen were also coded as to whether they met DSM-IV-TR A1 stressor criteria (yes/no). Traumas meeting criteria included events which involved actual or threatened death to the self (taken from the probe question asking participants if they thought they were going to be killed or seriously injured); witnessing death or serious injury of another; and learning of the sudden death of a close friend or relative. For traumatic events relating to sexual victimisation, it was also ascertained as to whether the event included sexual penetration. In cases with a lack of detail, the coding was conservative. The frequency of each trauma was then calculated, as was the number of traumas experienced by each individual.

Due to the skewed distributions of the data, independent samples Mann-Whitney U tests were conducted to examine differences in schizotypy scores (across different

dimensions) between those having experienced different trauma types and those who had not. Similar analyses were conducted for anxiety, depression, schema and interpersonal sensitivity to examine whether scores on these variables differed between individuals who had been exposed to a traumatic event and those who had not.

Following this, hierarchical linear multiple regression analyses were conducted to investigate whether trauma exposure predicted schizotypal symptoms. Separate regression analyses were conducted for each dimension of schizotypy that was found to be increased in groups exposed to trauma. In each case, schizotypy served as the dependent variable. In the first stage of the analyses, trauma type served as the only independent variable. Where trauma was shown to be a significant predictor of schizotypal symptoms, emotion and schema were added as further independent variables to see if this affected the predictive value of trauma. If trauma remained a significant predictor of schizotypy even when controlling for emotion and schema, it was surmised that there was a direct association between trauma and symptoms. If however trauma was no longer a significant predictor of schizotypy when controlling for emotional and psychological variables, it was surmised that there was an indirect association between trauma and symptoms, potentially mediated by changes in schematic beliefs and emotional distress.

5.4 RESULTS

5.4.1 Descriptive Data

Descriptive data for emotion and schema variables are shown in Table 4.1 in the previous chapter.

Exposure to at least one traumatic event was reported by 616 participants (82.5%). However, when applying DSM-IV-TR A1 stressor criteria, 262 participants were found to have experienced a severe traumatic event (35.1%). This is lower than some other studies have reported (e.g. Gracie et al., 2007; Kubany et al., 2000) but may be a result of the strict criteria used. The mean number of severe traumas experienced by the sample was 0.58 (*SD* = 1.01), minimum = 0, maximum = 7. Frequencies of exposure to different types of traumatic event are shown in Table 5.1. Increased trauma exposure

(i.e. exposure to more traumas) was associated with higher scores on the Anomalous Experiences subscale of the SSI, $r(747) = .09$, $p = .02$; increased anxiety, $r(747) = .08$, $p = .02$); and more negative beliefs about self, $r(747) = .08$, $p = .03$) and others, $r(747) = .11$, $p = .003$).

Table 5.1

Frequency of Severe Trauma Exposure (N = 747)

Type of Trauma	% Yes
Have you ever been in a serious car accident or serious accident at work, or somewhere else?	6.2
Has anyone ever done anything particularly nasty or cruel to you?	10.0
Did you ever have sexual contact with anyone who was at least five years older than you before you reached the age of thirteen?	3.1
Before you were aged eighteen, did anyone ever use pressure, coercion, or non-physical threats to have sexual contact with you?	5.4
At any other time in your life, has anyone ever used physical force or threat of force to make you have some type of unwanted sexual contact with them?	5.2
Has anyone ever attacked you with a gun, knife or some other weapon, regardless of whether you ever reported it?	7.0
Has anyone ever attacked you without a weapon but with the intent to kill or seriously harm you?	6.2
Have you ever experienced (or seen) any other events that were life threatening, caused serious injury, or were highly disturbing or distressing?	14.9
Endorsement of at least one traumatic experience	35.1

5.4.2 Between-group Differences

Individuals who had experienced a severe traumatic life event and those who had not were compared on SSI scores and on measures of anxiety, depression, schematic beliefs, and interpersonal sensitivity using Mann-Whitney U tests. The results are shown in Table 5.2. Individuals who had been exposed to a severe traumatic event scored significantly higher on the Anomalous Experiences subscale of the SSI; and had significantly more negative beliefs about others than individuals who had not been exposed to a severe traumatic event. There were also trends indicating higher levels of anxiety and negative schematic beliefs about self in the group who had been exposed to a severe traumatic event.

In order to further investigate which type of trauma was associated with increased schizotypy and emotional distress, traumas were grouped into interpersonal (sexual abuse, bullying, physical victimisation with and without a weapon) and non-interpersonal (road traffic accident, natural disaster, witnessing an accident) subtypes. Exposure to non-interpersonal trauma was not associated with any increases in schizotypal symptoms, emotional distress, or negative schematic beliefs (see Table 5.3). Conversely, exposure to interpersonal trauma was associated with higher scores on all types of schizotypal symptoms (trend for Anomalous Experiences); increased anxiety, depression, and interpersonal sensitivity; and increased negative and decreased positive schematic beliefs about self and others (see Table 5.4).

Table 5.2

Differences in Schizotypy, Emotional Distress and Schematic Beliefs between Groups who have and have not been Exposed to a Severe Traumatic Event

	Exposed to trauma Mean (*SD*)		*U*	*p*
	Yes (*N* = 262)	No (*N* = 485)		
Schizotypal Symptoms				
- Total	10.16 (9.88)	9.28 (8.83)	61520.50	.47
- Social Anxiety	4.67 (5.53)	4.36 (5.01)	62865.50	.81
- Paranoia	2.94 (3.78)	2.84 (3.55)	62981.50	.84
- Anomalous Experiences	2.56 (3.52)	2.08 (3.31)	57230.50	.02
Anxiety	7.03 (7.67)	5.36 (5.82)	58115.50	.07
Depression	11.37 (11.77)	9.54 (9.28)	61163.00	.48
BCSS				
- Negative Self	4.83 (4.83)	3.97 (3.91)	57568.50	.07
- Positive Self	12.88 (5.38)	13.17 (4.56)	61171.00	.62
- Negative Other	5.43 (4.48)	4.58 (4.02)	55580.50	.01
- Positive Other	12.17 (4.22)	12.64 (3.61)	59455.00	.27
Interpersonal Sensitivity	95.00 (16.20)	93.32 (15.07)	58709.50	.12

Table 5.3

Differences in Schizotypy, Emotional Distress and Schematic Beliefs between Groups who have and have not been Exposed to a Non-interpersonal Traumatic Event

	Exposed to trauma Mean (*SD*)		*U*	*p*
	Yes (*N* = 81)	No (*N* = 666)		
Schizotypal Symptoms				
- Total	9.30 (11.75)	9.63 (8.87)	23648.00	.07
- Social Anxiety	3.91 (5.12)	4.54 (5.21)	24200.00	.13
- Paranoia	2.60 (4.27)	2.90 (3.55)	23937.00	.09
- Anomalous Experiences	2.78 (4.27)	2.18 (3.27)	25622.00	.44
Anxiety	6.60 (7.92)	5.87 (6.39)	26183.50	.71
Depression	11.73 (12.86)	10.00 (9.89)	26476.00	.84
BCSS				
- Negative Self	4.63 (5.22)	4.23 (4.15)	26028.00	.82
- Positive Self	13.06 (5.73)	13.07 (4.75)	26345.00	.96
- Negative Other	5.54 (4.93)	4.80 (4.10)	24318.50	.24
- Positive Other	12.85 (4.04)	12.43 (3.81)	24330.00	.24
Interpersonal Sensitivity	91.67 (16.59)	94.18 (15.33)	24508.50	.20

Table 5.4

Differences in Schizotypy, Emotional Distress and Schematic Beliefs between Groups who have and have not been Exposed to an Interpersonal Traumatic Event

	Exposed to trauma Mean (*SD*)		*U*	*p*
	Yes (*N* = 170)	No (*N* = 577)		
Schizotypal Symptoms				
- Total	11.30 (10.07)	9.09 (8.89)	42388.00	.01
- Social Anxiety	5.25 (5.56)	4.23 (5.07)	43847.00	.03
- Paranoia	3.36 (3.98)	2.73 (3.52)	44299.00	.05
- Anomalous Experiences	2.68 (3.69)	2.12 (3.29)	44661.50	.06
Anxiety	7.87 (8.00)	5.38 (5.98)	41114.00	.002
Depression	11.92 (12.08)	9.67 (9.60)	45874.00	.24
BCSS				
- Negative Self	5.19 (4.81)	4.00 (4.06)	41611.50	.004
- Positive Self	12.21 (5.33)	13.33 (4.69)	43412.00	.04
- Negative Other	5.58 (4.51)	4.67 (4.09)	42880.50	.02
- Positive Other	11.41 (4.14)	12.79 (3.69)	39638.00	<.001
Interpersonal Sensitivity	96.75 (15.97)	93.07 (15.25)	41503.00	.004

To investigate whether a particular subtype of interpersonal trauma was specifically associated with increased schizotypy and emotional distress; differences in schizotypy scores, anxiety, depression, interpersonal sensitivity, and schematic beliefs about self and others were compared between individuals who had and had not been exposed to different types of interpersonal trauma. The specific subtypes investigated were: severe bullying, physical attack (with and without a weapon), and sexual abuse. Sexual abuse was split into that occurring in childhood (before the age of 13) and that occurring at other times. These analyses are presented in Tables F1 to F4 in Appendix F. Exposure to severe bullying and physical victimisation were associated with increased scores on the Social Anxiety subscale of the SSI. Exposure to sexual abuse occurring in childhood (CSA) was associated with increased scores on the Social Anxiety and Paranoia subscales of the SSI. Sexual abuse occurring outside of childhood was not associated with any increase in schizotypy. No specific subtype of interpersonal trauma was associated with increased scores on the Anomalous Experiences subscale of the SSI. Increased emotional distress was associated with all types of interpersonal trauma apart from sexual abuse occurring outside of childhood. CSA was found to be associated with the most emotional distress and highest schizotypy scores.

5.4.3 Direct and Indirect Pathways

In order to examine direct and indirect pathways between exposure to interpersonal trauma and schizotypy, hierarchical multiple linear regression analyses were conducted. There were two stages to each regression analysis. In the first stage, trauma exposure was the only independent variable. If interpersonal trauma was found to significantly predict schizotypy, then emotion and schema variables were added as further independent variables in a second stage. All types of schizotypy were found to be increased in individuals who had been exposed to interpersonal trauma. Therefore, three regression analyses were conducted using each schizotypy subscale as the dependent variable.

5.4.3.1 Social Anxiety

The results for the hierarchical regression analyses on the Social Anxiety subscale of the SSI are shown in Table 5.5. Exposure to interpersonal trauma was shown to be a significant predictor of increased Social Anxiety scores. However, when controlling for emotion and schema variables, interpersonal trauma was no longer a significant predictor. This suggests that the association between trauma and social anxiety is mediated by changes in anxiety, interpersonal sensitivity, and schematic beliefs about self.

5.4.3.2 Paranoia

The results for the hierarchical regression analysis on the Paranoia subscale of the SSI are shown in Table 5.6. Exposure to interpersonal trauma was shown to be a significant predictor of increased Paranoia scores. However, when controlling for emotion and schema variables, interpersonal trauma was no longer a significant predictor. This suggests that the association between trauma and paranoia is mediated by changes in anxiety, interpersonal sensitivity, and schematic beliefs about self and others.

5.4.3.3 Anomalous Experiences

The results for the hierarchical regression analysis on the Anomalous Experiences subscale of the SSI are shown in Table 5.7. Exposure to interpersonal trauma was shown to be a significant predictor of increased Anomalous Experiences scores. However, when controlling for emotion and schema variables, interpersonal trauma was no longer a significant predictor. This suggests that the association between trauma and Anomalous Experiences is mediated by changes in anxiety.

Table 5.5

Summary of Hierarchical Regression Analysis Investigating the Prediction of Social Anxiety Schizotypal Symptoms by Exposure to Interpersonal Trauma, Before and After Controlling for Emotion and Schema Variables (N = 746)

Variable	*B*	*SE B*	β
Step 1			
Constant	4.19	0.22	
Interpersonal Trauma	1.01	0.45	.08*
Step 2			
Constant	-4.11	1.37	
Interpersonal Trauma	-0.09	0.38	-.01
Anxiety	0.16	0.03	.20***
Depression	-0.01	0.02	-.02
Negative Self Schema	0.12	0.06	.10†
Positive Self Schema	-0.15	0.04	-.14**
Negative Other Schema	0.04	0.04	.03
Positive Other Schema	-0.02	0.05	-.02
Interpersonal Sensitivity	0.10	0.01	.29***

Note. $R^2 = .01$, $p = .03$ for Step 1; $\Delta R^2 = .32$, $p < .001$ for Step 2

*$p < .05$, **$p < .01$, ***$p < .001$, $^{\dagger}p < .10$

Table 5.6

Summary of Hierarchical Regression Analysis Investigating the Prediction of Paranoid Schizotypal Symptoms by Exposure to Interpersonal Trauma, Before and After Controlling for Emotion and Schema Variables (N = 746)

Variable	*B*	*SE B*	β
Step 1			
Constant	2.69	0.15	
Interpersonal Trauma	0.63	0.32	.07*
Step 2			
Constant	-1.59	0.94	
Interpersonal Trauma	-0.16	0.26	-.02
Anxiety	0.09	0.02	.16***
Depression	0.02	0.02	.06
Negative Self Schema	0.15	0.04	.18***
Positive Self Schema	0.02	0.03	.03
Negative Other Schema	0.19	0.03	.22***
Positive Other Schema	-0.08	0.03	-.09*
Interpersonal Sensitivity	0.03	0.01	.14***

Note. $R^2 = .01$, $p = .05$ for Step 1; $\Delta R^2 = .34$, $p < 0.001$ for Step 2

*$p < .05$, **$p < .01$, ***$p < .001$

Table 5.7

Summary of Hierarchical Regression Analysis Investigating the Prediction of Anomalous Schizotypal Symptoms by Exposure to Interpersonal Trauma, Before and After Controlling for Emotion and Schema Variables (N = 746)

Variable	*B*	*SE B*	β
Step 1			
Constant	2.05	0.14	
Interpersonal Trauma	0.62	0.29	.08*
Step 2			
Constant	-0.17	1.01	
Interpersonal Trauma	0.22	0.28	.03
Anxiety	0.15	0.02	.30***
Depression	0.03	0.02	.08
Negative Self Schema	-0.01	0.05	-.01
Positive Self Schema	0.04	0.03	.06
Negative Other Schema	0.02	0.03	.03
Positive Other Schema	0.02	0.04	.02
Interpersonal Sensitivity	<0.01	0.01	.02

Note. $R^2 = .01$, $p = 0.03$ for Step 1; $\Delta R^2 = .11$, $p < 0.001$ for Step 2

*$p < .05$, **$p < .01$, ***$p < .001$

Similar regression analyses were also conducted for subtypes of interpersonal trauma. The results from these analyses are presented in Tables F5 to F8 in Appendix F. For all types of interpersonal trauma (i.e. CSA, bullying, and physical victimisation), the relationship between trauma exposure and increased schizotypal symptoms was mediated by changes in emotion and schema.

5.4.4 Summary of Findings

Increased schizotypal symptoms, emotional distress, and changes in schematic beliefs about self and others appear to be specifically related to exposure to interpersonal (as opposed to non-interpersonal) traumas. When investigating the nature of the association between interpersonal trauma and schizotypy, it appears that rather than a direct link, the relationship is mediated by the influence of trauma on emotion and schematic beliefs. Indeed, exposure to interpersonal trauma alone predicted very little of the variability in schizotypy scores (approximately 1%). Moreover, when combined with emotional and psychological variables, trauma contributed no unique variance to schizotypy scores. These results are consistent with the hypothesis that changes in emotion and schema, brought about by interpersonal trauma exposure, may impact upon the development and maintenance of psychotic-like symptoms.

5.5 DISCUSSION

This chapter has examined the associations between schizotypal symptom types and exposure to a traumatic life event. This section will review the findings of the current study. The results will initially be considered in relation to each of the research hypotheses outlined at the beginning of this chapter. They will then be discussed in relation to the current literature and the clinical implications of the findings will be examined. Following on from this, potential weaknesses of the study will be outlined, as will possibilities for future research.

5.5.1 Evaluation of the Findings in Relation to Research Hypotheses

5.5.1.1 Hypothesis 1

It was hypothesised that exposure to an interpersonal trauma (i.e. bullying, physical victimisation, sexual abuse) would be associated with increased schizotypal symptoms and emotional distress; whereas exposure to a non-interpersonal trauma (i.e. road traffic accidents, natural disaster, witnessing an accident) would not. This finding was supported by the data. Individuals exposed to interpersonal trauma had higher scores on

all dimensions of the SSI; higher levels of anxiety and depression; higher interpersonal sensitivity scores; and increased negative and decreased positive beliefs about self and others. All of these between-group differences reached statistical significance, apart from the Anomalous Experiences subscale of the SSI, where differences were at trend level. Conversely, there were no significant differences on any of these variables between individuals who had and had not experienced a non-interpersonal trauma. These findings support those from previous research (e.g. Mueser et al., 1998).

Subtypes of interpersonal trauma were also found to be associated with increased schizotypal symptoms. Social anxiety was increased in individuals who had been exposed to severe bullying and physical victimisation. Social anxiety and paranoia were particularly elevated in individuals who had been exposed to child sex abuse. No specific subtype of interpersonal trauma was associated with increased anomalous experiences. Interestingly, sexual abuse occurring outside of childhood was not associated with increased schizotypal symptoms on any dimension. Similarly, emotional distress in this group was not as pronounced as that occurring in individuals exposed to CSA. These findings replicate previous research suggesting that sexual abuse occurring in childhood has a particularly severe and prolonged impact on mental health (Browne & Finkelhor, 1986). The differences between these groups could be attributed to differences in the nature of sexual abuse occurring in and out of childhood. In contrast to sexual abuse in later life, CSA is likely to occur over a prolonged period of time in the context of reduced social support and when coping mechanisms are not yet fully developed (Browne & Finkelhor, 1986).

5.5.1.2 Hypothesis 2

It was hypothesised that the pathway between schizotypal symptoms and exposure to interpersonal trauma may be different for different symptom types. In line with previous research on CSA and hallucinations (e.g. Read et al., 2003), it was predicted that exposure to interpersonal trauma may be specifically and directly associated with increased scores on the Anomalous Experiences subscale of the SSI. This hypothesis was not supported by the data. Whilst individuals who had been exposed to interpersonal trauma had increased scores on the Anomalous Experiences subscale of

the SSI (at trend level), trauma exposure was not an independent predictor of anomalous experiences when controlling for anxiety.

In terms of social anxiety and paranoia, it was predicted that exposure to interpersonal trauma would be associated with increased scores on the corresponding subscales of the SSI, and that these relationships would be mediated by increases in both emotional distress and the synthesis of negative beliefs about self and others. These hypotheses were supported by the data. Individuals who had been exposed to interpersonal trauma had significantly higher scores on Social Anxiety and Paranoia dimensions of the SSI. Hierarchical regression analyses suggested that the relationship between trauma exposure and social anxiety was mediated by anxiety, interpersonal sensitivity, and a lack of positive beliefs about self; and that the relationship between trauma exposure and paranoia was mediated by anxiety, interpersonal sensitivity, and extreme negative schematic beliefs about self and others.

5.5.2 Relevance to the Literature and Clinical Implications of Findings

The finding that individuals with a history of severe interpersonal trauma experience increased levels of psychotic-like symptoms replicates findings of previous research (Gracie et al., 2007). The potential mediation of this relationship by emotional and psychological processes further supports the role of emotion in psychosis; and suggests an indirect route between trauma and psychotic symptoms, as outlined in other studies (Fowler, Freeman, Steel et al., 2006; Morrison et al., 2003). The findings of the current study also support those of the previous chapter in this thesis, highlighting the importance of emotion and schema in the development and maintenance of psychotic symptoms.

The results do not provide any support for theories postulating a direct route between trauma and psychotic symptoms (Read et al., 2003). However, the presence of such a pathway cannot be ruled out on the basis of current findings. Previous studies investigating the direct pathway have examined re-experiencing-type symptoms of psychosis and hallucinations, which differ in content to items included on the Anomalous Experiences subscale of the SSI. Moreover, the current study did not assess symptoms of PTSD as has been done in other studies (Mueser, Rosenberg et al., 2002).

It is often a re-experiencing element of PTSD that is linked to hallucinations and therefore it may be this same element that would be linked to anomalous schizotypal phenomena (Gracie et al., 2007; Morrison et al., 2003). Previous studies have also analysed the content of psychotic symptoms and linked this to the content of the traumatic event itself (e.g. Hardy et al., 2005). Such detail was beyond the scope of the current study.

Experimental studies proposing a direct route between trauma and psychotic symptoms have focused on the way in which a deficit in the contextual processing of trauma memories may result in the presence of intrusions (Holmes & Steel, 2004; Steel et al., 2005). In the current study, trauma history was not found to be an independent predictor of anomalous experiences. Rather, the relationship was mediated by anxiety. Anxiety has also previously been linked with contextual processing anomalies (Frith, 1992; J. A. Gray, 1982). As such, it may be the case that increases in anxiety disrupt normal processing and provide a trigger for the occurrence of trauma-related intrusions, which may then be experienced as hallucinations or anomalous perceptions (Morrison, 2001; Steel et al., 2005). This is in contrast to social anxiety and paranoia where schema appears to play more of a role. However, further research would need to be conducted in order to confirm this hypothesis.

In line with the continuum hypothesis, the associations between trauma and schizotypal symptoms highlighted in the current study could be generalised to a clinical sample and thus have implications for clinical practice. Indeed, when considered in the context of a history of interpersonal trauma, the symptoms of psychosis may be understood as being personally meaningful to the client (Fowler, 2000a). The content of psychotic symptoms may mirror the content of the traumatic event; or perhaps more commonly, exposure to a traumatic event of an interpersonal nature may provide the individual with a sense that they are vulnerable and that the world is a dangerous place. Fowler, Garety, and Kuipers (1995) suggest a role for cognitive behaviour therapy for psychosis in assisting the individual to make sense of their psychotic experiences in the context of psychological processes and previous life events.

5.5.3 Weaknesses of the Current Study

In addition to the methodological weaknesses outlined in previous chapters, there are a number of limitations specific to the current study which need to be considered when interpreting the findings. First, the study is cross-sectional and thus it is difficult to establish the direction of relationships between trauma, schizotypy, and psychological variables. It is unknown whether trauma exposure increases predisposition to psychosis, or whether the predisposition was present prior to the traumatic event and influences the associated response (Mueser, Rosenberg et al., 2002). However, as this study was assessing the state presence of schizotypal symptoms rather than schizotypy as a personality trait, it can be assumed with reasonable certainty that the phenomena reported by participants occurred after the traumatic event (i.e. in the last two weeks). Nevertheless, state schizotypal symptoms will be influenced by underlying trait schizotypy which may predate trauma exposure.

Second, the reporting of trauma in the current study was retrospective, which is problematic but somewhat unavoidable. In order to unpick the nature of relationships between trauma, emotion, schema, and schizotypy; longitudinal prospective studies would need to be conducted. This was however beyond the scope of the current study. Furthermore, although the findings suggest that emotional and schematic variables mediate the relationship between trauma and psychosis, it is possible that emotion and schematic beliefs have an influence on the retrospective reporting of trauma. In other words, regression analyses do not discriminate between confounding and mediational effects (Dunn & Bentall, 2007). True mediational analyses can only be conducted on longitudinal data.

Finally, the power of the secondary analyses conducted on subtypes of interpersonal trauma is questionable. Power calculations revealed that at least 85 participants per group would be needed for between-group comparisons of schizotypal symptoms, emotion, and schema. Due to the strict criteria applied to the data, endorsement of subtypes of interpersonal traumas resulted in groups with a smaller sample size than this (see Tables F1 to F8 in Appendix F), and thus the results of these analyses should be regarded with caution. However, the primary focus of this study was exposure to

interpersonal trauma in general, rather than specific subtypes. These analyses were more than adequately powered. Secondary analyses were exploratory and further studies would need to be conducted to confirm their findings.

5.5.4 Summary

Despite some methodological weaknesses, the findings of the current study support research highlighting a role for trauma history in the presence of psychotic symptomatology. Indeed, the finding that exposure to a traumatic event is associated with increased emotional distress, negative schematic beliefs, and increased schizotypal symptoms provides further evidence for the role of emotion and psychological variables in the development and maintenance of psychotic phenomena, thus supporting the findings of Study 3. Moreover, if there is an emotional route into psychosis, it is likely that there is also an emotional route to recovery from the disorder. This will be examined in the next chapter.

CHAPTER SIX:
STUDY FIVE: SCHIZOTYPAL SYMPTOMS AND DIMENSIONS OF RECOVERY FROM PSYCHOSIS

6.1 RATIONALE AND CONTEXT FOR THE STUDY

6.1.1 Overview

The concept of recovery from psychosis is an area of increasing interest to both researchers and clinicians. Traditional views suggest that the notion of recovery from psychosis is somewhat paradoxical. Indeed, lack of recovery and poor outcome formed part of the original diagnostic criteria for the disorder (Bleuler, 1950; Kraepelin, 1919), and this view has long since dominated psychiatric and societal views about severe mental illness. However, more recent long-term follow-up studies have shown that approximately half of individuals diagnosed with psychosis have a favourable outcome (e.g. Harding, 1988; G. Harrison et al., 2001). This literature has been supplemented by service user accounts of the recovery process, showing that recovery from psychosis is in fact possible (Deegan, 1988; Leete, 1989; Lovejoy, 1984; Unzicker, 1989). This paradigm shift has been accompanied by changes in government policy for severe mental illness (Department of Health, 2001) and a move towards "recovery-oriented services" (Anthony, 1993; Jacobson & Greenley, 2001; Turner-Crowson & Wallcraft, 2002).

Despite advances in the understanding of positive outcomes for people diagnosed with psychosis, there currently exists no single agreed definition of recovery. The overwhelming message in the literature is that recovery from psychosis is a complex and arguably multidimensional construct, which can mean different things to different people at different points in their illness pathway (Lester & Gask, 2006). However, such a subjective and disparate view does not aid research into the recovery process. There is a need to operationalise the recovery concept in order to allow recovery-focused interventions to be reliably assessed (Liberman & Kopelowicz, 2002). Within psychiatry and the wider medical profession, recovery is still generally assessed using symptom-related criteria (Whitehorn et al., 2002). Whilst easily defined, this emphasis

on symptom remission as a marker of recovery from psychosis means that social outcomes and psychological well-being are often neglected, despite the fact that these constructs are meaningful to patients and feature heavily in the user literature (Pitt et al., 2007).

When considering recovery, McGorry (1992) proposes the need for a distinction to be made between primary impairments, and secondary impairments occurring as a consequence of psychosis. Primary impairments refer to psychotic symptoms themselves, whereas secondary impairments refer to the psychological effects of the experience of psychosis. McGorry (1992) argues that primary and secondary impairments occur as a result of different processes, follow a different time course of recovery, and may be influenced by different forms of treatment. It is suggested that whereas primary impairments are the direct result of psychotic illness and generally require biological treatments (i.e. medication); secondary impairments are the consequence of the impact of psychosis as a major life event (i.e. on relationships, role functioning, etc) and the way in which the individual interprets and makes sense of their illness.

A distinction has also been made between symptomatic and functional recovery from psychosis, with studies highlighting that social outcome is often independent of symptomatic recovery (Davidson & McGlashan, 1997; Strauss & Carpenter, 1977; Tohen et al., 2000). Indeed, individuals who have recovered symptomatically from a psychotic episode do not always achieve a social recovery (i.e. return to work or education). Similarly, other individuals may have high levels of functioning and yet still be experiencing psychotic symptoms on a daily basis (Harding et al., 1987). This dissonance suggests that factors other than symptoms may influence long-term recovery from psychosis. These have been hypothesised to include loss of confidence and self-esteem (Pallanti et al., 2004); feelings of entrapment and stigmatisation (Birchwood et al., 1993); and fear of relapse (Gumley & Schwannauer, 2006; Gumley et al., 1999). In addition to these so-called “internal” factors, wider external and societal factors such as culture (Sartorious, Jablensky, & Shapiro, 1977) and rates of employment (Warner, 1985), have also been shown to be strongly associated with social outcome following psychosis.

Based on the complex interplay of factors outlined above, some studies have attempted to operationally define recovery from psychosis utilising a multidimensional approach. For example, Liberman, Kopelowicz, Ventura, and Gutkind (2002) suggest that full recovery from psychosis involves sustained improvement in positive and negative symptoms, role functioning (i.e. vocational activity and independent living), and social adjustment (i.e. meaningful peer relationships). Similarly, Whitehorn et al. (2002) outline "symptom control", "autonomous daily living", and "return to the social and occupational lifeline" as important dimensions of recovery. However, these studies use different outcome measures and cut-offs to define recovery on each dimension. Moreover, other operational definitions focus on more subjective aspects of the recovery process. For example, following a review of service user literature, Noordsy et al. (2002) propose "hope", "self-responsibility", and "getting on with life" as dimensional definitions of recovery; although how such aspects can be measured in a standardised way, if at all, remains to be ascertained. Thus, at present no single set of criteria for defining recovery has been universally adopted.

The concept of recovery is still a relatively new area of research. Indeed, in the early nineties, Anthony (1993) stated that "we are nowhere near understanding the recovery concept" (p. 22). Since this paper, much research has been conducted into factors constituting recovery from psychosis, but a general consensus on exactly what recovery is has yet to be reached. A major reason for this is the incredibly subjective and personal nature of recovery. This has led to a focus on personal narratives, following suggestions that more formal assessment methods are "doomed to failure" (G. Shepherd, Boardman, & Slade, 2008). Although service user literature provides a detailed description of the process of recovery for individual users, how this relates to existing standardised assessments of outcome from psychosis, or to the wider population with psychosis, has yet to be established. Operationalised assessment of recovery is important, especially in order to examine the efficacy of recovery-focused mental health services, introduced as a result of government guidelines (Department of Health, 2001). However, there are currently numerous outcome measures used in psychosis research. This study will examine the dimensional structure of such outcome measures and how this may relate to the assessment of recovery from psychosis.

The focus of this thesis is schizotypal symptoms. Previous chapters have shown that schizotypal symptoms are common in individuals in recovery from acute psychosis, and that they are associated with emotional disturbance and psychological distress. This chapter will investigate how different types of schizotypal symptoms relate to various dimensions of outcome in psychosis. Schizotypal symptoms may be viewed as residual symptoms of psychotic illness, but may also reflect emotional and psychological recovery from the disorder. Although previous studies have highlighted the independence of symptomatic recovery (as measured by traditional symptom measures) and functional recovery (i.e. return to competitive employment or education), there have been no studies to date which have considered the impact of residual, or schizotypal symptoms on the wider recovery process. This chapter will investigate different dimensions of recovery and how these relate to different types of schizotypal symptoms.

6.1.2 Aims of the Study

There are two main aims of this study, the first of which is to investigate the multidimensionality of recovery from an episode of psychosis and to propose a theoretical model of the different dimensions of recovery. The second aim of this study is to investigate how different schizotypal symptom types are related to these dimensions of outcome. Both of these aims will be examined using exploratory factor analysis conducted on subscale scores from a range of assessment tools designed to assess different aspects of outcome in psychosis.

6.2 RESEARCH HYPOTHESES

1. The exploratory factor analysis will highlight the multi-dimensional nature of recovery from psychosis, and suggest that outcome should be assessed across a number of different domains.

2. Schizotypal symptom types, as measured by the SSI, will exist on different dimensions of recovery:

    a) The Anomalous Experiences subscale of the SSI may be reflective of residual and low-level psychotic symptoms

    b) The Social Anxiety subscale of the SSI may be reflective of emotional and psychological recovery following an episode of psychosis

    c) The Paranoia subscale of the SSI may be reflective of both residual psychotic symptoms and emotional and psychological recovery following an episode of psychosis

6.3 METHODOLOGY

6.3.1 Design

A sample of patients in recovery from psychosis was surveyed in a cross-sectional design. All participants completed a range of self-report questionnaires and clinician-administered assessments as part of the baseline assessment of the "Improving Social Recovery in Early Psychosis" (ISREP) trial (see Appendix B for trial paper).

Subscales from the assessment tools were entered into an exploratory factor analysis using maximum likelihood estimation in Mplus (Muthén & Muthén, 1998). Factor analysis is a shared variance technique commonly used to identify the core psychological constructs evaluated by tests or scales (Delis, Jacobson, Bondi, Hamilton,

& Salmon, 2003). Shared variance techniques are based on the assumption that a significant correlation between two or more testing variables indicates that the variables are measuring a similar construct. The specific aim of factor analysis is to "determine the number and nature of latent variables or factors that account for the variation and covariation among a set of observed measures" (Brown, 2006; p. 13). A factor is defined as "an unobservable variable that influences more than one observed measure and that accounts for correlations among those observed measures" (Brown, 2006; p. 13). EFA is an exploratory or descriptive form of factor analysis (i.e. no a priori restrictions are placed on the data) used in the early stages of construct validation and designed to "determine the appropriate number of common factors [in a set of observed variables] and to uncover which measured variables are reasonable indicators of the various latent dimensions" (Brown, 2006; p. 14). Thus in this study, outcome measures commonly used in psychosis research were entered into an exploratory factor analysis to examine the main important constructs or dimensions of recovery from psychosis.

EFA differs from Principal Components Analysis (PCA) as used in Study 1. The main aim of PCA is data reduction, whereas the goal of EFA is to identify latent variables which make sense of correlations existing between measured variables. PCA is used mainly in scale construction, whereas EFA is used when a researcher wishes to identify a set of latent constructs underlying a battery of measures (Fabrigar, Wegener, MacCallum, & Strahan, 1999). EFA is also a useful approach for examining construct validation in relation to theoretical concepts. For example, within psychiatry, symptoms thought to be manifestations of a single mental disorder would be expected to be highly correlated and thus to load on the same factor. Conversely, indicators of theoretically distinct constructs would not be expected to be correlated and thus to load on different factors. EFA also accounts for measurement error, whereas PCA does not, thus providing arguably more accurate results.

6.3.2 Participants

Seventy-seven participants were recruited from secondary mental health services in the East Anglia Region of the UK, from a catchment area with a semirural population of around two million people, living in small cities, towns and rural areas. The sample

were in relative symptom remission from their psychosis but had high levels of social disability.

The sample had a mean age of 29.0 years (*SD* = 6.8 years). Seventy-one per cent were male and 29% were female. Ninety-one per cent of the sample was white and 9% were from other ethnic origin. Sixty-five per cent had received a diagnosis of non-affective psychosis (predominantly schizophrenia) and 35% had a diagnosis of affective psychosis (predominantly bipolar disorder). Mean illness duration was 4.8 years (*SD* = 2.3 years) and mean unemployment length was 209 weeks (*SD* = 182 weeks). See Appendix B for a report on the ISREP study and for a fuller description of the sample.

6.3.2.1 Sample size and power analysis

A sample size of 77 is satisfactory to conduct a factor analysis, although larger samples are recommended (Gorsuch, 1993). In EFA, between three and five participants per measured variable are said to be required in order to produce accurate estimates of the population parameters (Kline, 1993). The current study has 23 measured variables and thus 3.3 participants per measured variable.

6.3.3 Measures

All participants completed the Brief Schizotypal Symptoms Inventory (SSI) as described in previous chapters. A range of other measures were also completed and these are outlined below.

6.3.3.1 Social functioning measures

Time Use Survey (modified from Office for National Statistics, 2003). The UK 2000 Time Use Survey (Short, 2006) was modified in the ISREP study in order to make it suitable for use in a psychosis population. The original Time Use Survey consists of seven daily diaries, completed by participants and accounting for every ten minutes of their time. This is supplemented by a structured interview administered by a researcher. The original time use survey has been shown to be an adequately reliable tool for assessing time use (Short, 2006). See Appendix E for a copy of the Time Use Survey.

The version of the time use survey used in the current study differs from the original in two ways. First, the modified interview is shorter than that used by the Office of National Statistics. Questions were removed which did not relate to the areas of time use under investigation (as outlined below). Second, the seven daily self-report diaries were removed in order to reduce demand on participants, who were already being asked to complete a range of self-report instruments. However, all questions that were included in the modified version were taken from the original, and the same coding framework was applied to the data.

The modified time use survey used in this study consists of a semistructured interview in which the participant is asked about how they have spent their time over the last month. Activities enquired about include: work, education, voluntary work, leisure, sports, hobbies, socialising, resting, housework/chores, childcare, and sleep. As a result of the interview, time spent on each of the activities is calculated in terms of the number of hours per week allocated to that activity. Two summary measures can be derived from the Time Use Survey: hours in *Constructive Economic Activity* and hours in *Structured Activity*. Constructive Economic Activity is calculated as the sum of hours per week over the last month spent in work, education, voluntary work, housework and chores, and childcare. Structured Activity is calculated as the sum of hours per week over the last month spent in constructive economic activity, but also includes hours engaged in structured leisure activity, sports and hobbies.

The time use survey provides a direct and objective measure of the number of hours an individual is spending in activity. This is contrast to other measures of social functioning for use in psychosis populations, which provide a more subjective assessment of the quality of an individual's life (Barry & Crosby, 1996). A further strength of the Time Use Survey is that it has been applied and validated in a normative community population and thus allows the recovery of individuals with psychosis to be compared with societal norms, i.e. has functioning improved to a degree which is similar to that occurring in the general population? This comparative facility is something which is lacking in other outcome measures for psychosis (Liberman, 2002).

Hours spent in Constructive Economic Activity and Structured Activity were found to be significantly associated with quality of life ($r(76) = .39$, $p = .001$; and $r(76) = .43$, $p < .001$ respectively) measured by the Quality of Life Scale (Heinrichs, Hanlon, & Carpenter, 1984); social functioning ($r(77) = .27$, $p = .02$; and $r(77) = .31$, $p = .006$ respectively) measured by the Social and Occupational Functioning Scale (Goldman et al., 1992); and activity ($r(77) = .53$, $p < .001$; and $r(77) = .57$, $p < .001$ respectively) measured by the Time Budget (S. Jolley et al., 2006). Thus, the Time Use Survey has good convergent validity with other measures of functioning. Hours in activity were not however associated with positive, negative, or general symptoms measured by the PANSS (Kay et al., 1987). This is a strength of the Time Use Survey as scores on some functioning assessment tools have previously been shown to be confounded by negative symptoms (Barry & Crosby, 1996). Moreover, it supports the notion that symptomatic and functional recoveries are independent of one another (e.g. Tohen et al., 2000).

Quality of Life Scale (QLS; Heinrichs et al., 1984). The QLS is a 21-item scale which utilises a semistructured interview approach. It is designed to assess the functional impairments associated with psychosis, including problems with interpersonal relationships and occupational role functioning. The measure is a commonly used tool for assessing deficit symptoms in psychosis and consists of four categories: Intrapsychic Foundations (e.g. sense of purpose, motivation, curiosity and empathy); Interpersonal Relations (e.g. social contact, relationships with family); Instrumental Role Functioning (e.g. employment, accomplishment, role satisfaction); and Common Objects and Activities (e.g. participation and engagement in regular activities). This structure was confirmed by a factor analysis conducted by the authors,

thus confirming the validity of the measure. The QLS has also been shown to have good reliability, with the authors reporting an inter-rater reliability alpha of .94 for the total scale and .91 to .97 for the subscales.

Each of the 21 items has suggested probe questions to assist the interviewer in making a judgement on the level of the disability. Scores are rated on a scale of 0 (*severe impairment*) to 6 (*unimpaired*). The QLS is a commonly used outcome measure in psychosis research (Cramer et al., 2000) and was included in the current study to examine how it is related to other dimensions of outcome. Total QLS scores and scores on the Instrumental Role Functioning dimension were used in the analysis. The Instrumental Role Functioning dimension was chosen because it was thought that this may be particularly important when considering recovery. Other subscales were not included due to issues of power (i.e. a larger sample size would be required).

Social and Occupational Functioning Assessment Scale (SOFAS; Goldman et al., 1992). The SOFAS is a visual analogue scale whereby the interviewer makes an objective rating of the individual's current social and occupational functioning on a scale of 0 to 100, with higher scores indicating better functioning. The SOFAS was originally devised for use in Axis V assessment in DSM-IV (American Psychiatric Association, 1994) and is a reliable and commonly used measure of social functioning in psychosis (Goldman et al., 1992; Whitehorn et al., 2002).

EuroQol (Brooks, 1996; EuroQol Group, 1996). The EuroQol is a self-report visual analogue scale on which participants rate their health state (mental and physical) on a 0-100 point scale. Higher ratings correspond to better quality of life. The scale has been used in a variety of health settings to assess quality of life and outcome (e.g. Dorman, Waddell, Slattery, Dennis, & Sandercock, 1997). Validity of the scale for use in a population with schizophrenia has also been investigated and shown to be good (König, Roick, & Angermeyer, 2007). In the current study this measure was used to gain a subjective assessment of global health status/quality of life, for use in combination with researcher rated quality of life scales.

6.3.3.2 Symptom assessments

Positive and Negative Syndrome Scale (PANSS; Kay et al., 1987). The PANSS has been described elsewhere in this thesis (section 2.4.4.3) and as such a description will not be repeated here. The PANSS is commonly used to assess symptom remission in operationalised criteria for recovery from psychosis (e.g. Liberman et al., 2002; Whitehorn et al., 2002) and was used in the current study to assess the level of psychotic symptoms present in the sample.

Scale for the Assessment of Positive Symptoms (SAPS; Andreasen, 1984) and Scale for the Assessment of Negative Symptoms (SANS; Andreasen, 1981). The SAPS and SANS were used in addition to the PANSS. These measures provide a detailed description of specific positive and negative symptoms of psychosis. For both scales, symptoms are rated using a 6-point (0-5) rating scale, with higher scores indicating more severe symptoms. Symptoms are rated over the last month. In the current study, total scores were used for both the SAPS and the SANS.

The SAPS is a 35-item rating scale for the assessment of the positive symptoms of psychosis. Items are rated based on patients' responses to a clinical interview. Generally, higher scores on items reflect increased frequency of occurrence of that particular symptom. However, in some cases (e.g. delusions) conviction, preoccupation and severity are also included in the rating. The SAPS consists of five subscales: Hallucinations (e.g. auditory hallucinations, somatic hallucinations); Delusions (e.g. persecutory delusions, delusions of jealousy); Bizarre Behaviour (e.g. clothing and appearance, repetitive and stereotyped behaviour); Positive Formal Thought Disorder (e.g. derailment, pressure of speech); and Inappropriate Affect. Internal consistency of the SAPS has been shown to be good, as has inter-rater reliability for the scale (Andreasen, 1990). The measure is commonly used to assess positive psychotic phenomenology in psychosis for both clinical and research purposes (e.g. Perry & Braff, 1994).

The SANS is a 24-item rating scale for the assessment of the negative symptoms of psychosis. Items included in the SANS are predominantly observational in nature and

the rater is required to make an objective judgement about their presence or absence. The scale contains five subscales of negative symptoms: Alogia (e.g. poverty of speech, blocking); Affective Flattening (e.g. unchanging facial expression, decreased spontaneous movements); Avolition-Apathy (impersistence at work and school, physical anergia), Anhedonia-Asociality (e.g. ability to feel intimacy and closeness), and Attention (e.g. social inattentiveness). The SANS has been shown to have good internal consistency, with Cronbach's alphas ranging from .67 to .90 for the five subscales. Furthermore, the measure has been found to correlate well with the negative symptoms items of the Brief Psychiatric Rating Scale (BPRS; Overall & Gorham, 1962), suggesting good concurrent validity (Thiemann, Csernansky, & Berger, 1987). Reliability of the SANS has been confirmed by a multi-site investigation (Mueser, Sayers, Schooler, Mance, & Haas, 1994).

Global Assessment of Symptoms Scale (GAS; American Psychiatric Association, 2000b). The GAS is part of the Global Assessment of Functioning scale (GAF; American Psychiatric Association, 2000b), a 0-100 visual analogue scale used by mental health clinicians to rate social, occupational, and psychological functioning. Symptoms and functioning can be rated separately. The scale is similar to the Global Assessment Scale, featuring in DSM-IV (American Psychiatric Association, 1994). For the purpose of this study, only the symptoms part of the measure was used to assess global symptoms. Higher scores on the scale correspond to lower levels of symptoms. The GAS was used in this study to complement the PANSS, SAPS and SANS and to assess general symptomatology. Both the GAF and the GAS have good reliability and validity and are widely used in research, thus enabling comparability with other studies (Endicott, Spitzer, Fleiss, & Cohen, 1976).

6.3.3.3 Assessments of emotional and psychological well-being

Beck Depression Inventory (BDI-II; Beck, Steer, & Brown, 1996). The BDI-II is self-report measure designed to assess the symptoms of depression and their intensity in clinical and non-clinical samples. The BDI-II consists of 21 items rated on a 4-point scale (0-3) of increasing severity. Symptoms are rated dependent on their presence in the last two weeks. Scores on the BDI-II range from 0 to 63 and specific cut-off scores are provided for different bands of severity. The BDI-II is an extensively

used measure of depression and the symptoms it enquires about are in line with DSM-IV criteria for the disorder. The BDI-II has also been shown to correlate at .71 with the Hamilton Depression Rating Scale (Hamilton, 1960) and has a one-week test-retest reliability of .93. In addition, the scale has been shown by the authors to have good internal consistency, demonstrating a coefficient alpha of .91. Moreover, the BDI-II has been used in many studies with patients with psychosis (e.g. Fialko et al., 2006) and has also been shown to correlate at .91 with the interview-based Calgary Depression Scale for Schizophrenia (Addington, Addington, & Maticka-Tyndale, 1993). Depression has previously been shown to impede recovery from psychosis (Birchwood & Iqbal, 1998) and thus the BDI-II was included in the current study to examine how levels of depression were associated with other dimensions of outcome.

Beck Anxiety Inventory (BAI; Beck & Steer, 1987). The BAI is a self-report measure designed to assess common symptoms of anxiety. The BAI includes 21 items which are descriptive of subjective, somatic, or panic-related symptoms of anxiety. Individuals are asked to rate each item on a 4-point scale dependent on how much they have been affected by each of the symptoms in the last week, from 0 (*not at all*) to 3 (*severely*). Scores on the scale range from 0 to 63 and specific cut-off scores are provided for different bands of severity. The scale has been shown by the authors to have high internal consistency, with item-total correlations ranging from .30 to .71. Furthermore, test-retest reliability has been reported as .75. The scale has been shown to have validity for use with patients with panic disorder, social phobia, obsessive-compulsive disorder and generalised anxiety. It has also been used in many studies with patients with psychosis (e.g. Freeman & Garety, 1999) and is suitable for use in both clinical and research settings. Anxiety has previously been shown to occur in the recovery stages of psychosis (Freeman & Garety, 2003) and thus the BAI was included in the current study to examine how levels of anxiety were associated with other dimensions of outcome.

Beck Hopelessness Scale (BHS; Beck & Steer, 1988). The BHS is a 20-item self-report scale designed to assess three main aspects of hopelessness: feelings about the future; loss of motivation; and expectations. Items are rated using a dichotomous true/false response format. Total scores from the BHS were used in the current study. Higher scores reflect increased levels of hopelessness. The BHS was originally

designed to highlight individuals who may be at risk of committing suicide and has been shown to have good predictive value with respect to suicidal ideation (Beck, Brown, Berchick, Stewart, & Steer, 1990). However, the BHS has also been used to assess negative attitudes about the future (Beck & Steer, 1988). The authors report an internal consistency of the scale of between .82 to .93; and a six-week test-retest reliability of .66. BHS scores have also been found to correlate at .75 with severity of depression, as measured by the BDI-II (Enns, Inayatulla, Cox, & Cheyne, 1997). The BHS was used in the current study as hope is thought to be an important aspect of recovery from psychosis (Noordsy et al., 2002).

Personal Beliefs about Illness Questionnaire (PBIQ; Birchwood et al., 1993). The PBIQ is a 16-item measure designed to assess an individual's cognitive appraisals about their illness. It encompasses five subscales: Loss; Humiliation; Shame/Stigma; Attribution of Behaviour to the Self or the Illness; and Entrapment in Psychosis. Total PBIQ scores were used in the current study. The scale was specifically designed for use in psychosis and has been shown by the authors to have good reliability and validity (Birchwood et al., 1993). Low scores on the scale indicate favourable attitudes towards the self and the psychosis, e.g. high perceived control over illness, positive expectations of the future, low awareness of stigma, and a view of the illness as being separate to the self. The PBIQ was included in the current study as previous quantitative and qualitative studies have shown that illness cognitions may be important in long-term recovery from psychosis (e.g. Anthony, 1993; Gumley & Schwannauer, 2006; Hoffmann & Kupper, 2002; C. Jackson & Iqbal, 2002; Lobban, Barrowclough, & Jones, 2003).

Brief Core Schema Scales (BCSS; Fowler, Freeman, Smith et al., 2006). The BCSS has been described elsewhere in this thesis (section 4.3.3.3) and as such a description will not be repeated here. This measure was used in the current study to examine how individuals' beliefs about self and others may be related to outcome.

6.3.4 Procedure

Participants completed all of the above measures as part of the baseline assessment process for the ISREP study. Self-report measures were completed independently by the

participants, although assistance was provided if required. All other assessments were conducted by a researcher. Formal training in all measures was provided and interviews were audio-taped for reliability and quality control. Researchers met on a regular basis to ensure reliability in ratings. Some qualitative data was also collected from the interviews and used in defining the factor structure.

6.3.5 Data Analysis Plan

Initial screening and cleaning of the data has been described in Study 1 (section 2.4.6.1). In the initial stage of the analysis, descriptive statistics were calculated for all variables using SPSS for Windows, version 14 (SPSS, 2005). Exploratory factor analysis (EFA) was conducted in Mplus for Windows, version 4.1 (Muthén & Muthén, 1998). Mplus was used rather than SPSS because EFA is a special case of structural equation modelling and Mplus is specialised software developed for this purpose.

In order to assess the multidimensionality of recovery in psychosis, an EFA using maximum likelihood estimation with promax rotation was conducted using subscale scores for all of the assessment tools described above. A key advantage of maximum likelihood estimation is that it allows for a statistical evaluation of how well a factor solution fits the data. Promax rotation was used as it is an oblique as opposed to orthogonal rotation, and thus allows the factors defined to be correlated. As it is likely that different dimensions of recovery will be associated with one another, this was decided to be the most appropriate type of rotation (Costello & Osborne, 2005; Fabrigar et al., 1999).

EFA attempts to determine the minimum number of latent variables or factors that can adequately describe the correlations among a set of observed variables. It is important that the factors be interpretable according to a recognised theory in addition to the model fitting the data well. For the current data set, an initial examination of eigenvalues and the scree plot determined the maximum number of factors a suitable model may contain. Models with one through to six factor solutions were then run and the goodness of fit of each model was established using the root mean square residuals provided by Mplus. Interpretation of factors was also influenced by the recovery

literature and qualitative data collected during the interviews. This procedure complies with best practice guidelines for EFA (Costello & Osborne, 2005; Fabrigar et al., 1999).

6.4 RESULTS

6.4.1 Descriptive Data

Table 6.1 provides descriptive data for all variables in the ISREP sample. Despite being in relative remission from positive symptoms, the descriptive data reflect a plethora of other concerns reflective of poor recovery. There are moderate levels of depression and anxiety in the sample, as well as relatively high levels of hopelessness. Moreover, positive schematic beliefs are low in comparison to a normative sample. Hours in constructive activity are also low compared to non-clinical norms (Office for National Statistics, 2003). Data on some variables were skewed (e.g. SSI, Time Use Survey, BCSS) and the data was resistant to normalising transformation.

Table 6.1

Descriptive Statistics for all Study Variables

	N	Min-Max	Mean (*SD*)	Skewness (*SE*)
Schizotypal Symptoms Inventory				
- Total	68	0-80	19.12 (16.02)	1.72 (0.29)
- Social Anxiety	68	0-24	8.75 (6.45)	0.48 (0.29)
- Paranoia	68	0-24	5.85 (6.33)	1.37 (0.29)
- Anomalous Experiences	68	0-32	4.51 (6.31)	2.43 (0.29)
Time Use Survey (hours)				
- Constructive Economic	77	0-94	12.43 (17.05)	3.20 (0.27)
- Structured	77	2.25-97	29.03 (19.42)	1.41 (0.27)
Quality of Life Scale				
- Total	76	31-110	64.54 (14.85)	0.53 (0.28)
- Instrumental Role	77	0-18	6.04 (3.96)	0.54 (0.27)
SOFAS	77	35-80	50.06 (8.47)	1.06 (0.27)
EuroQol	69	0-100	55.38 (23.50)	-0.36 (0.29)
PANSS				
- Total	77	37-87	56.74 (10.83)	0.33 (0.27)
- Positive	77	7-24	12.22 (3.83)	0.63 (0.27)
- Negative	77	7-25	13.64 (3.67)	0.67 (0.27)
- General	77	19-48	30.88 (5.94)	0.23 (0.27)
SAPS Total	77	0-55	14.71 (12.56)	0.88 (0.27)
SANS Total	77	8-62	32.10 (11.76)	0.23 (0.27)
Global Assessment of Symptoms	77	30-75	56.83 (9.76)	-0.64 (0.27)
Beck Depression Inventory	73	0-57	21.90 (13.75)	0.46 (0.28)
Beck Anxiety Inventory	74	0-50	16.97 (12.56)	0.68 (0.28)
Beck Hopelessness Scale	74	1-20	8.80 (5.74)	0.49 (0.28)
Personal Beliefs About Illness	70	3-40	21.52 (7.53)	0.07 (0.29)
Brief Core Schema Scales				
- Negative Self	74	0-24	6.15 (5.80)	1.23 (0.28)
- Positive Self	74	0-21	8.83 (6.03)	0.44 (0.28)
- Negative Other	74	0-24	6.62 (6.41)	1.00 (0.28)
- Positive Other	74	0-24	10.42 (6.15)	0.29 (0.28)

6.4.2 Dimensions of Recovery

In order to investigate the multidimensionality of recovery from psychosis, an EFA using maximum likelihood estimation with promax rotation was performed on the subscale scores of all of the measures outlined above. The analysis accounted for all missing data so that the full set of data from all 77 participants could be used. The outcome of this analysis is reported here.

Factors were selected using a range of criteria from good practice guidelines for factor selection in EFA (Brown, 2006; Fabrigar et al., 1999). Initial analyses revealed the presence of six factors with eigenvalues exceeding one (i.e. Kaiser criterion). This was confirmed by an examination of the scree plot (see Figure 6.1) using the scree test (Cattell, 1966). Models with between one and six factors were then fitted to the data and the goodness of fit was examined using root mean square residuals provided by Mplus. The root mean square residual is the average of the differences between the observed variable correlations and the correlations estimated by the model (Brown, 2006). If a model provides a good fit to the data, the value of the root mean square residual should be below 0.05. The eigenvalues for each factor and root mean square residuals for each of the models are shown in Table 6.2.

An item was defined as loading on a factor if it had a loading of 0.30 or above (Tabachnick & Fidell, 2001). Joint loadings were considered to occur if an item loaded on more than one factor with a difference of less than 0.10. Each factor had to have more than two items loading on it in order to be considered as a dimension of recovery.

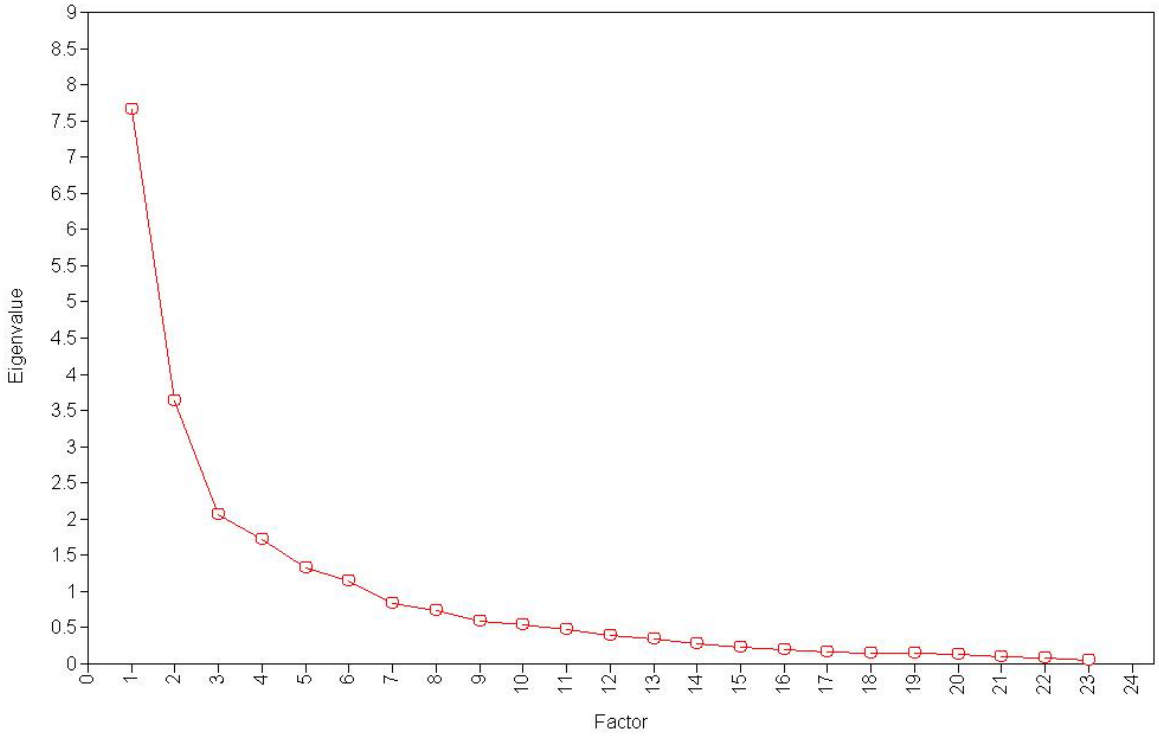


Figure 6.1

Scree plot from exploratory factor analysis

Table 6.2

Eigenvalues and Root Mean Square Residuals for Different Factor Solutions

Number of factors	Eigenvalue	Root Mean Square Residual
1	7.65	0.17
2	3.64	0.11
3	2.07	0.09
4	1.72	0.07
5	1.33	0.06
6	1.14	0.039

After an examination of each of the models, based on the above rules and with reference to the recovery literature, it was decided that the six factor model provided the best fit to the data. The factor loadings of this model are shown in Table 6.3. Correlations between each of the factors are shown in Table 6.4. Each factor will now be described.

The factor structure was supported by qualitative data collected during the assessments. Relevant quotes are used to illustrate each factor.

6.4.2.1 Factor 1: Activity Levels

> If I can start some voluntary work and succeed at that, then I can keep on moving onto the next step, and improve my confidence. I don't want to sit on my backside and do nothing… Eventually I want to go back to work and get off benefits… Having plans in place for what I'm doing in the week definitely helps, rather than just thinking, "well, I've got a whole week ahead of me and nothing to do"… 'cos that would just drive me to distraction I think. (Participant 73)

The first factor was labelled "Activity Levels" and reflects the amount of time that an individual is spending in structured and constructive economic activity. The two subscales of the Time Use Survey were the only loadings on this factor, suggesting that the measure reflects an independent dimension of recovery. This factor was also independent of symptom-related factors.

6.4.2.2 Factor 2: Positive Symptoms

> These days, I sometimes just see things out of the corner of my eye, and when I look, there's nothing there. (Participant 20)

> I feel mildly paranoid now… mental panics… like some days when I'm going around the supermarket, I'm constantly looking over my shoulder… and I don't know why. (Participant 58)

The second factor was labelled "Positive Symptoms" and reflects the traditional conceptualisation of recovery from psychosis. All measures relating to the assessment of positive symptoms loaded on this factor, including the SAPS; the PANSS Positive subscale; the GAS; and the Paranoia and Anomalous Experiences subscales of the SSI.

6.4.2.3 Factor 3: Clinician-rated Recovery

> My CPN says, "you're ready to do more" (Participant 73)

The third factor was labelled "Clinician-rated Recovery" and reflects functional recovery, or quality of life, as assessed by measures which are rated by clinicians or researchers. Measures loading on this factor include the Quality of Life Scale and the SOFAS. Although this factor correlates with the Activity Levels factor, the finding that it came out as an independent dimension of recovery suggests that the measures loading on it are assessing something qualitatively different from actual hours spent in activity.

6.4.2.4 Factor 4: Negative Symptoms

> I have big problems with motivation… it can be awful, to the point that sometimes I haven't gone out for days. Just couldn't be bothered to do anything. I know I need to do things, but sometimes I just can't be bothered. (Participant 20)

> It's difficult to think clearly… simple decisions sometimes feel very difficult... I'm stumbling over my thoughts and mumbling away… and I find that quite frustrating. (Participant 58)

The fourth factor was labelled "Negative Symptoms" and includes those measures designed to assess negative symptoms – the SANS and the Negative symptoms subscale of the PANSS. This factor was moderately associated with both the Positive Symptoms factor and the Clinician-rated Recovery factor. However, it was independent of actual time spent in activity (i.e. the Activity Levels factor).

6.4.2.5 Factor 5: Resilience and Optimism

> Everything I do is one step further to getting back, and that makes me feel good, it improves my self-esteem… It's not until you've been through it that you know… I don't want to fail… and I think this time, feeling positive has got me through. (Participant 73)

The fifth factor was labelled "Resilience and Optimism" and reflects feelings of positivity and hope. Measures loading on this factor include positive beliefs about self and positive beliefs about others subscales of the BCSS, and the Beck Hopelessness

Scale (negative loading). Interestingly the EuroQol also loads on this factor, even though it is designed to be an assessment of functioning. This suggests that those individuals who are more positive or hopeful may rate themselves as having higher quality of life using this scale. High scores on this factor may be conducive to better recovery.

6.4.2.6 Factor 6: Emotional Barriers

> I feel like I'm constantly having this barrage of negative thoughts and I'm blaming myself and just getting annoyed with the condition I've got… There's this complete loss of control in my life. I don't know why I was thinking like that then… I had this overly inflated sense of self importance then, and yet now I feel completely unimportant. I find it quite distressing that I could think like that. (Participant 58)

> I'm not sure I have the confidence [to work] yet… Because my life has been so negative in places, I find it difficult to envisage the future. …I find it difficult to be around other people… to explain about my illness. (Participant 36)

The sixth factor was labelled "Emotional Barriers" and includes measures which may be considered to reflect barriers towards recovery, notably emotional dysfunction (BDI and BAI scores) and negative evaluative beliefs about self and others. Personal Beliefs about Illness scores also loaded positively on this factor suggesting that negative illness cognitions (i.e. feelings of stigma, etc) may be an emotional barrier to recovery. The Social Anxiety and Paranoia subscales of the SSI also loaded positively on this factor. A negative correlation was shown between this factor and the Resilience and Optimism factor, suggesting that they may be opposite ends of the same spectrum.

Table 6.3

Rotated Factor Solution for Six-factor Recovery Model (N = 77)

	Activity Levels	Positive Symptoms	Clinician Rated	Negative Symptoms	Resilience Optimism	Emotional Barriers
Cons Economic Activity	**0.86**	0.10	0.06	-0.01	0.11	0.04
Structured Activity	**0.83**	-0.05	0.09	0.09	0.06	0.06
QLS Total	0.04	0.05	**0.87**	0.09	0.02	0.05
QLS Role Function	0.26	-0.11	**0.83**	-0.20	-0.17	-0.09
GAS	-0.10	**0.48**	***0.38***	<-0.01	0.12	-0.03
BDI	0.02	0.20	-0.06	-0.03	-0.15	**0.94**
BAI	0.15	-0.19	0.01	0.18	-0.07	**0.62**
BHS	-0.08	0.14	-0.02	0.15	**-0.55**	**0.47**
SOFAS	-0.06	0.08	**0.81**	0.05	0.03	-0.01
EuroQol	-0.04	0.03	-0.06	0.10	**0.43**	***-0.31***
SAPS	0.01	**-1.03**	-0.04	0.07	-0.04	-0.18
SANS	-0.04	0.05	-0.27	**-0.66**	0.14	0.18
PBIQ	0.09	0.09	-0.15	0.13	-0.16	**0.77**
BCSS – Negative Self	-0.02	0.18	0.04	-0.11	<0.01	**0.95**
BCSS – Positive Self	0.12	0.02	-0.08	0.08	**0.74**	-0.14
BCSS – Negative Other	0.01	-0.19	-0.04	-0.13	0.03	**0.46**
BCSS – Positive Other	0.03	0.09	-0.06	0.07	**0.83**	0.06
SSI – Social Anxiety	-0.13	-0.14	0.12	0.10	0.01	**0.57**
SSI – Paranoia	0.03	**-0.50**	0.16	-0.09	0.17	***0.45***
SSI – Anomalous	-0.19	**-0.63**	0.12	0.06	0.19	0.34
PANSS – Positive	-0.01	**-0.97**	-0.04	<0.01	-0.14	-0.20
PANSS – Negative	-0.02	0.04	0.08	**-1.03**	-0.05	-0.09
PANSS – General	0.06	***-0.31***	-0.12	-0.25	**-0.40**	0.19

Note. Loadings can be higher than +/-1 in a promax rotation, as factors are not orthogonal
Loadings above 0.30 are highlighted in bold font, dual loadings are italicised

Table 6.4

Correlations between Recovery Factors

	Activity Levels	Positive Symptoms	Clinician Rated	Negative Symptoms	Resilience/ Optimism	Emotional Barriers
Activity Levels	1.00					
Positive Symptoms	.03	1.00				
Clinician Rated	.35	.26	1.00			
Negative Symptoms	.13	.34	.41	1.00		
Resilience/Optimism	-.11	.27	.23	.16	1.00	
Emotional Barriers	-.01	-.53	-.14	-.15	-.44	1.00

6.5 DISCUSSION

This chapter has outlined a multidimensional model of recovery based on an exploratory factor analysis of commonly used outcome measures for psychosis. It has also examined how schizotypal symptom types relate to different recovery dimensions. This section will review the findings of the current study. The results will initially be considered in relation to each of the research hypotheses outlined at the beginning of this chapter. They will then be discussed in relation to the current literature and the clinical implications of the findings will be examined. Following on from this, potential weaknesses of the study will be outlined, as will possibilities for future research.

6.5.1 Evaluation of Findings in Relation to Research Hypotheses

6.5.1.1 Hypothesis 1

It was hypothesised that recovery would be a multidimensional construct. This hypothesis was supported by an exploratory factor analysis which highlighted six dimensions of recovery. These include: Activity Levels, Positive Symptoms, Negative Symptoms, Clinician-rated Recovery, Resilience/Optimism, and Emotional Barriers. This model fitted the data well, based on both good practice guidelines for EFA (Brown, 2006; Fabrigar et al., 1999) and in relation to current recovery literature.

6.5.1.2 Hypothesis 2

It was hypothesised that different schizotypal symptom types, as measured by the SSI, would exist on different dimensions of recovery. This hypothesis was supported by the findings. The Social Anxiety subscale of the SSI loaded on the Emotional Barriers factor, along with illness cognitions, depression, anxiety, and negative beliefs about self and others. The Anomalous Experiences subscale of the SSI loaded on the Positive Symptoms factor, along with other symptom measures including the SAPS, the positive symptoms subscale of the PANSS, and the Global Assessment of Symptoms scale. The Paranoia subscale of the SSI loaded on both the Emotional Barriers and Positive Symptoms factors. This suggests that the Social Anxiety subscale of the SSI may be

tapping emotional recovery from the episode of psychosis; whilst the Anomalous Experiences subscale may be tapping residual symptoms of psychosis. The Paranoia subscale appears to be tapping both of these constructs.

6.5.2 Relevance to the Literature

The outcome dimensions proposed in this study concur with literature suggesting that recovery from psychosis involves more than just symptom remission (Anthony, 1993; Liberman & Kopelowicz, 2002; J. Lieberman et al., 2008). Indeed, the model highlights symptomatic recovery as one dimension but also takes into account other factors, such as emotional disturbance and functioning. Moreover, it supports the notion that symptomatic and functional recovery are relatively independent of one another (e.g. Tohen et al., 2000) due to the loading of symptom and functioning measures on separate, uncorrelated factors. In addition, the factors outlined in the model correspond with the dimensions of outcome featured in existing operational definitions of recovery. For example, Whitehorn et al's (2002) "symptom control", "autonomous living", and "return to the social and occupational lifeline" are arguably reflected in the Positive Symptoms, Negative Symptoms, Clinician-rated Recovery and Activity Levels factors.

However, as well as supporting operational criteria, the model also provides validity for the use of specific measures in assessing suggested dimensions of recovery. This is an important addition to the literature as many previous studies have argued that recovery research is limited by difficulties in assessing this somewhat abstract and multifaceted concept (Liberman, 2002; G. Shepherd et al., 2008). Indeed, Estroff (1995) suggests:

> The challenge for researchers… is to develop methods and principles that reflect accurately the experiences, meanings and needs of people with severe, persistent, mental illness. The challenge is not to reduce the complexity of the task, but to make it understandable. The reconstitution of lives in a complex process, much of which we fail to find in our outcome research not necessarily because of the bleak course of schizophrenia, but because of conceptual and methodological shortcomings. (p. 87)

Symptomatic recovery is often assessed by scores on the positive subscale of the PANSS (e.g. Liberman et al., 2002; Whitehorn et al., 2002). This subscale had a high and independent loading on the Positive Symptom factor in the current model,

suggesting that it is a valid tool for assessing the remission of full-blown positive psychotic symptoms. Clinician-rated measures such as the SOFAS and QLS are often used to assess functional recovery from psychosis (e.g. Whitehorn et al., 2002). However, this practice was called into question by the findings of the current study. The results of the factor analysis suggest that these measures may be assessing something qualitatively different to actual time spent in structured activity, which loaded on a separate and independent factor. Moreover, the Clinician-rated Recovery factor was highly correlated with the Negative Symptoms factor, implying that measures such as the QLS and SOFAS may be assessing more of a generic "deficit syndrome" rather than actual activity. This supports existing criticisms of the SOFAS, which argue that it does not capture the complexity of "real life" functioning (Wallace, Liberman, Tauber, & Wallace, 2000). In addition, the EuroQol, which is traditionally viewed as a measure of quality of life, loaded with measures assessing positivity and hopefulness. This supports previous literature proposing differences in objective and subjective assessment of quality of life (Narvaez, Twamley, McKibbin, Heaton, & Patterson, 2008); and suggests that careful thought needs to be given to the measures used when defining operational criteria for recovery.

In terms of schizotypal symptoms – the main focus of this thesis – the results of the current study support those outlined in previous chapters and highlight the importance of using a measure of schizotypal symptoms in the assessment of recovery from psychosis. The finding that the Anomalous Experiences subscale of the SSI loaded strongly and independently with positive symptom measures validates the use of this scale in assessing the residual symptoms of psychosis. Moreover, it supports the notion that Anomalous Experiences are not influenced by emotion and psychological variables to the same extent as Paranoia and Social Anxiety. The Social Anxiety subscale loaded with measures tapping emotional distress, negative illness cognitions and negative schematic beliefs about self and others. This supports research suggesting that social anxiety may be reactive, arising from threats associated with the experience of psychosis, i.e. from feelings of shame, personal vulnerability and stigma (Birchwood et al., 1993). Paranoia loaded with both Anomalous Experiences (Positive Symptoms factor) and Social Anxiety (Emotional Barriers factor), suggesting that paranoid beliefs may occur as residual forms of persecutory delusions, and/or as interpersonal concerns

resulting from the psychological impact of psychosis, e.g. “people are looking at me and judging me because they know I have a mental illness”.

The factors highlighted from this exploratory analysis also mirror themes outlined in service user literature on recovery (e.g. Anthony, 1993; Davidson, 2003; Deegan, 1988; Pitt et al., 2007). “Renewing hope and commitment”, “accepting illness” and overcoming the “interpersonal effects” of an episode of psychosis feature heavily in user-defined criteria for recovery from psychosis (Davidson, 2003; Noordsy et al., 2002). Moreover, it has been proposed that these themes may be important mediators of functional recovery (Liberman & Kopelowicz, 2002), and argued that they should feature as central components in recovery oriented services (Anthony, 1993). However, as the majority of these concepts have emerged from qualitative research and service user reports of recovery, it is difficult to incorporate them into standardised assessment frameworks, due to their somewhat subjective nature. Despite this, service user recovery themes are arguably reflected in the Resilience/Optimism and Emotional Barriers factors of the model outlined in this study. This suggests that they can in fact be measured to a certain extent by standardised tools such as the SSI, BHS, BCSS and PBIQ. Further investigation is however required.

6.5.3 Clinical Implications of Findings

The current study has highlighted the complex interplay of factors involved in recovery from a psychotic episode. The findings suggest that focusing on one aspect of recovery in isolation, such as symptom remission, may be inadequate. The proposed multidimensional model could be agued to provide a framework for the assessment of recovery from psychosis, outlining which tools may be most appropriate for assessing particular types of outcome. This framework could also be used to create “recovery profiles”, tailored to the individual and outlining the relative contribution of different dimensions to the wider recovery process. It is likely that individuals will encounter all of the factors associated with recovery from a psychotic episode, but at varying levels of severity and at different time points along the recovery pathway. Thus, all of these factors need to be monitored and responded to appropriately.

This assessment framework may be particularly relevant for the delivery and evaluation of recovery-oriented services for people with severe mental health problems. Central features of these services include: symptom management; developing awareness of early warning signs/relapse plans; adjusting to the psychological impact of the episode; instilling hope for the future; empowering individuals to develop valued relationships and roles; and increasing social and occupational activity (Department of Health, 2001; Jacobson & Greenley, 2001; G. Shepherd et al., 2008; Tauscher-Wisniewski & Zipursky, 2002; Turner-Crowson & Wallcraft, 2002). Such a multidimensional approach supports the multidimensional nature of the model outlined in this study. Indeed, different types of recovery are likely to require different forms of treatment. Suggested interventions include: maintenance doses of medication for residual symptoms; social skills training, psychoeducation, and supported employment (see Mueser, Corrigan et al., 2002 for a review). Evaluating the efficacy of these targeted interventions requires the use of specific tools designed to assess the particular outcome of interest. For example, an intervention designed to instil hope and increase self-esteem would be best assessed by tools loading on the Optimism/Resilience factor outlined in this study (e.g. BHS, BCSS). Conversely, an intervention designed for improving social skills to increase functioning may be best assessed by tools loading on the Activity Levels factor, (i.e. the Time Use Survey).

This study also highlights the importance of assessing schizotypal symptoms in the recovery stages of psychosis. Monitoring schizotypal symptoms may be important when thinking about early warning signs for psychotic relapse. Indeed, in a prospective study of the six-week period preceding relapse in a group of individuals with schizophrenia, subtle increases in "quasi-psychotic phenomena" (i.e. unusual perceptual experiences and unusual thought content as featuring in the Anomalous Experiences and Paranoia subscale of the SSI) were found to predict relapse (Subotnik & Nuechterlein, 1988). However, the authors noted that such slight increases were difficult to assess using traditional assessment tools such as the BPRS (Overall & Gorham, 1962). With this in mind, the Anomalous Experiences and Paranoia subscales of the SSI may be more sensitive in detecting these subtle changes than more traditional assessment tools. These subscales of the SSI may also be useful in the creation of relapse plans (Birchwood, Spencer et al., 2000); and for monitoring the efficacy of interventions designed for psychotic symptom control, for example maintenance doses of antipsychotic medication

(Tauscher-Wisniewski & Zipursky, 2002). Conversely, an intervention designed to target the interpersonal effects of an episode of psychosis, may be best assessed by the Social Anxiety subscale of the SSI, which appears to reflect emotional disturbance associated with psychosis.

6.5.4 Limitations of the Current study and Future Directions

Previous studies have criticised the use of factor analysis in the development of theoretical concepts (Delis et al., 2003). This criticism is mostly based on a study by Armstrong (1967) who created an artificial data set with a known structure and then showed that factor analysis failed to accurately represent this structure. Subsequent studies have however argued that it is not factor analysis per se that is problematic, but rather the way in which it is commonly misused by researchers (Fabrigar et al., 1999). EFA, rather than PCA, is suggested as the best technique for studies investigating latent constructs. In addition, maximum likelihood estimation is proposed as gold-standard methodology as it allows the goodness-of-fit of models to be established. Moreover, it is recommended that researchers use multiple criteria when deciding upon the number of factors to include in a model (e.g. eigenvalues, scree test, and descriptive fit indexes); and to use oblique (rather than orthogonal) rotation techniques, unless it is known a priori that factors are uncorrelated (Brown, 2006; Fabrigar et al., 1999). Thus, it could be argued that the EFA conducted in this study was appropriate and well informed in terms of both statistical methodology and theoretical literature on recovery. As such, the interpretation of the findings can be viewed with relative confidence. Despite this, some limitations do apply. The analysis was based on a relatively small sample size of individuals who were selected on the basis that they had poor social functioning. It has been argued that factor analyses should not be conducted on samples with less than 100 participants (Fabrigar et al., 1999). Furthermore, some data was skewed and this can affect the accuracy of maximum likelihood methods (Curran, West, & Finch, 1996). As a result of these weaknesses, replication studies are required in order to provide further support for the factorial structure that has been proposed.

When interpreting the results, it is important to bear in mind that factor analysis simply highlights correlations existing between different measures and thus any interpretation should be informed by current theoretical knowledge. However, the correlations

highlighted in this study do support an a priori hypothesis that recovery from psychosis is multidimensional. Nevertheless, the model proposed is not definitive and requires validation from further, and larger, confirmatory studies. In addition, there is a need for triangulation with other forms of investigation, such as treatment studies targeting specific dimensions of outcome (Liberman et al., 2002). If such studies showed change on the standardised assessment tools loading on the dimension of interest, this would further validate the model. There are also other outcome measures used in psychosis research which have not been included in this study and which should be investigated as part of developing a more substantive model (e.g. the Independent Living Skills Survey; Wallace et al., 2000). Finally, it must be remembered that psychosis, and thus recovery from psychosis, is extremely heterogeneous, influenced by many internal and personal variables. Numerous studies have identified characteristics which are predictive of recovery from psychosis, including marital status, premorbid functioning, and neuropsychological deficits (Hoffmann & Kupper, 2002; Liberman et al., 2002). These variables also need to be taken into account when considering the process of recovery from psychosis. In particular, future studies should examine how these predictors relate to different dimensions of outcome.

Future studies should also focus on applying this dimensional outcome model to theoretical and explanatory research investigating the process of recovery from psychosis. Indeed, the current study was cross-sectional and thus no conclusions can be drawn regarding temporal aspects of recovery. For example, previous literature suggests that symptomatic improvement occurs first, followed by recovery from the psychological impact of psychosis as a major life event (McGorry, 1992). This could not be investigated in the current study. Moreover, the literature also suggests that certain factors may either impede or promote recovery and thus may act as mediators in the recovery process (Liberman & Kopelowicz, 2002). For example, personal resilience and hope are highlighted as important components of functional recovery from psychosis (Noordsy et al., 2002; Pitt et al., 2007). Conversely, negative illness cognitions and high levels of distress are hypothesised to impede recovery from psychosis (Birchwood et al., 1993). As such, it could be argued that individuals scoring highly on the Emotional Barriers factor may have problems with recovery, whereas individuals scoring highly on the Resilience/Optimism factor may have better recoveries. If this is the case, interventions targeting these constructs may be successful

in facilitating long term recovery from psychosis (see Appendix B for ISREP trial paper). However, longitudinal research needs to be conducted to investigate these potential mediational pathways further. This will be the focus of the final study in this thesis.

6.5.5 Summary

In conclusion, this study has proposed an exploratory six-factor assessment model of different dimensions of recovery from psychosis. This supports the notion that recovery is a multidimensional construct, encompassing both symptomatic and functional recovery, as well as emotional and psychological well-being. The importance of schizotypal symptoms in the recovery process has also been highlighted. The current study provides a useful framework for further investigations of the recovery concept. However, the model requires further validation and replication using confirmatory factor analysis in a larger sample. The final study in this thesis will examine the potential role of schizotypal symptoms as mediators of social recovery.

CHAPTER SEVEN:
STUDY SIX: SCHIZOTYPAL SYMPTOMS AS AN OUTCOME AND MEDIATOR OF COGNITIVE BEHAVIOUR THERAPY FOR IMPROVING SOCIAL RECOVERY IN PSYCHOSIS

7.1 RATIONALE AND CONTEXT FOR THE STUDY

Previous studies in this thesis have highlighted the importance of schizotypal symptoms in recovery from psychosis, both in terms of the disease process itself (i.e. occurring as residual symptoms) and in terms of emotional and psychological recovery from the disorder. A model outlining a potential dimensional approach to recovery has also been postulated, suggesting that the presence of schizotypal symptoms may have an influence on long-term outcome from an episode of acute psychosis. However, thus far these findings have all been demonstrated using a cross-sectional design. As such, the temporal process by which schizotypal symptoms may influence recovery from psychosis is unknown. This study will adopt a longitudinal design, in the context of a randomised controlled trial, in order to investigate this further. It will examine whether an intervention designed to improve social recovery in psychosis has an impact on schizotypal phenomena, and whether changes in schizotypal symptoms mediate changes in social functioning.

As outlined above, data collection for this study took place in the context of a randomised controlled trial. The "Improving Social Recovery in Early Psychosis" (ISREP) study is an MRC-funded platform trial, designed to investigate the efficacy of a new psychosocial intervention to improve social recovery in early psychosis and severe affective disorder. The intervention – Social Recovery oriented Cognitive Behaviour Therapy (SRCBT) – specifically focuses on improving constructive and structured social activity, while managing sensitivity to stress and low-level psychotic symptoms. Thus, it aims to target a range of different dimensions of recovery outlined in the previous chapter. The primary outcome of the trial was hours spent in structured and constructive economic activity. A range of other psychological and intrapsychic outcomes were also assessed, including schizotypal symptoms, hopelessness, symptoms of depression and anxiety, and beliefs about self and others. See Appendix B for the ISREP trial outcome paper.

Past clinical trials of interventions which have attempted to promote social activity without taking careful account of sensitivity to psychosis and anxiety, have shown increased risk of relapse, especially amongst people still experiencing psychotic symptoms (Hogarty, Goldberg, Schooler, & Ulrich, 1974; Hogarty et al., 1997). This fits with stress-vulnerability models of psychosis (Zubin & Spring, 1977) and the suggestion that vocational rehabilitation into competitive employment can be demanding and stressful for individuals with psychosis (Hoffmann, Kupper, & Kunz, 2000; Nithsdale, Davies, & Croucher, 2008). It has been argued that in addition to supported employment, more emphasis should be placed on managing residual symptoms, inducing positive self-concepts, and instilling optimistic but realistic expectations (Mueser, Corrigan et al., 2002; Resnick, Rosenheck, & Lehman, 2004). This approach, adopted by the ISREP trial, may provide the coping strategies and self-confidence required to produce a favourable recovery outcome. Although the main outcome of the ISREP study was weekly hours in constructive and structured activity, the present study aims to evaluate the impact of SRCBT on schizotypal phenomena, using the SSI. In addition, the role of schizotypal symptoms as mediators of change in time use will be investigated. If schizotypal symptoms do indeed play a key role in recovery, changes in these phenomena would arguably be accompanied by changes in activity.

7.2 RESEARCH HYPOTHESES

1. It is predicted that the provision of SRCBT added to treatment as usual (TAU) will have an effect on schizotypal symptoms in comparison to TAU alone.

2. It is predicted that SRCBT but not TAU will lead to changes in levels of schizotypal symptoms, and that these changes may be specifically associated with changes in activity.

7.3 METHOD

7.3.1 Design

The study was a single blind randomised controlled treatment trial (RCT) comparing cases who received SRCBT in addition to TAU (treatment group) with those receiving TAU alone (control group). Participants were randomised to either the treatment or control group following a baseline assessment and initial screening for suitability. Randomisation was stratified for diagnosis (affective/non-affective psychosis) and centre (Norfolk/Cambridgeshire). It is well established that RCTs provide gold-standard methodology to test the effectiveness of new treatments. However, there is an increasing emphasis on RCT methods to examine the process of treatment as well as its outcome (J. Green, 2006; J. Green & Dunn, 2008). This is the focus of the current study.

7.3.2 Participants

Seventy-seven participants were recruited from secondary mental health services in the East Anglia region of the UK, localised around two sites: Norfolk and Cambridgeshire.

7.3.2.1 Sample size and power analysis

The ISREP trial was designed to compare the effectiveness of a new intervention (SRCBT) in comparison to a control condition (TAU). The sample size was predicated on testing the effectiveness of SRCBT on a range of outcome measures with an effect size of .60. A power analysis calculation revealed that to achieve 90% power with a significance level of .05 and an estimated effect size of .60, a minimum sample size of 30 participants per group was required (Cohen, 1988). However, the aim was to recruit beyond this to account for potential drop-outs. It was known that investigations of hypotheses regarding mediators of change would be underpowered and would therefore need to be regarded with caution. However, they were still undertaken as useful preliminary steps in empirically informed exploratory research.

7.3.2.2 Inclusion and exclusion criteria

Inclusion criteria for the study were: (a) a current diagnosis of affective or non-affective psychosis (including schizophrenia, schizoaffective disorder, bipolar disorder, and psychotic depression); (b) illness duration of 8 years or less (onset of illness was defined as the first contact with psychiatric services for psychotic symptoms, checked in case notes); (c) positive psychotic symptoms (hallucinations and delusions) in relative remission (defined by a score of 4 of less on individual symptoms on the PANSS); and (d) currently unemployed or engaged in less that 16 hours per week paid employment or education. Participants were excluded if the psychotic disorder was thought to have an organic basis, acute psychosis was present, or the primary diagnosis was drug dependency on opiates or cocaine.

7.3.2.3 Participant characteristics

Thirty-five participants were randomised to the treatment condition and 42 to TAU, the control condition. Key demographic, clinical and social characteristics of the sample are summarised in Table 7.1. This shows that randomisation resulted in well-balanced groups between treated and control cases in terms of age, gender, ethnicity, diagnosis and illness length, and social characteristics. There were no significant differences on any of these variables between the treatment and control groups. Participant flow throughout the trial is outlined in a CONSORT diagram in Figure 7.1.

7.3.3 Ethical Considerations

As outlined in Study 1 of this thesis, the study was reviewed and approved by the ethics committees of all participating institutions (section 2.4.3). All participants were given an information sheet about the trial and asked to sign a consent form (see Appendix D). A minimum of 72 hours was allowed between giving information about the study and taking informed consent. Any potential participant who was considered by a clinician as unable to give informed consent was not approached to take part.

Table 7.1

Baseline Characteristics of ISREP Participants

	SRCBT (*N* = 35)	TAU (*N* = 42)	Total (*N* = 77)
Demographic characteristics:			
Mean Age in years (*SD*)	27.8 (6.1)	30.0 (7.2)	29.0 (6.8)
Gender (% male)	71.4%	71.4%	71.4%
Ethnicity (% white)	85.7%	95.2%	90.9%
Diagnosis (% non-affective)	65.7%	64.3%	64.9%
Mean illness length in yrs (*SD*)	4.9 (2.2)	4.8 (2.4)	4.8 (2.3)
Medication level in mg (*SD*) (chlorpromazine equivalence)	265.1 (200.8)	223.7 (167.0)	242.2 (182.7)
Social and Clinical characteristics Mean (*SD*):			
Unemployment length in weeks	202.4 (146.0)	214.8 (209.2)	209.1 (182.2)
Time Use in hours per week:			
- Constructive Economic Activity	14.8 (20.2)	10.4 (13.9)	12.4 (17.1)
- Structured Activity	30.4 (19.9)	27.8 (19.2)	29.0 (19.4)
Current IQ	101.8 (11.3)	103.7 (11.3)	102.8 (11.3)
Contacts with Secondary Mental Health Services in last 6 months	32.1 (35.3)	25.9 (23.1)	32.1 (35.3)
Contacts with Voluntary Services in last 6 months	11.0 (18.3)	7.4 (14.4)	9.0 (16.2)

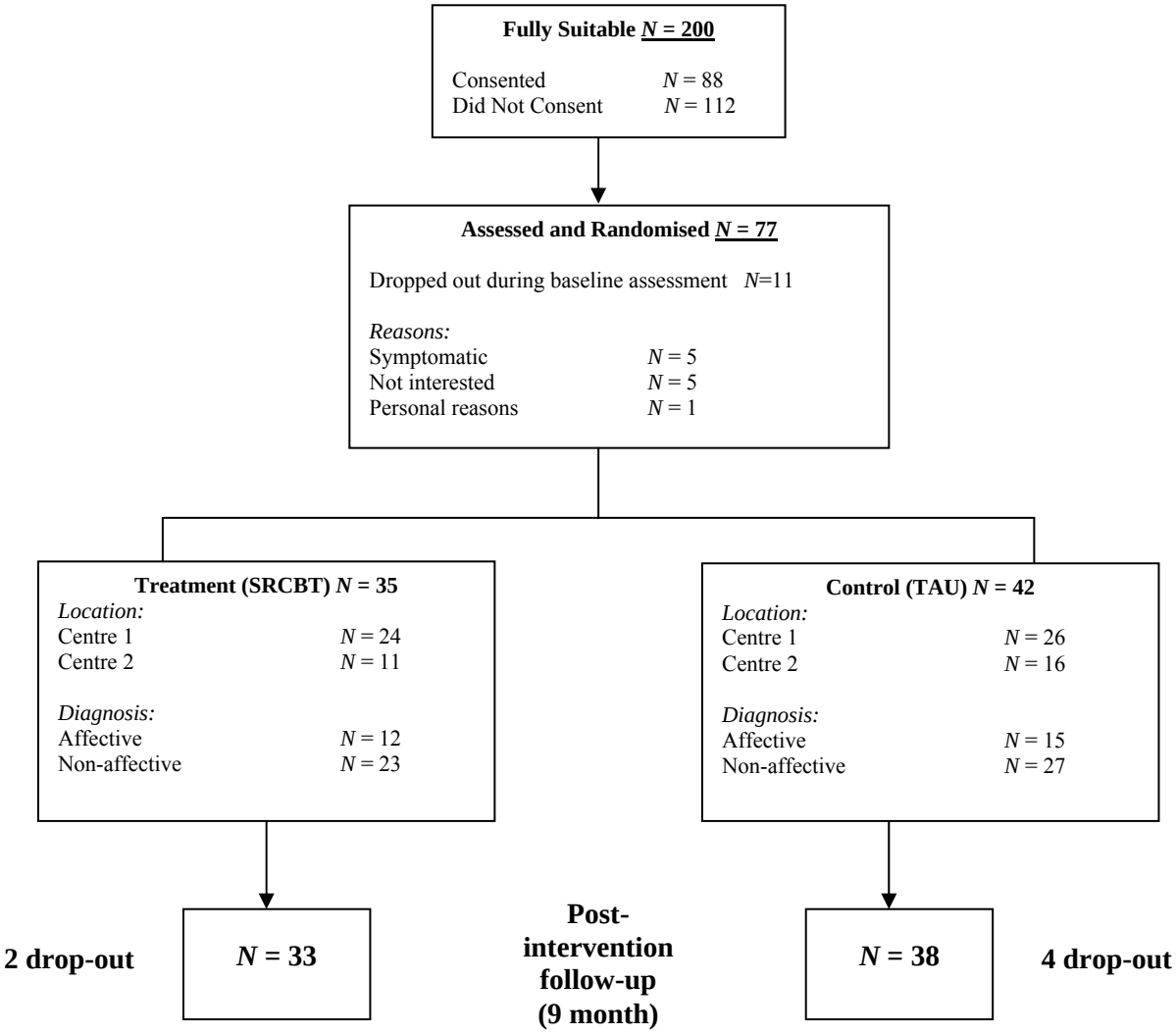


Figure 7.1

CONSORT diagram of participant flow throughout the ISREP trial

7.3.4 Treatments

Details of SRCBT and TAU conditions are outlined below.

7.3.4.1 Social Recovery Cognitive Behavioural Therapy (SRCBT)

Therapy was adapted from the CBT for psychosis manual (Fowler et al., 1998), particularly focusing on aspects promoting social recovery, and was also informed by manuals for cognitive therapy of depression (Beck, Rush, Shaw, & Emery, 1979); and social anxiety (G. Butler, 1999). Therapy in both centres was supervised by experienced CBT specialists. Adherence and competence were monitored using tape recordings and individual and group supervision. Participants received a mean of 14 sessions (*SD* = 7). For a more detailed description of SRCBT, see trial outcome paper (Appendix B)

7.3.4.2 Treatment as Usual (TAU)

Active case management was provided by multidisciplinary secondary care mental health teams as treatment as usual. This consisted of multidisciplinary case management, and was backed by the availability of services to provide supported employment for people with severe and enduring mental health problems. There were no significant differences in the level of support given to treated cases and controls at baseline (see Table 7.1), $t(75) = -1.3$, $p = .20$.

7.3.5 Measures

The sample completed a range of measures at both baseline and post-treatment. The primary outcome of the ISREP trial was weekly hours in structured and constructive economic activity, measured by the Time Use Survey (TUS; adapted from Short, 2006) as described in the previous chapter (section 6.3.3). However, for the purpose of this study, schizotypal symptoms measured by the brief version of the Schizotypal Symptoms Inventory (SSI) were the main outcome of interest. The SSI has been

described in detail elsewhere in this thesis and as such a description will not be repeated here. Total scale and subscale scores were used in the analyses.

Other measures administered in the study included the Positive and Negative Syndrome Scale (PANSS; Kay et al., 1987); the Quality of Life Scale (QLS; Heinrichs et al., 1984); the Beck Hopelessness Scale (BHS; Beck & Steer, 1988); the Beck Depression Inventory (BDI-II; Beck et al., 1996); the Beck Anxiety Inventory (BAI; Beck & Steer, 1987); the Brief Core Schema Scales (BCSS; Fowler, Freeman, Smith et al., 2006); the Personal Beliefs about Illness Questionnaire (PBIQ; Birchwood et al., 1993); the EuroQol visual analogue scale (EQ-VAS; Brooks, 1996); the Global Assessment of Symptoms Scale (GAS; American Psychiatric Association, 2000b); and the Social and Occupational Functioning Assessment Scale (SOFAS; Goldman et al., 1992). All of these measures have been described in detail in previous chapters of this thesis.

7.3.6 Procedure

Participants were assessed at two time points by a researcher who was blind to the outcome of randomisation and thus did not know if the participant had received therapy or not. The baseline assessment (T1) occurred after participants had consented to take part in the study and before randomisation. The post-treatment assessment (T2) took place at the end of the study, approximately nine months after the baseline assessment. For those individuals who did not wish to attend a post-treatment assessment appointment but who were willing to still be involved in the project, assessments took place over the telephone and were also triangulated with discussions with carers and/or care co-ordinators.

Baseline and post-treatment assessments were conducted by research assistants who were independent of treatment delivery and randomisation. Every effort was made to ensure they were kept blind to allocation. Formal training in all measures was provided and interviews were audio-taped for reliability and quality control. Research assistants met regularly throughout the trial to maintain reliability of procedures and ratings. Where blindness was broken, another research assistant conducted the follow-up assessment. Ninety-three percent of the post-treatment assessments were completed blind. The research assistants made allocation guesses after post-treatment assessments.

These were correct for treatment (SRCBT) for 58% and for control (TAU) guessed correctly for 64%. This is within the levels that would be expected by chance and thus blindness can be considered successful.

7.3.7 Data Analysis Plan

Data was cleaned and screened as outlined in Study 1 (section 2.4.6.1). Descriptive statistics were then computed for all variables. All data was analysed using SPSS for Windows, version 14.0 (SPSS, 2005).

Formal analyses and statistical testing were conducted on an intention-to-treat (ITT) basis using Analysis of Covariance (ANCOVA) models. These analyses allow for the presence of missing outcome data under the assumption that the data are missing at random conditional on the covariates included in the regression model (i.e. allocation, schizotypal symptoms, length of unemployment, and baseline values of the outcome variables). The sensitivity of the results to departures from this assumption was checked for those analyses indicating a significant treatment effect. For these analyses, missing data at follow up was imputed using an expectation-maximisation (EM) model.

7.3.7.1 Hypothesis 1

ANCOVA models were used to test the significance of differences between the treatment and control groups for all outcome measures. This study specifically focuses on the effects of treatment on schizotypal symptoms. A separate ANCOVA was conducted for each subscale of the SSI, using the post-treatment (T2) score on the measure as the dependent variable. Allocation to treatment, centre and diagnosis were used as fixed factors; and two key variables assumed to be associated with outcome and predictive of drop-out were used as covariates. The covariates were: baseline (T1) score on the measure (e.g. schizotypal symptoms at baseline); and length of unemployment. Non-significant interactions were removed before testing for main effects.

7.3.7.2 Hypothesis 2

A further set of ANCOVAs were conducted in order to examine the effect of schizotypal symptoms as mediators of change in social functioning in the context of SRCBT. For each ANCOVA, hours in structured activity at post-treatment (T2) was used as the dependent variable, with baseline (T1) hours in structured activity as a covariate. Allocation to treatment, and change in the mediating variable (i.e. T2-T1 change in schizotypal symptoms) were then included as explanatory variables. If a significant interaction was found between allocation and change in schizotypal symptoms, this was interpreted as suggesting that schizotypal symptoms may mediate the effect of SRCBT on functioning (i.e. the intervention may enhance change in that variable). If this interaction was not significant then it was removed and the analysis was repeated. A significant main effect of change in schizotypal symptoms on activity would highlight a longitudinal relationship between these two variables (i.e. change in one variable influences change on another, but not specifically in the context of therapy).

7.4 RESULTS

7.4.1 Descriptive Data

Outcome data on activity was available for 92% of the recruited sample. This information was obtained via a combination of face-to-face and telephone interview, conversations with case managers, and case note information. Eighty percent of the sample completed post-treatment face-to-face interview. Questionnaire assessments were available for around 75% of the sample. Descriptive statistics (means and standard deviations) are shown in Table 7.2 for the SSI and in Table 7.3 for all other variables. For the SSI, means and standard deviations are provided for baseline and post-treatment assessments and also for participant change scores (T2-T1). The descriptive statistics are broken down by treatment and diagnostic group at baseline and post-treatment (9 months) and derive from analyses of the cases available at post-treatment assessment

(i.e. the completers). Data for some variables was skewed and was resistant to normalising transformation.

Table 7.2

Descriptive Statistics for SSI Scores by Treatment and Diagnosis – Mean (SD)

		Total Sample (*N* = 68)		Non-Affective (*N* = 45)		Affective (*N* = 23)	
		TAU (*N* = 38)	CBT (*N* = 30)	TAU (*N* = 25)	CBT (*N* = 20)	TAU (*N* = 13)	CBT (*N* = 10)
Total	0	21.7 (14.5)	15.9 (17.5)	24.8 (16.5)	10.5 (9.3)	15.8 (6.7)	26.6 (2.8)
	9	24.8 (16.6)	18.6 (12.8)	26.0 (18.4)	18.0 (12.3)	22.8 (13.8)	19.7 (14.4)
	Change	2.1 (15.5)	6.1 (11.5)	-0.5 (17.6)	10.0 (9.8)	6.5 (10.6)	-1.1 (11.6)
Social Anxiety	0	10.8 (6.1)	6.2 (6.0)	10.8 (6.4)	4.1 (4.1)	10.8 (5.7)	10.4 (7.1)
	9	10.1 (6.3)	9.9 (6.7)	9.3 (6.2)	10.3 (7.0)	11.3 (6.5)	9.2 (6.6)
	Change	-0.9 (5.6)	5.2 (7.1)	-1.7 (6.0)	7.6 (6.3)	0.5 (4.7)	0.6 (6.5)
Paranoia	0	6.5 (6.1)	5.0 (6.6)	8.0 (6.7)	3.3 (3.7)	3.6 (3.2)	8.5 (9.7)
	9	7.4 (5.8)	5.2 (5.1)	8.5 (6.4)	4.6 (4.3)	5.7 (4.6)	6.1 (6.6)
	Change	0.8 (5.2)	1.0 (4.0)	0.2 (6.0)	1.7 (3.5)	1.8 (3.6)	-0.5 (4.6)
Anomalous Experiences	0	4.4 (6.5)	4.7 (6.2)	6.0 (7.4)	3.2 (3.2)	1.3 (1.8)	9.3 (2.9)
	9	7.3 (6.6)	3.6 (4.5)	8.2 (7.2)	3.1 (4.2)	5.8 (5.4)	5.2 (1.7)
	Change	2.2 (7.4)	0.0 (4.5)	1.0 (8.7)	0.7 (2.5)	4.2 (4.6)	-1.3 (6.8)

Note. TAU = Treatment as Usual; SRCBT = Social Recovery-oriented Cognitive Behaviour Therapy

Table 7.3

Descriptive Statistics for ISREP Trial Outcome Variables by Treatment and Diagnosis – Mean (SD)

		Total Sample		Non-Affective		Affective	
		TAU	SRCBT	TAU	SRCBT	TAU	SRCBT
Structured Activity	0	27.9 (19.2)	30.4 (19.9)	27.7 (20.0)	25.1 (10.9)	28.2 (18.4)	40.6 (28.5)
	9	34.4 (20.6)	40.0 (22.8)	31.8 (21.3)	37.1 (17.2)	39.8 (18.9)	45.4 (31.2)
Constructive Economic Activity	0	10.4 (13.9)	14.8 (20.2)	8.7 (13.3)	10.3 (7.3)	13.6 (14.7)	23.6 (32.1)
	9	15.6 (15.9)	19.2 (21.0)	11.9 (13.6)	14.7 (12.9)	22.4 (18.1)	28.6 (30.6)
PANSS Total	0	56.0 (10.3)	57.6 (11.6)	58.1 (9.4)	57.5 (10.8)	52.1 (11.0)	58.0 (13.4)
	9	50.4 (10.1)	50.5 (9.2)	53.2 (8.3)	50.3 (8.2)	44.5 (11.3)	50.7 (11.3)
QLS Total	0	62.7 (14.8)	66.8 (14.8)	58.2 (11.0)	64.1 (10.2)	70.7 (17.5)	71.7 (20.5)
	9	72.5 (18.5)	76.1 (14.0)	67.1 (15.0)	72.8 (12.3)	83.8 (20.5)	82.3 (15.5)
QLS Role Function	0	5.6 (3.8)	6.6 (4.1)	4.6 (2.9)	5.8 (3.5)	7.4 (4.6)	8.2 (4.9)
	9	7.2 (5.7)	9.0 (5.6)	6.1 (5.3)	8.3 (5.6)	9.5 (5.9)	10.5 (5.4)
Beck Hopelessness Scale	0	8.7 (5.8)	8.9 (5.8)	8.0 (5.5)	8.3 (5.5)	10.2 (6.4)	10.2 (6.3)
	9	7.9 (5.8)	6.4 (4.7)	8.2 (5.9)	4.9 (2.3)	7.3 (5.9)	9.3 (6.6)
BCSS – Negative Self	0	6.7 (6.3)	5.5 (5.1)	6.4 (6.7)	4.0 (3.5)	7.2 (5.8)	8.3 (6.5)
	9	4.9 (4.4)	4.0 (5.3)	4.2 (3.8)	2.7 (2.3)	5.9 (5.2)	6.2 (7.8)
BCSS – Positive Self	0	9.2 (6.4)	8.4 (5.6)	8.8 (6.8)	8.6 (5.5)	10.0 (5.8)	8.1 (6.1)
	9	10.6 (6.8)	11.9 (6.0)	10.0 (7.6)	11.6 (5.7)	11.5 (5.3)	12.3 (6.8)
BCSS – Negative Other	0	6.7 (6.6)	6.6 (6.3)	7.7 (7.1)	5.7 (5.7)	4.8 (5.2)	8.1 (7.2)
	9	4.9 (4.6)	3.5 (4.2)	4.3 (3.8)	2.9 (3.6)	5.8 (5.8)	4.3 (5.2)
BCSS – Positive Other	0	11.7 (6.5)	8.9 (5.5)	11.2 (6.9)	8.5 (5.4)	12.6 (5.6)	9.6 (5.8)
	9	10.0 (6.1)	11.9 (6.2)	9.5 (6.5)	12.4 (5.9)	10.8 (5.6)	11.2 (6.8)
SOFAS	0	48.9 (7.9)	51.5 (9.0)	47.3 (6.8)	50.1 (6.8)	51.8 (9.1)	54.2 (12.1)
	9	53.8 (12.3)	54.8 (9.4)	51.5 (11.3)	53.7 (9.2)	58.3 (13.3)	56.9 (10.1)
EuroQol	0	57.5 (21.8)	52.9 (25.4)	55.7 (22.5)	58.4 (21.9)	60.5 (21.2)	42.3 (29.2)
	9	65.7 (18.2)	65.8 (19.8)	67.2 (16.9)	67.9 (13.9)	63.7 (20.4)	62.0 (27.9)
Personal Beliefs About Illness	0	21.8 (7.6)	21.2 (7.6)	22.5 (8.4)	19.8 (7.3)	20.4 (5.6)	23.9 (7.7)
	9	19.7 (6.8)	18.9 (6.2)	19.9 (7.2)	17.8 (4.0)	19.2 (6.4)	20.6 (8.8)
Global Assessment of Symptoms	0	57.2 (8.8)	56.4 (10.9)	55.4 (8.6)	56.7 (8.9)	60.5 (8.4)	55.8 (14.4)
	9	60.2 (14.1)	59.2 (10.9)	56.6 (12.6)	58.5 (10.9)	67.9 (14.6)	60.7 (11.0)
Beck Depression Inventory	0	22.6 (13.8)	21.1 (13.9)	21.4 (14.4)	17.9 (11.3)	24.7 (12.8)	27.0 (16.5)
	9	14.4 (12.7)	13.6 (10.6)	14.3 (11.5)	11.3 (7.5)	14.7 (14.9)	17.2 (14.0)
Beck Anxiety Inventory	0	17.0 (11.8)	16.9 (13.5)	16.6 (13.0)	14.8 (12.8)	17.7 (9.8)	21.1 (14.5)
	9	13.2 (10.5)	13.0 (12.8)	12.3 (9.7)	11.6 (11.9)	14.7 (12.0)	15.3 (14.6)

Note. TAU = Treatment as Usual

SRCBT = Social Recovery-oriented Cognitive Behaviour Therapy

7.4.2 Outcome Analyses

7.4.2.1 Combined group (non-affective and affective psychosis)

Table 7.3 shows that all participants made large improvements in most domains, including activity and symptoms, as a result of both CBT and TAU interventions. Outcome for variables outlined in Table 7.3 are discussed in detail in the trial outcome paper (Appendix B). To summarise, ANCOVAs on these variables highlighted a significant main effect of treatment on both positive beliefs about self and positive beliefs about others.

When examining the descriptive data for schizotypal symptoms (Table 7.4), it appears that total SSI scores increased in both TAU and CBT groups over the course of the trial. It must also be noted that the CBT group had a lower baseline total SSI score, although this difference did not reach statistical significance, $t(66) = 1.50$, $p = .14$. When broken down into subscales, it is specifically Social Anxiety scores which increased in the CBT group (adjusted effect size = 0.95), and Anomalous Experiences scores which increased in the TAU group (adjusted effect size = 0.40).

Analyses of the main effects of CBT treatment on schizotypal symptoms for the combined group using ANCOVAs showed no significant effect of treatment on total SSI scores, $F(1, 46) = 0.06$, $p = .80$; or scores on the paranoia subscale, $F(1, 46) = 0.31$, $p = .58$. However, a trend was found for a main effect of treatment on the anomalous experiences subscale of the SSI, $F(1, 46) = 3.75$, $p = .06$. Moreover, a treatment by diagnosis interaction was found for the social anxiety subscale, $F(1, 42) = 4.16$, $p = .05$. Further investigations of the effects of treatment within each diagnostic group are therefore reported below.

7.4.2.2 Non-affective psychosis group

The non-affective group consisted of 50 cases (23 treatment, 27 controls) for whom 47 post-treatment assessments were available (22 treatment, 25 control). Descriptive results

are reported in Tables 7.2 and 7.3. ANCOVAs for primary, secondary and tertiary outcome variables in the non-affective sample are described in detail in the trial outcome paper (Appendix B). To summarise, the findings of these analyses indicated a positive main effect of treatment on constructive economic activity, structured activity, PANSS total scores, and positive beliefs about self. There were also trends indicating a main effect of treatment on improvements in hopelessness and QLS instrumental role functioning.

In terms of schizotypal symptoms, total SSI scores increased in both CBT and TAU arms of the non-affective group over the course of the trial. As with the combined group, the main increase in the non-affective CBT subgroup was on the Social Anxiety subscale (adjusted effect size = 1.5). Analyses of the main effects of CBT treatment for the non-affective group using ANCOVAs showed that there was a significant main effect of allocation to therapy on the Social Anxiety subscale of the SSI, $F(1, 27) = 6.37$, $p = .02$. In line with the descriptive statistics, this suggests that there was a significant increase in Social Anxiety SSI subscale scores in those individuals who received therapy, compared to those in the control group. There were no significant effects of allocation on the Paranoia or Anomalous Experiences subscales of the SSI.

7.4.2.3 Affective psychosis group

The affective group consisted of 27 cases (12 treatment, 15 controls) for whom 24 post-treatment assessments were available (11 treatment, 13 controls). Unsurprisingly given the small sample size, there were no significant effects on any of the outcome variables. However, there were striking improvements in activity levels in both the CBT and TAU arms of the affective psychosis group.

In terms of schizotypal symptoms, descriptive statistics in Table 7.2 show suggestions of effects favouring SRCBT on reductions in SSI scores. In the CBT arm there were post-treatment reductions on all dimensions of the SSI. This is in comparison to the TAU arm where SSI scores increased on all dimensions. However, the sample size was too small to analyse meaningfully and no significant effects of treatment on schizotypal symptoms were found in the affective psychosis group.

7.4.3 Mediation Analysis

A significant interaction was found between allocation to therapy and change in schizotypal symptoms (total SSI scores) on weekly hours in structured activity at post-treatment, $F(1, 45) = 10.30$, $p = .002$. This mediation analysis suggests that schizotypal symptoms increased more in the CBT group in response to increases in structured activity, compared to the control group. More specifically, this mediation effect was found to be significant for the Social Anxiety subscale of the SSI, $F(1, 45) = 16.17$, $p < .001$, but not for other SSI subscales. When non-significant schizotypal symptom change by allocation interactions were removed for the Paranoia and Anomalous Experiences subscales, there were no significant main effects of schizotypal symptom change on activity. These findings imply a moderating effect of therapy on schizotypal symptoms, specific to the Social Anxiety dimension and not the Paranoia or Anomalous Experiences dimensions.

7.4.4 Missing Data Analysis

A sensitivity analysis of the results was conducted by repeating ANCOVA analyses using the EM estimates for missing data. These were consistent with those using only completers for all variables. In the combined group the EM ANCOVA analyses showed a main effect of treatment allocation on Anomalous Experiences scores, $F(1, 71) = 4.39$, $p = .04$; and a treatment by diagnosis interaction on Social Anxiety scores, $F(1, 67) = 4.16$, $p = .05$). In the non-affective subgroup the EM ANCOVA analyses showed a main effect of treatment allocation on Social Anxiety scores, $F(1,45) = 3.95$, $p = .05$). The findings of the mediation ANCOVA analyses were also replicated using EM estimates, for both total SSI scores and scores on the Social Anxiety subscale of the SSI. No additional significant main or interaction effects were found using EM estimates rather than data from completers. This suggests that the completer analyses were not biased by missing data.

7.5 DISCUSSION

7.5.1 Summary of Results

The full results of the trial are explained in detail in the trial outcome paper (Appendix B). In summary, the trial provided no clear evidence for the benefit of CBT on activity in a combined sample of patients with both affective and non-affective psychosis. However, some evidence was revealed for the potential of CBT to improve constructive economic and structured activity amongst a more homogenous sample of patients with non-affective psychosis. There was an average gain of 12 hours per week in structured activity for CBT in comparison to 4 hours for TAU in the non-affective psychosis group. This was achieved in association with clinically meaningful and significant improvements in symptoms (PANSS) and beliefs about self and others (BCSS). The results for affective psychosis were less clear. However, the size of this group was small and thus clear conclusions cannot be drawn.

Although the primary outcome of the ISREP trial was activity, the current study has focused on the impact of SRCBT on schizotypal symptoms; and whether a change in these symptoms mediates the effect of therapy on activity. The results suggest that certain types of schizotypal symptoms are important in recovery and should therefore be considered in recovery-oriented interventions. It was predicted that SRCBT would lead to changes in schizotypal symptoms and that these changes may be specifically associated with changes in activity. These hypotheses were supported by the findings. In general there was an increase in total SSI scores in both TAU and CBT arms in the combined non-affective and affective psychosis group over the course of the trial. However, this change did not reach statistical significance. When examining change on the individual subscales of the SSI, Social Anxiety scores were found to significantly increase in the non-affective intervention group over the course of the trial when compared to the non-affective control group. Moreover, mediation analyses showed that there were larger increases in social anxiety in the CBT group in response to increases in activity when compared to the control group. The finding that SSI scores increased in line with increasing activity in the treatment group could be taken to suggest that

schizotypal symptoms, particularly social anxiety, increased as a result of individuals in the treatment arm engaging in more structured activity.

This is not to say that it is necessary for schizotypal symptoms to increase in order for there to be gains in activity. Rather it is more likely that increases in social anxiety were a by-product of the therapeutic techniques used to assist individuals in engaging in new activities. Nevertheless, this finding does highlight a relationship between social recovery (measured in terms of weekly hours in activity) and social anxiety schizotypal symptoms. Based on this, it could be argued that a targeted intervention for social anxiety may enhance improvements in social recovery. However, further studies would need to be conducted in order to confirm this hypothesis. The finding that changes in schizotypal symptoms were confined to the Social Anxiety subscale of the SSI, and not Paranoia or Anomalous Experiences, suggests that social anxiety may have a specific role in social recovery from psychosis. Paranoia and anomalous experiences may be related to other dimensions of recovery, as outlined in the previous chapter. Future research should focus on investigating the relationships between different types of schizotypal symptoms and different outcome dimensions using targeted interventions.

7.5.2 Relevance to the Literature and Clinical Implications of Findings

The increases in social anxiety found in the current study need to be contrasted to findings of other studies which have suggested that vocational interventions may increase risk of relapse (Hogarty et al., 1974; Hogarty et al., 1997). Only social anxiety increased in the treatment group with other types of schizotypal symptoms, including paranoia and anomalous experiences, remaining stable. This is in contrast to the control group who showed a significant increase in Anomalous Experiences scores over the course of the trial. Moreover, it is somewhat expected that social anxiety would increase in line with increases in activity, particularly when considering that the sample had a very low level of activity at baseline, were socially isolated, and had been out of work or education for a mean of 4 years. Any increase in activity, particularly that of a social nature, is thus likely to act as a stressor, producing some short-term anxiety (Phillips, Francey, Edwards, & McMurray, 2007). However, whilst increasing anxiety, it is important to note that paranoia or anomalous experiences did not increase in conjunction with increases in social anxiety. Moreover, there was a significant reduction

in total PANSS scores in the CBT arm. There were also reductions in anxiety measured by the BAI. These findings suggest that the intervention successfully managed residual psychotic symptoms whilst increasing time spent in structured activity, despite an increase in social anxiety.

In line with the mediation analysis, the largest increase in social anxiety occurred in the subgroup with the largest gain in activity: the CBT arm of the non-affective psychosis group. Although this fits with the hypothesis that individuals with psychosis have an increased sensitivity to stress (e.g. Myin-Germeys, van Os, Schwartz, Stone, & Delespaul, 2001), these findings must also be considered in light of the nature of the intervention. The aim of the CBT intervention was to assist individuals in adopting new social and occupational activities. This often involved the use of structured active behavioural interventions, such as exposure work, which could potentially lead to short-term increases in anxiety (Bennett-Levy, Butler, Fennell, & Hackmann, 2004). If associated with recent engagement in new activities, it may be the case that this anxiety diminishes over time as individuals habituate to their new roles. A longer follow-up period is required in order to investigate this further. Moreover, it must be remembered that increases in the Social Anxiety dimension of the SSI were somewhat offset by improvements in symptoms, hope, and positive beliefs about self and others. These improvements could be taken as support for the cognitive model underpinning the intervention, which had a deliberate focus on fostering positive self-esteem and hope, while working toward adopting new social activities.

Despite the above, given the increased stress sensitivity of this sample, and findings postulating social anxiety as a precursor to paranoia (Freeman & Garety, 2003; Freeman, Garety et al., 2005; Freeman et al., 2008; M. J. Green & Phillips, 2004); it is important that such symptoms are monitored and managed. The SSI is a useful measure for this purpose. Indeed, the SSI appears to be sensitive in assessing changes in specific dimensions of low-level psychotic-like experiences. This is contrast to the PANSS which showed an overall improvement in the treatment group over the course of the trial, despite the increase in social anxiety. Moreover, the Social Anxiety dimension of the SSI is specific to the types of anxiety relevant to individuals in recovery from psychosis. This is in contrast to more generic assessments of anxiety, such as the BAI, which showed a general improvement in individuals over the course of the trial.

A further point of interest is the difference in schizotypal symptom change over the course of the trial between affective and non-affective subgroups. Although the non-affective intervention subgroup displayed an increase in social anxiety symptoms in comparison to non-affective controls; the opposite pattern was seen in the affective group. Indeed, affective treatment cases experienced a decrease in symptoms, whilst affective control cases experienced an increase. However, it should be noted that there was a large baseline difference in SSI scores between the affective control and treatment subgroups, with the treatment group scoring more highly. Moreover, the sample size of the affective group is too small to warrant formal conclusion. Despite this, differences in schizotypal symptoms between affective and non-affective psychoses may be an area for further research, particularly considering previous findings that individuals with affective psychosis have a better general social recovery course than individuals with non-affective psychosis (Macmillan et al., 2007).

7.5.3 Limitations of the Current Study and Future Directions

There are a number of considerations which should be borne in mind when interpreting the results of this study. First, the sample size is not large enough to draw formal conclusions about the exact role of schizotypal symptoms in the recovery process from an episode of psychosis. The ISREP trial was designed to be exploratory rather than confirmatory and lacks power to detect effects, particularly within diagnostic subgroups. Results for the non-affective group are therefore suggestive and those for the affective group are too small to warrant any formal conclusion. However, the study does provide an indication of hypotheses for further investigation in future and larger studies. Moreover, it confirms the usefulness and sensitivity of the SSI in assessing change in low-level psychotic-like phenomena and social anxiety. This is in contrast to more traditional measures such as the PANSS and BAI.

A further weakness of the current study is the baseline differences in SSI scores between the treatment and control groups. Despite randomisation, the treatment group generally reported lower baseline SSI scores than the control group. On some dimensions of the SSI, post-treatment scores are comparable between treatment and control groups, with between-group differences only noticeable when controlling for

baseline scores (i.e. in change scores). This could be taken to suggest that within both groups there is a degree of "regression toward the mean" occurring, with control group scores decreasing from their baseline level and treatment group scores increasing from their baseline level (Bland & Altman, 1994). However, this is not the case on all schizotypal symptom dimensions and it must also be remembered that baseline differences in schizotypal symptoms did not reach statistical significance. Nevertheless, further replication studies are required in order to confirm the current findings.

A further limitation of this study concerns the statistical methodology used to conduct the mediation analyses. In line with traditional approaches (e.g. R. M. Baron & Kenny, 1986; Kraemer, Fairburn, & Agras, 2002), these analyses rely on the assumption that no hidden confounding exists between mediator and outcome variables. That is, the analyses ignore the possibility that another unmeasured variable may influence both schizotypal symptoms and time spent in structured activity. This approach also ignores the presence of measurement error in the assessment of mediator and outcome variables. Whilst problematic, these same criticisms are true of other treatment studies assessing mediation using traditional approaches (e.g. Wykes et al., 2007). Moreover, the current study provides a suggestion of future mediation hypotheses and also confirms the theoretical underpinning of the intervention tested. That said, in order to control for potential confounding, newer mediation approaches need to be adopted, such as those outlined by Dunn and Bentall (2007) and Ten Have et al. (2007). These methods require large sample sizes and for potential confounders to be identified in the study design stages. This was beyond the scope and power of the current study, but should be borne in mind for future study designs.

7.5.4 Summary

The current study has highlighted the effect of an intervention designed to improve social recovery from psychosis, on the presence of schizotypal symptoms. Changes in activity were specifically associated with increases in scores on the Social Anxiety dimension of the SSI. However, there was no increase in acute psychotic symptomatology and increases in social anxiety were offset by gains in hope and positive beliefs about self and others. The findings support those of previous studies in this thesis and suggest that the management of low-level psychotic symptoms is

important in recovery, even when the more florid symptoms of psychosis have remitted. The study provides preliminary evidence for the role of schizotypal symptoms in social recovery from psychosis. Future research should focus on investigating differential relationships between subtypes of schizotypal symptoms and other dimensions of recovery.

CHAPTER EIGHT: DISCUSSION

8.1 OVERVIEW

This thesis highlights the importance of investigating the role of schizotypal symptoms in psychosis. This is a novel approach in the context of existing literature in this field which tends to conceptualise schizotypy as a normally distributed personality trait, highlighting potential vulnerability to the development of psychosis. This thesis argues that although trait schizotypy may reflect risk of psychosis onset, state schizotypal symptoms may be at the core of the disorder, existing not only premorbidly but throughout the course of psychotic illness and into the recovery phase. As such, investigating the underlying mechanisms of schizotypal phenomena may provide a deeper insight into the nature of psychosis than the study of acute symptoms, which may be a transient "flare-up" of a long-standing underlying problem.

This chapter will first summarise the findings of the six studies conducted in this thesis before relating them to a psychological model of recovery from psychosis. Clinical implications of the findings will then be discussed as will possibilities for future research.

8.2 SUMMARY OF FINDINGS

8.2.1 Study 1

The first study in this thesis described the creation of the Schizotypal Symptoms Inventory (SSI), a revised version of the Schizotypal Personality Questionnaire (SPQ) designed for use in assessing the current presence and frequency of low-level psychotic symptoms in clinical and non-clinical populations. The SSI assesses schizotypal symptoms over three domains: Social Anxiety, Paranoia, and Anomalous Experiences. This is advantageous as allows a range of symptom types to be assessed simultaneously, and thus potential differential associations with explanatory variables to be examined.

The SSI has been shown to have good internal and test-retest reliability; and good validity with more traditional assessments of psychotic symptoms (i.e. PANSS). The long version of the SSI was shown to have a four-factor structure (social anxiety, paranoia, anomalous experiences, and disorganisation). This was reduced to three factors in the development of a brief version of the scale, due to the finding that clinical and non-clinical samples could not be discriminated using the disorganisation subscale. The distribution of symptom counts on the SSI were shown to be half-normal. This is in contrast to the normal distribution of symptom counts on the original SPQ and suggests that the SSI has a more clinical focus (Johns & van Os, 2001). In addition to demonstrating the robust psychometric properties of the SSI, Study 1 compared levels of schizotypal symptoms in clinical and non-clinical samples. In line with the continuum hypothesis, schizotypal symptoms were not uncommon in the non-clinical sample. However, their presence was significantly higher in the clinical sample. Social Anxiety symptoms were found to be the largest discriminator between clinical and non-clinical samples, whilst Anomalous Experiences were relatively rare in both groups. This suggests that social anxiety is a large problem in individuals recovering from acute psychosis.

8.2.2 Study 2

The underlying mechanisms of schizotypal symptoms were investigated in Study 2 by examining associations between SSI scores and visual processing in a large sample of individuals with psychosis, using the McCollough Effect visual illusion paradigm (ME; McCollough, 1965). Previous research has suggested that anomalous experiences are associated with a basic cognitive dysfunction, which provides a basis for the later development of hallucinations and delusions (J. A. Gray, 1998b; J. A. Gray et al., 1991; Hemsley, 2005b). Antipsychotic medication is hypothesised to target this dysfunction (Kapur, 2003). It is proposed that a failure in automatic processing results in the allocation of attention to details of the environment which would not normally reach awareness, also known as *mismatches*, thus producing anomalies in perception (Corlett et al., 2007; Hemsley, 2005a). Perceived strength of the ME illusion was used as an index of sensitivity to these mismatches.

Increased strength of the ME illusion was found to significantly correlate with increased scores on the Anomalous Experiences and Paranoia subscales of the SSI, but not with scores on the Social Anxiety subscale. Thirty-seven percent of the clinical sample did not perceive the ME illusion at all. This is a significant proportion given that the effect is considered to be a phenomenon of "normal" visual perception. Further analyses showed that those who did not perceive the ME were receiving a significantly higher dose of antipsychotic medication than those who did perceive the effect, suggesting that antipsychotic medication may block perception of the illusion. Taken together, these findings could be argued to provide preliminary evidence to support the notion that a basic cognitive dysfunction may be at least partially responsible for the presence of certain subtypes of schizotypal symptoms, namely anomalous experiences and low-level paranoia, but not social anxiety.

8.2.3 Study 3

Study 3 examined relationships between schizotypal symptoms and emotional and psychological variables in a non-clinical sample. The aim of this study was to further explore potential mechanisms underlying different types of schizotypal symptoms. The use of an analogue sample allows this to be investigated without the influence of medication effects or other factors associated with the disease process of psychosis (Raine & Lencz, 1995). Higher SSI scores were found to be associated with increased anxiety and depression, increased negative beliefs about self and others, and decreased positive beliefs about self and others. All types of schizotypal symptoms were significantly associated with emotional and psychological variables. However, correlations were stronger for the Social Anxiety and Paranoia subscales than the Anomalous Experiences subscale.

In order to unpick the nature of the relationships between different types of schizotypal symptoms and emotional and psychological variables, multiple regression analyses were conducted. Anxiety was found to be a significant predictor of all types of schizotypal symptoms. In addition, reduced positive beliefs about self, increased fear of negative evaluation and increased interpersonal sensitivity were found to predict social anxiety schizotypal symptoms. Negative beliefs about self and others, and increased fear of negative evaluation were found to predict paranoid schizotypal symptoms.

Anxiety was the only predictor of anomalous experiences. This suggests that the mechanisms underlying anomalous experiences may be less influenced by psychological factors than those underlying social anxiety and paranoia. This fits with the ideas of the cognitive model of psychosis which argues that anomalous experiences may be caused by a basic cognitive dysfunction (Garety et al., 2001), and also fits with the findings of Study 2. Conversely, other symptom types are thought to be more psychologically constructed, arising from core schematic beliefs and emotional dysfunction (Fowler, Freeman, Smith et al., 2006; Freeman & Garety, 2003; Smith et al., 2006). That these findings occurred in a non-clinical sample supports the continuum hypothesis of psychosis.

8.2.4 Study 4

Study 4 examined the role of trauma history in the development and maintenance of schizotypal symptoms in a non-clinical sample. Thirty-five percent of the sample reported having experienced a traumatic event meeting DSM-IV-TR A1 stressor criteria (American Psychiatric Association, 2000a) at some point in their lives. Individuals with a history of interpersonal trauma (e.g. bullying, child sex abuse, physical victimisation) had significantly higher SSI scores (across all domains) than individuals who had not experienced an interpersonal trauma. These individuals also exhibited higher levels of anxiety, depression, and interpersonal sensitivity; and more negative beliefs about self and others. A history of non-interpersonal trauma (e.g. road traffic accidents, non-interpersonal violence, and other non-specific events) was not associated with higher levels of schizotypal symptoms or higher scores on any of the emotional and psychological variables. In order to clarify the potential role of trauma in psychosis, regression analyses were conducted. For all types of schizotypal symptoms, exposure to interpersonal trauma lost its predictive ability when controlling for emotion and psychological variables. This supports the notion of an emotional route to psychosis and suggests that trauma may influence the development of psychosis in an indirect way, i.e. via its impact on emotion and core schematic beliefs. These findings concur with literature investigating trauma and the acute symptoms of psychosis (Fowler, Freeman, Steel et al., 2006; Mueser, Rosenberg et al., 2002).

8.2.5 Study 5

The finding that schizotypal symptoms are common in individuals with psychosis, even when acute symptoms have remitted, poses an interesting question about whether such phenomena have an impact on long-term recovery from the disorder. In addition, the finding of differential associations between schizotypal symptom types and emotional, psychological, and cognitive variables; suggests that different types of schizotypal symptoms may impact upon recovery in different ways. An exploratory factor analysis was conducted in Study 5 to examine how different schizotypal symptom types were related to different recovery outcomes.

A six-factor model was found to provide the best fit to the data. The factors were labelled: Activity Levels, Positive Symptoms, Clinician-rated Recovery, Negative Symptoms, Resilience/Optimism, and Emotional Barriers. The Anomalous Experiences subscale of the SSI loaded onto the Positive Symptoms factor with tools such as the PANSS and SAPS, suggesting that this dimension of the SSI assesses core residual symptoms of psychosis. The Social Anxiety subscale of the SSI loaded on the Emotional Barriers factor with measures assessing anxiety, depression, hopelessness, negative beliefs about self and others, and illness cognitions. This suggests that Social Anxiety schizotypal symptoms may reflect emotional and psychological recovery from psychosis. The Paranoia subscale of the SSI loaded on both the Positive Symptoms and Emotional Barriers factors. This could be taken as evidence to suggest that paranoid schizotypal symptoms are reflective of both residual psychotic symptoms and emotional and psychological recovery from psychosis.

8.2.6 Study 6

The final study in this thesis examined the effect of a psychological intervention for social recovery on the presence and frequency of schizotypal symptoms, in the context of a randomised controlled trial. In addition, the role of schizotypal symptoms as mediators of recovery was investigated. Scores on the Social Anxiety subscale of the SSI were found to significantly increase in the group who received the intervention. Moreover, increases in social anxiety were found to be specifically related to increases in weekly hours of activity (an index of social recovery). Scores on the Paranoia and

Anomalous Experiences subscales of the SSI were not influenced by therapy. These findings indicate that social anxiety may be a mediator of social recovery from psychosis.

8.3 INTERPRETATION OF FINDINGS

It is well established that individuals in the prodromal stages of psychosis experience a range of low-level psychotic phenomena and social withdrawal (Addington & Addington, 2005; Hodges et al., 1989; Yung & McGorry, 1996). In terms of face validity, it could be argued that there are similarities between prodromal and schizotypal symptoms. That such phenomena predate the onset of acute psychosis, suggests that they may provide a foundation for further symptoms to be built upon. This thesis has also highlighted the presence of schizotypal symptoms in the recovery phase of acute psychosis. The prevalence of these symptoms is significantly higher than that occurring in a non-clinical sample. This could be taken as evidence to suggest that following an episode of psychosis, individuals inhabit a *postdromal* phase, returning to an almost at-risk mental state. This idea supports the "rollback phenomenon", a theory which suggests that symptoms of mental illness remit via a process which is the mirror image of their development (Detre & Jarecki, 1971; Fava, 1999). Indeed, studies examining the recovery process from acute psychosis describe a reduction in the frequency and severity of symptoms, combined with a gradual increase in insight and awareness (Andresen, Oades, & Caputi, 2003; Carr, 1983; Drury, 1992). This may be accompanied by anxiety and depression as the individual begins to come to terms with what has happened to them (Forchuk et al., 2003). This "recovering state" may be best assessed using a measure of schizotypal symptoms.

In line with the continuum hypothesis, the findings of this thesis also support studies examining the development and maintenance of acute symptoms of psychosis, and suggest specific underlying mechanisms for different types of schizotypal symptoms. Social anxiety is related to a lack of positive beliefs about self, i.e. low self-esteem; whereas paranoia is related to a feeling of personal vulnerability combined with beliefs that others are hostile. Anomalous experiences are relatively independent from psychological factors, apart from anxiety, suggesting that they may be organic in nature and relate to a basic cognitive dysfunction. The finding that anomalous experiences are

associated with cognitive disturbance and not with emotional and psychological variables; and that the converse is true for social anxiety, suggests separate and independent pathways in the development of different symptom types. Thus, there appears to be a biological route to anomalous experiences, and an emotional route to other symptom types. The development of paranoid schizotypal symptoms may be associated with both pathways. Indeed, scores on the Paranoia subscale of the SSI were found to be related to both psychological variables (negative beliefs about self and others) and ME strength (an index of anomalies in basic cognitive processes). These findings concur with the cognitive model of psychosis (Garety et al., 2001) and suggest that core symptoms of the disorder may arise from an anomaly in automatic processing, resulting in the occurrence of odd and unusual experiences (J. A. Gray et al., 1991). The interpretation of these experiences is hypothesised to be influenced by emotional state and core beliefs about self and others, resulting in the development of further psychotic symptoms (Garety et al., 2001).

The separate pathways to different schizotypal symptom types are likely to occur throughout the course of psychosis, continuing into the recovery stages. This is supported by the findings of the factor analysis in Study 5, suggesting that different types of schizotypal symptoms were associated with different types of recovery. Previous literature examining the recovery process suggests that the core positive symptoms of psychosis (e.g. anomalous experiences) are the first to remit (Agid, Kapur, Arenovich, & Zipursky, 2003; Kapur et al., 2005). This is argued to be due to the fact that their underlying physiology is the specific target of antipsychotic medication. Antipsychotic medication is hypothesised to dampen the aberrant salience underlying anomalous experiences by blocking dopamine receptors (Kapur, 2003; Kapur & Mamo, 2004; Seeman & Lee, 1975). However, other symptoms will take longer to remit as they require a certain amount of cognitive restructuring (Kapur, 2003; Rector & Beck, 2001). Similarly, the impact of an acute episode of psychosis on the way in which an individual perceives themselves and the world can be large and take time to deconstruct. Some of this restructuring and deconstruction is perhaps reflected in the high prevalence of schizotypal symptoms in individuals who would be defined as “recovered” using strict psychiatric criteria. Indeed, although there is no direct link between acute psychotic symptoms and functioning, the presence of low-level or schizotypal phenomena after acute symptoms have remitted may impede long-term functional

recovery. A model of recovery from psychosis incorporating schizotypal symptoms will be discussed in the next section.

8.4 COMBINING SCHIZOTYPAL SYMPTOMS AND PSYCHOSIS: TOWARDS A PSYCHOLOGICAL MODEL OF RECOVERY

This thesis has highlighted three types of schizotypal symptoms occurring after an episode of psychosis, and possibly premorbidly. Anxiety is a common predictor of all three symptom types. Core beliefs about self and others also predict schizotypal symptoms, but differentially, with a lack of positive beliefs about self predicting social anxiety, and the presence of negative beliefs about self and others predicting paranoia. Anomalous experiences are not predicted by schematic beliefs, suggesting that they are less psychologically constructed than other symptom types, potentially reflecting a core vulnerability to psychosis (e.g. basic cognitive dysfunction as in Hemsley, 2005b). Trauma exposure may impact upon the development of schizotypal symptoms indirectly, via increases in emotional distress and the synthesis of negative schematic beliefs about self and others. Together, these variables moderate levels of schizotypal symptoms which then act as differential mediators of recovery. See Figure 8.1 for a diagram of this model.

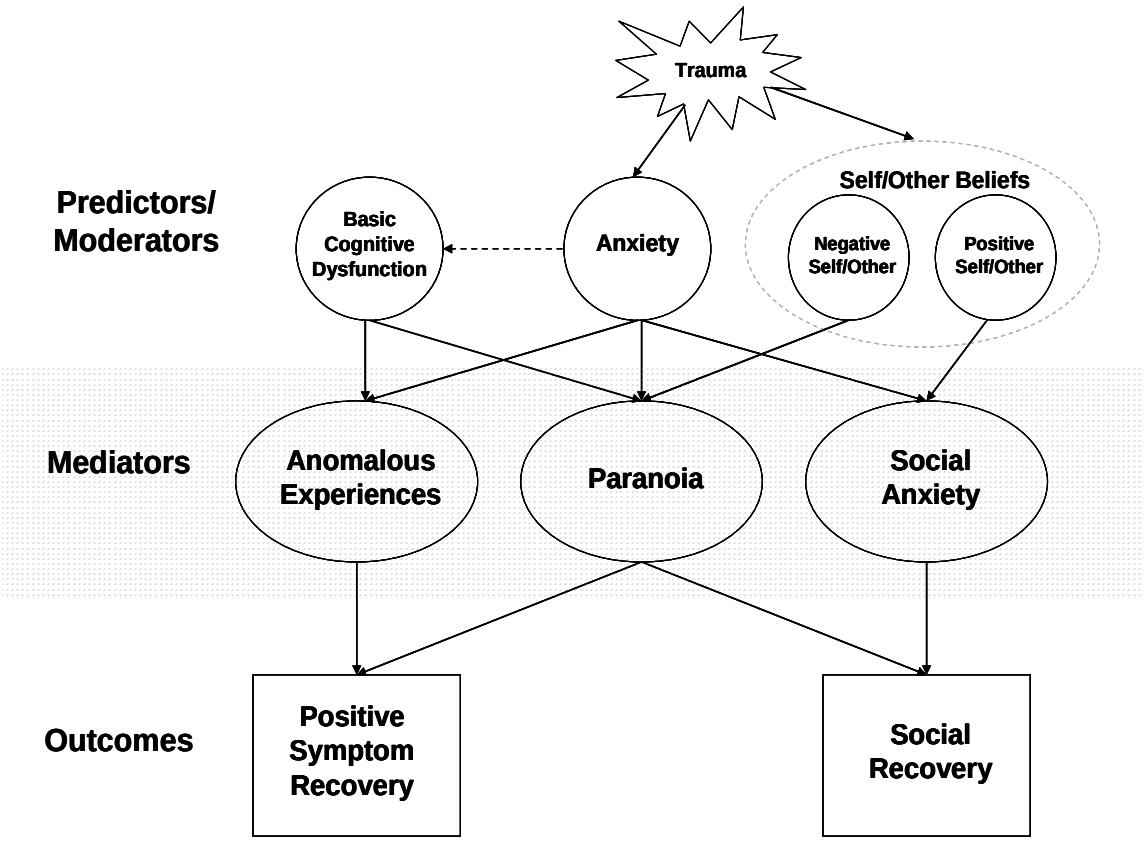


Figure 8.1

Psychological model of recovery from psychosis

Moderated by a basic cognitive dysfunction and anxiety, anomalous experiences are hypothesised to mediate positive symptom recovery from psychosis, potentially reflecting residual symptoms of the acute episode. Fluctuations in anomalous experiences may therefore be important in terms of monitoring the early warning signs of relapse. Moderated by schematic beliefs and anxiety, social anxiety schizotypal symptoms are hypothesised to mediate social recovery from psychosis. This is supported by the findings of Study 6. These symptoms may reflect underlying schematic beliefs about self (i.e. low self-esteem), but may also be related to how an individual has appraised, and in turn been personally affected by their psychotic episode. Paranoid schizotypal symptoms are moderated both by a basic cognitive dysfunction and schematic beliefs and anxiety. The notion that there are two routes into paranoia suggests that paranoid schizotypal symptoms may mediate both symptomatic and social recovery. This is supported by the finding that the Paranoia subscale of the

SSI loaded on both Positive Symptoms and Emotional Barriers dimensions of recovery, outlined in Study 5.

The above model incorporates the psychological effects of the psychotic episode on the development of schizotypal symptoms and thus recovery from the disorder. As outlined in previous chapters of this thesis, there is evidence to suggest that experiencing an episode of psychosis can be a traumatic life event and have profound effects on an individual's sense of self (McGorry et al., 1991; Shaner & Eth, 1989). Such emotional and psychological disturbance may feed into the development and maintenance of schizotypal symptoms, particularly social anxiety and paranoid subtypes, further impeding recovery. Thus, there may be a role for schizotypal symptoms as predictors of outcome.

Although the focus of this thesis is recovery from psychosis, the model outlined in Figure 8.1 may also be applicable to the development of acute psychosis, i.e. symptom development rather than symptom recovery; and social disability rather than social recovery. Thus, it is suggested that schizotypal phenomena are important at all stages throughout the course of psychosis. Anomalous experiences may reflect an individual's core biological vulnerability to psychosis. This is likely to be the result of a basic cognitive dysfunction, potentially exacerbated by state fluctuations in anxiety (J. A. Gray et al., 1991; Hemsley, 2005a). Conversely, social anxiety and paranoid schizotypal symptoms may reflect emotional and psychological factors underlying symptom development, possibly as a result of life events. An exacerbation of schizotypal symptoms may result in their escalation to an acute episode of psychosis.

Although preliminary and requiring further validation, this model may provide an explanation for the independence of symptomatic and functional recovery following an episode of psychosis (e.g. Davidson & McGlashan, 1997; M. Shepherd et al., 1989; Strauss & Carpenter, 1977). If an individual's route into psychosis occurs predominantly via the biological pathway (i.e. high core vulnerability), their recovery may be mostly symptomatic. As such, functional or social recovery may go unimpeded, other than the psychological and emotional impact of the episode itself. Conversely, if an individual's route into psychosis occurs via both biological and psychological pathways (i.e. due to a combination of core vulnerability and the presence of a traumatic

life event or severe negative beliefs about self and others), it is likely that emotional and psychological factors will also have an impact upon their recovery. In other words, if there are biological and emotional/psychological routes into psychosis, there may also be biological and emotional/psychological routes out of psychosis. Findings of previous research outlining poorer prognoses for individuals with psychosis and co-morbid trauma histories could be argued to provide support for this claim (Mueser, Rosenberg et al., 2002; Read & Ross, 2003). Even when acute symptoms are in remission, levels of schizotypal symptoms may be elevated in these individuals due to severe underlying negative schematic beliefs and high levels of emotional distress. However, further research would need to be conducted to substantiate this hypothesis.

8.5 CLINICAL IMPLICATIONS OF THE FINDINGS

The findings of this thesis support the use of a measure of schizotypal symptoms in assessing low-level psychotic phenomena at various points throughout the course of psychotic disorder. Traditionally, schizotypy measures have been used to identify individuals who may be at risk of making transition to psychosis based on personality traits, tendencies, and lifetime experiences. However, assessing the presence of state psychotic-like phenomena is arguably more clinically relevant as can be used to highlight an individual's current symptom profile, and thus current problem list, as opposed to general predispositions. Moreover, in non-clinical populations, state schizotypy may be a better predictor than trait schizotypy of an individual's risk of making transition to psychosis.

Results of the current work suggest that individuals in recovery from psychosis experience schizotypal symptoms over and above levels occurring in a non-clinical sample. In particular, social anxiety appears to be a large problem for individuals in recovery from psychosis, with over 50% of individuals reporting having experienced symptoms of social anxiety often or always in the past two weeks. This finding supports previous research reporting a high prevalence of social anxiety disorder following psychosis (e.g. Birchwood et al., 2006; Pallanti et al., 2004). Findings from this thesis have shown that social anxiety may also be a mediator of social recovery from psychosis. Thus, individuals recovering from psychosis may require specialised interventions to address their social anxiety. The SSI provides a useful tool for assessing

social anxiety specific to individuals in recovery from psychosis. Future research should examine the performance of the Social Anxiety subscale of the SSI in relation to traditional social anxiety assessment tools (e.g. Liebowitz Social Anxiety Scale; Liebowitz, 1987).

Schizotypy is traditionally conceptualised as a moderator of psychosis, i.e. a stable personality trait highlighting vulnerability to the development of a psychotic episode (Claridge, 1997b). This view suggests that schizotypy cannot be modified. An alternative view proposed by this thesis is of viewing schizotypal symptoms as mediators of outcome, moderated by a range of cognitive, emotional, and psychological variables. This view has considerable clinical implications and suggests that although trait schizotypy may be stable, state fluctuations of schizotypal symptoms may be modifiable and influence outcome. Indeed, Study 6 revealed changes in schizotypal symptoms following a recovery-focused intervention and in relation to increased activity. If different schizotypal symptom types are indeed mediators of different types of outcome, interventions targeting these symptoms (and the variables which moderate them) are likely to be successful in improving recovery. Further research using targeted interventions is warranted.

The findings of this thesis support literature arguing for a wider process than symptom remission in recovery from acute psychosis. In line with previous research, the data suggest that following an acute episode of psychosis, individuals experience a plethora of concerns which may contribute to poor recovery over and above the acute symptoms themselves (Anthony, 1993; Corrigan et al., 1999; McGorry, 1992; Mueser, Corrigan et al., 2002; Pitt et al., 2007). These concerns are likely to arise from an emotional and psychological response to the psychotic episode as a major life event and are distinct from an irreversible "deficit syndrome" or "burnout", previously described as responsible for poor functional recovery from psychosis (Gourevitch, Abbadi, & Guelfi, 2004; Tek, Kirkpatrick, & Buchanan, 2001). The notion that schizotypal symptoms, and thus recovery, may be associated with an individual's appraisal of their illness, suggests that interventions for recovery should focus on the emotional and psychological impact of an episode of psychosis; in addition to normalising and providing coping mechanisms for dealing with residual symptoms. This supports the work of Gumley and colleagues who propose a role for the meaning that an individual ascribes to their

experiences on the future course of psychosis (Gumley et al., 2003; Gumley et al., 1999). For example, a negative appraisal of the illness experience (e.g. "I can't cope") may be accompanied by the activation of underlying negative schematic beliefs and increased emotional distress. This may maintain schizotypal symptoms and further impede social recovery. Negative appraisals in response to low-level psychotic symptoms (e.g. "I am going mad") may also precipitate relapse (Gumley et al., 2003; Gumley et al., 1999; Lobban et al., 2003).

8.6 LIMITATIONS AND FUTURE RESEARCH

The limitations of specific studies conducted in this thesis have been outlined in the discussion sections of their respective chapters. As such they will not be repeated here. This section will discuss general limitations of the current work and highlight areas for future research.

All of the studies outlined in this thesis rely on the self-report of schizotypal symptoms. It has been argued that self-reports are unreliable in psychiatric populations and that individuals may find it difficult to subjectively rate their own experiences (Enns, Larsen, & Cox, 2000). However, evidence exists to suggest that this is not the case, even in individuals with poor insight (M. Bell, Fiszdon, Richardson, Lysaker, & Bryson, 2007; Eaton, Romanoski, Anthony, & Nestadt, 1991; Liraud, Droulout, Parrot, & Verdoux, 2004). Moreover, in the current work, scores on the SSI were shown to strongly correlate with researcher ratings on the PANSS. In addition, it has been argued that self-report measures enable researchers and clinicians to gain insight into the "inner world" of patients, and may also be less intimidating for the individual (Iancu, Poren, Lehman, Shamir, & Kotler, 2005). The use of self-report techniques is also more favourable when conducting investigations with large samples, due to time and resource constraints (Howitt & Cramer, 2005).

A major conclusion of this thesis is that schizotypal symptoms may be prodromal and postdromal features of psychosis, involved in both the development and maintenance of psychotic symptoms and in recovery from the disorder. However, it must be noted that due to the predominantly cross-sectional nature of this research, nothing is known about the symptoms experienced by participants prior to the onset of their psychosis, or during

the acute episode itself. As such, it is not possible to conclude with any certainty that symptoms measured by the SSI are residual in nature; or that they provide a mirror image of prodromal symptoms, although in terms of face validity this seems likely. In order to examine this assumption in more detail, longitudinal studies would need to be conducted using the SSI to observe fluctuations in schizotypal symptomatology over time. Content analysis could also be used to investigate whether the content of schizotypal symptoms occurring in recovery mirrors that of prodromal and acute psychotic phenomena.

Similarly, further research would need to be conducted in order to confirm the associations highlighted between schizotypal symptoms, cognitive, emotional, and psychological variables. Although the studies conducted in this thesis suggest differential associations for different symptom types; increases in ME strength, anxiety, and negative schema could arguably occur in response to schizotypal experiences themselves, rather than being involved in their development. Longitudinal research would enable these relationships to be modelled over time to see whether change in one variable (e.g. negative schema) has an effect on change in another (e.g. paranoid schizotypal symptoms). Moreover, experimental studies of mediation could be conducted to investigate the effect of manipulating explanatory variables on schizotypal symptoms in controlled conditions (i.e. if anxiety is increased, do scores on the SSI also increase?). It is likely that there are reciprocal relationships between schizotypal symptoms and emotional and psychological variables, as has been hypothesised to be the case for psychotic symptoms (Freeman & Garety, 2003). Thus, high levels of negative schematic beliefs and anxiety may precipitate symptom development, but the experience of symptoms is also likely to maintain negative beliefs and result in emotional distress. However, further research needs to be conducted to investigate this hypothesis.

Moreover, although this thesis highlights the presence of schizotypal symptoms in a non-clinical sample, it does not necessarily follow that they are causing distress or that they will develop into an episode of psychosis. Indeed, low-level psychotic phenomena are not uncommon in the general population and many people are able to function more than adequately whilst experiencing such symptoms (V. Bell et al., 2006). This is supported by the high level of schizotypal symptoms reported by the non-clinical

sample in Study 1. Thus, it is likely that there are factors other than high levels of schizotypal symptoms which are involved in the onset of psychosis. Further areas for future research would be to examine the predictive validity of the SSI; and to investigate relationships between SSI scores and other variables in individuals with at-risk mental states. For example, previous research has highlighted the importance of attachment style in the development of psychotic symptoms (Berry, Band, Corcoran, Barrowclough, & Wearden, 2007; Berry, Barrowclough, & Wearden, 2007; MacBeth, Schwannauer, & Gumley, 2008). Attachment style has also been implicated in the recovery process (Drayton et al., 1998; Gumley & Schwannauer, 2006; L. Tait, Birchwood, & Trower, 2004). As such, it would be interesting to investigate associations between different types of schizotypal symptoms and attachment and recovery styles. In terms of recovery, it would also be interesting to further examine the role of illness cognitions in the maintenance of schizotypal symptoms. Although the factor analysis in Study 5 highlighted that Social Anxiety and Paranoia subscales of the SSI loaded on the same recovery dimension as the PBIQ, further research would need to be conducted to confirm that an individual's appraisal of their illness was involved in the presence of schizotypal phenomena.

8.7 CONCLUSION

The work conducted in this thesis has begun to highlight possible relationships between schizotypal symptoms and the course of psychotic disorder. It has proposed that schizotypal symptoms may be at the core of psychosis, occurring both prior to onset, and in the recovery phase. Assessing state fluctuations in these phenomena may help to explain variations in outcome following an episode of psychosis, thus bridging the symptom-disability gap. The SSI has been shown to be a robust tool for use in measuring state schizotypal symptoms. Potential relationships have also been outlined between different types of schizotypal symptoms and explanatory variables. In addition to proposing differential pathways to the development of schizotypal symptoms, the current work has also proposed a preliminary psychological model of recovery incorporating schizotypal phenomena as mediators in the recovery process. Future research should focus on providing confirmatory evidence for this model using longitudinal and experimental methodologies.

REFERENCES

Addington, J., & Addington, D. (2005). Patterns of premorbid functioning in first episode psychosis: relationship to 2-year outcome. *Acta Psychiatrica Scandinavica, 112*, 40-46.

Addington, J., Addington, J., & Maticka-Tyndale, E. (1993). Assessing depression in schizophrenia: the Calgary Depression Scale. *British Journal of Psychiatry, 163*, 39-44.

Addington, J., Young, J., & Addington, D. (2003). Social outcome in early psychosis. *Psychological Medicine, 33*, 1119-1124.

Agid, O., Kapur, S., Arenovich, T., & Zipursky, R. B. (2003). Delayed-onset hypothesis of antipsychotic action: a hypothesis tested and rejected. *Archives of General Psychiatry, 60*, 1228-1235.

Allan, L. G., & Siegel, S. (1993). McCollough effects as conditioned responses: reply to Dodwell and Humphrey. *Psychological Review, 100*, 342-346; discussion 347-350.

Allardyce, J., Suppes, T., & van Os, J. (2007). Dimensions and the psychosis phenotype. *International Journal of Methods in Psychiatric Research, 16*(suppl.), S34-S40.

American Psychiatric Association. (1987). *Diagnostic and Statistical Manual of Mental Disorders, 3rd edition revised (DSM-III-R)*. Washington DC: American Psychiatric Association.

American Psychiatric Association. (1994). *Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV)*. Washington DC: American Psychiatric Association.

American Psychiatric Association. (2000a). *Diagnostic and Statistical Manual of Mental Disorders, 4th edition Text Revision (DSM-IV-TR)*. Washington DC: American Psychiatric Association.

American Psychiatric Association. (2000b). Global Assessment of Functioning. In *DSM-IV-TR* (pp. 32). Washington DC: American Psychiatric Association.

Andreasen, N. C. (1981). *The Scale for the Assessment of Negative Symptoms (SANS)*. Iowa City: University of Iowa.

Andreasen, N. C. (1984). *The Scale for the Assessment of Positive Symptoms (SAPS)*. Iowa City: University of Iowa.

Andreasen, N. C. (1990). Methods for assessing positive and negative symptoms. In N. C. Andreasen (Ed.), *Schizophrenia: Positive and Negative Symptoms and Syndromes. Modern Problems in Pharmacopsychiatry* (pp. 73-85). Basel: Karger.

Andreasen, N. C., Flaum, M., & Arndt, S. (1992). The Comprehensive Assessment of Symptoms and History (CASH). An instrument for assessing diagnosis and pathology. *Archives of General Psychiatry, 49*, 615-623.

Andresen, R., Oades, L., & Caputi, P. (2003). The experience of recovery from schizophrenia: towards an empirically validated stage model. *Australian and New Zealand Journal of Psychiatry, 37*, 586-594.

Andrews, D. P. (1964). Error-correcting perceptual mechanisms. *Quarterly Journal of Experimental Psychology, 16*, 104-115.

Anonymous. (1989). How I've managed chronic mental illness. *Schizophrenia Bulletin, 15*, 635-640.

Anthony, W. A. (1993). Recovery from mental illness: the guiding vision of the mental health service in the 1990s. *Psychosocial Rehabilitation Journal, 16*(4), 11-23.

Argyle, N. (1990). Panic attacks in chronic schizophrenia. *British Journal of Psychiatry, 157*, 430-433.

Armstrong, J. S. (1967). Derivation of theory by means of factor analysis or Tom Swift and his electric factor analysis machine. *The American Statistician, 21*, 17-21.

Arndt, S., Alliger, R. J., & Andreasen, N. C. (1991). The distinction of positive and negative symptoms: the failure of a two-dimensional model. *British Journal of Psychiatry, 158*, 317-322.

Arseneault, L., Cannon, M., Witton, J., & Murray, R. M. (2004). Causal association between cannabis and psychosis: examination of the evidence. *British Journal of Psychiatry, 184*, 110-117.

Asarnow, R. F., Nuechterlein, K. H., & Marder, S. R. (1983). Span of apprehension performance, neuropsychological functioning, and indices of psychosis proneness. *Journal of Nervous and Mental Disease, 171*, 662-669.

Avramopoulos, D. A., Stefanis, N. C., Hantoumi, I., Smyrnis, N., Evdokimidis, I. K., & Stefanis, C. N. (2002). Higher scores of self-reported schizotypy in healthy young males carrying the COMT high activity allele. *Molecular Psychiatry, 7*, 706-711.

Barak, S., & Weiner, I. (2007). Scopolamine induces disruption of latent inhibition which is prevented by antipsychotic drugs and an acetylcholinesterase inhibitor. *Neuropsychopharmacology, 32*, 989-999.

Barkus, E., Stirling, J., Hopkins, R., & Lewis, S. (2006). The presence of neurological soft signs along the psychosis proneness continuum. *Schizophrenia Bulletin, 32*, 573-577.

Barlow, H. (1990). Conditions for versatile learning, Helmholtz's unconscious interference, and the task of perception. *Vision Research, 30*, 1561-1571.

Barnes, G. W., Rhinewine, J. P., & Docherty, N. M. (2000). Perceptual aberration and schizotypy: a cautionary note. *Journal of Neuropsychiatry and Clinical Neurosciences, 12*, 98-99.

Barnes, J., Howard, R. J., Senior, C., Brammer, M., Bullmore, E. T., Simmons, A., et al. (1999). The functional anatomy of the McCollough contingent colour after-effect. *Neuroreport, 10*, 195-199.

Baron, M., Asnis, L., & Gruen, R. (1981). The Schedule for Schizotypal Personalities (SSP): a diagnostic interview for schizotypal features. *Psychiatry Research, 4*, 213-228.

Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research. Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology, 51*, 1173-1182.

Barrowclough, C., Tarrier, N., Humphreys, L., Ward, J., Gregg, L., & Andrews, B. (2003). Self-esteem in schizophrenia: relationships between self-evaluation, family attitudes, and symptomatology. *Journal of Abnormal Psychology, 112*, 92-99.

Barry, M., & Crosby, C. (1996). Quality of life as an evaluative measure in assessing the impact of community care on people with long-term psychiatric disorders. *British Journal of Psychiatry, 168*, 210-216.

Baruch, I., Hemsley, D. R., & Gray, J. A. (1988). Differential performance of acute and chronic schizophrenics in a latent inhibition task. *Journal of Nervous and Mental Disease, 176*, 598-606.

Basso, M. R., Nasrallah, H. A., Olson, S. C., & Bornstein, R. A. (1998). Neuropsychological correlates of negative, disorganised and psychotic symptoms in schizophrenia. *Schizophrenia Research, 31*, 99-111.

Bebbington, P. E., Brugha, D., Brugha, T., Singleton, N., Farrell, M., Jenkins, R., et al. (2004). Psychosis, victimisation and childhood disadvantage. Evidence from the Second British National Survey of Psychiatric Epidemiology. *British Journal of Psychiatry, 185*, 220-226.

Bebbington, P. E., Wilkins, S., Jones, P., Foerster, A., Murray, R., Toone, B., et al. (1993). Life events and psychosis. Initial results from the Camberwell Collaborative Psychosis Study. *British Journal of Psychiatry, 162*, 72-79.

Bechdolf, A., Ruhrmann, S., Wagner, M., Kuhn, K., Janssen, B., Bottlender, R., et al. (2005). Interventions in the initial prodromal states of psychosis in Germany: concept and recruitment. *British Journal of Psychiatry, 187*(suppl.), S45-S48.

Beck, A. T., Brown, G., Berchick, R. J., Stewart, B. L., & Steer, R. A. (1990). Relationship between hopelessness and ultimate suicide: a replication with psychiatric outpatients. *American Journal of Psychiatry, 147*, 190-195.

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive Therapy for Depression*. New York: Guilford Press.

Beck, A. T., & Steer, R. A. (1987). *Beck Anxiety Inventory*. San Antonio, TX: The Psychological Corporation.

Beck, A. T., & Steer, R. A. (1988). *Beck Hopelessness Scale Manual*. San Antonio, TX: The Psychological Corporation.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *BDI-II Manual*. San Antonio, TX: The Psychological Corporation.

Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry, 4*, 561-571.

Bedwell, J. S., & Donnelly, R. S. (2005). Schizotypal personality disorder or prodromal symptoms of schizophrenia? *Schizophrenia Research, 80*, 263-269.

Beecham, J., & Knapp, M. (1992). Costing psychiatric interventions. In G. Thornicroft, C. Brewin & J. Wing (Eds.), *Measuring Mental Health Needs* (pp. 163-183). London: Gaskell.

Bell, M., Fiszdon, J., Richardson, R., Lysaker, P., & Bryson, G. (2007). Are self-reports valid for schizophrenia patients with poor insight? Relationship of unawareness of illness to psychological self-report instruments. *Psychiatry Research, 151*, 37-46.

Bell, V., Halligan, P. W., & Ellis, H. D. (2006). The Cardiff Anomalous Perceptions Scale (CAPS): a new validated measure of anomalous perceptual experience. *Schizophrenia Bulletin, 32*, 366-377.

Bennett-Levy, J., Butler, G., Fennell, M., & Hackmann, A. (2004). *Oxford Guide to Behavioural Experiments in Cognitive Therapy*. Oxford: Oxford University Press.

Bentall, R. P. (1990). The illusion of reality: a review and integration of psychological research on hallucinations. *Psychological Bulletin, 107*, 82-95.

Bentall, R. P. (2004). *Madness Explained: Psychosis and Human Nature*. London: Penguin.

Bentall, R. P., Claridge, G. S., & Slade, P. D. (1989). The multidimensional nature of schizotypal traits: A factor analytic study with normal subjects. *British Journal of Clinical Psychology, 28*, 363-375.

Bentall, R. P., Corcoran, R., Howard, R., Blackwood, N., & Kinderman, P. (2001). Persecutory delusions: a review and theoretical integration. *Clinical Psychology Review, 21*, 1143-1192.

Bentall, R. P., Jackson, H. F., & Pilgrim, D. (1988). Abandoning the concept of 'schizophrenia': some implications of validity arguments for psychological research into psychotic phenomena. *British Journal of Clinical Psychology, 27*, 303-324.

Bentall, R. P., Kinderman, P., & Kaney, S. (1994). The self, attributional processes and abnormal beliefs: towards a model of persecutory delusions. *Behaviour Research and Therapy, 32*, 331-341.

Bentall, R. P., & Slade, P. D. (1985). Reality testing and auditory hallucinations: a signal detection analysis. *British Journal of Clinical Psychology, 24*, 159-169.

Bergman, A. J., Harvey, P. D., Mitropoulou, V., Aronson, A., Marder, D., Silverman, J., et al. (1996). The factor structure of schizotypal symptoms in a clinical population. *Schizophrenia Bulletin, 22*, 501-509.

Berridge, K. C., & Robinson, T. E. (1998). What is the role of dopamine in reward: hedonic impact, reward learning, or incentive salience? *Brain Research Reviews, 28*, 309-369.

Berry, K., Band, R., Corcoran, R., Barrowclough, C., & Wearden, A. (2007). Attachment styles, earlier interpersonal relationships and schizotypy in a non-clinical sample. *Psychology and Psychotherapy: Theory, Research and Practice, 80*, 563-576.

Berry, K., Barrowclough, C., & Wearden, A. (2007). A review of the role of adult attachment style in psychosis: unexplored issues and questions for further research. *Clinical Psychology Review, 27*, 458-475.

Best, S. J., Krueger, B., Hubbard, C., & Smith, A. (2001). An assessment of the generalisability of internet surveys. *Social Science Computer Review, 19*, 131-145.

Birchwood, M. (1995). Early intervention in psychotic relapse: cognitive approaches to detection and management. *Behaviour Change, 12*, 2-19.

Birchwood, M. (1996). Early Intervention in Psychosis. In G. Haddock & P. Slade (Eds.), *Cognitive-Behavioural Interventions with Psychotic Disorders*. London: Routledge.

Birchwood, M. (2003). Pathways to emotional dysfunction in first episode psychosis. *British Journal of Psychiatry, 182*, 373-375.

Birchwood, M., & Iqbal, Z. (1998). Depression and suicidal thinking in psychosis. In T. Wykes, N. Tarrier & S. Lewis (Eds.), *Outcome and Innovation in Psychological Treatment of Schizophrenia*. Chichester: Wiley.

Birchwood, M., Iqbal, Z., Chadwick, P., & Trower, P. (2000). Cognitive approach to depression and suicidal thinking in psychosis, I. Ontogeny of post-psychotic depression. *British Journal of Psychiatry, 177*, 516-521.

Birchwood, M., MacMillan, F., & Smith, J. (1992). Early intervention. In M. Birchwood & N. Tarrier (Eds.), *Innovations in the Psychological Management of Schizophrenia*. Chichester: Wiley.

Birchwood, M., Mason, R., MacMillan, F., & Healy, J. (1993). Depression, demoralization and control over psychotic illness: a comparison of depressed

and non-depressed patients with a chronic psychosis. *Psychological Medicine, 23*, 387-395.

Birchwood, M., Smith, J., Cochrane, R., Wetton, S., & Copestake, S. (1990). The Social Functioning Scale: the development and validation of a new scale of social adjustment for use in family intervention programmes with schizophrenic patients. *British Journal of Psychiatry, 157*, 853-859.

Birchwood, M., Smith, J., MacMillan, F., Hogg, B., Prasad, R., Harvey, C., et al. (1989). Predicting relapse in schizophrenia: the development and implementation of an early signs monitoring system using patients and families as observers. *Psychological Medicine, 19*, 649-656.

Birchwood, M., Spencer, E., & McGovern, D. (2000). Schizophrenia: early warning signs. *Advances in Psychiatric Treatment, 6*, 93-101.

Birchwood, M., Trower, P., Brunet, K., Gilbert, P., Iqbal, Z., & Jackson, C. (2006). Social anxiety and the shame of psychosis: a study in first episode psychosis. *Behaviour Research and Therapy, 45*, 1025-1037.

Birnbaum, M. H. (2001). *Introduction to Behavioural Research on the Internet*. Englewood Cliffs, NJ: Prentice-Hall.

Blanchard, J. J., Mueser, K. T., & Bellack, A. S. (1998). Anhedonia, positive and negative affect, and social functioning in schizophrenia. *Schizophrenia Bulletin, 24*, 413-424.

Bland, J., & Altman, D. G. (1994). Regression towards the mean. *British Medical Journal, 308*, 1499.

Bleuler, E. (1908). Die Prognose der Dementia Praecox - Schizophreniegruppe. *Allgemeine Zeitschrift fur Psychiatrie, 65*, 436-464.

Bleuler, E. (1950). *Dementia praecox or the group of schizophenias* (J. Zinkin, Trans.). New York: International Universities Press.

Bowins, B., & Shugar, G. (1998). Delusions and self-esteem. *Canadian Journal of Psychiatry, 43*, 154-158.

Boyce, P., & Parker, G. (1989). Development of a scale to measure interpersonal sensitivity. *Australian and New Zealand Journal of Psychiatry, 23*, 341-351.

Boydell, J., van Os, J., McKenzie, K., Allardyce, J., Goel, R., McCreadie, R. G., et al. (2001). Incidence of schizophrenia in ethnic minorities in London: ecological study into interactions with environment. *British Medical Journal, 323*, 1336-1338.

Boydell, J., van Os, J., McKenzie, K., & Murray, R. M. (2004). The association of inequality with the incidence of schizophrenia - an ecological study. *Social Psychiatry and Psychiatric Epidemiology, 39*, 597-599.

Brenner, H. D., & Pfammatter, M. (2000). Psychological therapy in schizophrenia: what is the evidence? *Acta Psychiatrica Scandinavica, 102*(suppl.), S74-S77.

Brewin, C. R., & Holmes, E. A. (2003). Psychological theories of posttraumatic stress disorder. *Clinical Psychology Review, 23*, 339-376.

Brockington, I. (1992). Schizophrenia: yesterday's concept. *European Psychiatry, 7*, 203-207.

Brooks, R. (1996). EuroQol: the current state of play. *Health Policy, 37*, 53-72.

Brown, T. A. (2006). *Confirmatory Factor Analysis for Applied Research*. New York: Guilford Press.

Browne, A., & Finkelhor, D. (1986). Impact of child sexual abuse: a review of the research. *Psychological Bulletin, 99*, 66-77.

Buchanan, R. W., Ball, P., Weiner, E., Kirkpatrick, B., Gold, J. M., McMahon, R. P., et al. (2005). Olanzapine treatment of residual positive and negative symptoms. *American Journal of Psychiatry, 162*, 124-129.

Bunney, W. E., Hetrick, W. P., Bunney, B. G., Patterson, J. V., Jin, Y., Potkin, S. G., et al. (1999). Structured Interview for Assessing Perceptual Anomalies (SIAPA). *Schizophrenia Bulletin, 25*, 577-592.

Burns, T., Catty, J., Becker, T., Drake, R. E., Fioritti, A., Knapp, M., et al. (2007). The effectiveness of supported employment for people with severe mental illness: a randomised controlled trial. *Lancet, 370*, 1146-1152.

Bustamante, S., Maurer, K., Löffler, W., & Häfner, H. (1994). Depression in the early course of schizophrenia. *Fortschritte der Neurologie-Psychiatrie, 62*, 317-329.

Butler, G. (1999). *Overcoming Social Anxiety: A Self-Help Guide using Cognitive-Behavioural Techniques*. London: Constable and Robinson.

Butler, R. W., Mueser, K. T., Sprock, J., & Braff, D. L. (1996). Positive symptoms of psychosis in posttraumatic stress disorder. *Biological Psychiatry, 39*, 839-844.

Byrne, M., Hodges, A., Grant, E., Owens, D. C., & Johnstone, E. C. (1999). Neuropsychological assessment of young people at high genetic risk for developing schizophrenia compared with controls: Preliminary findings from the Edinburgh High Risk Study (EHRS). *Psychological Medicine, 29*, 1161-1173.

Byth, W., Logue, N. A., Bell, P., Best, S. J., & King, D. J. (1992). The McCollough Effect as a measure of central cholinergic activity in man. *Psychopharmacology, 106*, 75-84.

Byth, W., McMahon, D., & King, D. J. (2000). Cholinergic agents and the McCollough effect. *Perception, 29*, 461-480.

Candido, C. L., & Romney, D. M. (1990). Attributional style in paranoid vs. depressed patients. *British Journal of Medical Psychology, 63*, 355-363.

Cannon, M., Caspi, A., Moffitt, T. E., Harrington, H., Taylor, A., Murray, R. M., et al. (2002). Evidence for early-childhood, pan-developmental impairment specific to

schizophreniform disorder: results from a longitudinal birth cohort. *Archives of General Psychiatry, 59*, 449-456.

Cantor-Graae, E., & Selten, J. (2005). Schizophrenia and migration: a meta-analysis and review. *American Journal of Psychiatry, 162*, 12-24.

Carlsson, A., & Lindquist, M. (1963). Effect of chlorpromazine or haloperidol on the formation of 3-methoxytyramine and normetanephrine in mouse brain. *Annual Review of Pharmacology and Toxicology, 20*, 140-144.

Carmen, E. H., Rieker, P. P., & Mills, T. (1984). Victims of violence and psychiatric illness. *American Journal of Psychiatry, 141*, 378-383.

Carr, V. J. (1983). Recovery from schizophrenia: a review of patterns of psychosis. *Schizophrenia Bulletin, 9*, 95-121.

Cassano, G. B., Pini, S., Saettoni, M., Rucci, P., & Dell'Oso, L. (1998). Occurrence and clinical correlates of psychiatric co-morbidity in patients with psychotic disorders. *Journal of Clinical Psychiatry, 59*, 60-68.

Cattell, R. B. (1966). The scree test for the number of factors. *Multivariate Behavioral Research, 1*, 245-276.

Cattell, R. B., Eber, H. W., & Tatsuoka, M. M. (1970). *Handbook for the Sixteen Personality Factor Questionnaire*. Champaign, IL: Institute for Personality and Ability Testing.

Chadwick, P. (1997). Recovery from psychosis: learning more from patients. *Journal of Mental Health, 6*, 577-588.

Chadwick, P., & Birchwood, M. (1994). The omnipotence of voices. A cognitive approach to auditory hallucinations. *British Journal of Psychiatry, 164*, 190-201.

Chapman, J. P. (1966). The early symptoms of schizophrenia. *British Journal of Psychiatry, 112*, 225-251.

Chapman, J. P., Chapman, L. J., & Kwapil, T. R. (1994). Does the Eysenck Psychoticism Scale predict psychosis? A ten year longitudinal study. *Journal of Personality and Social Psychology, 17*, 369-375.

Chapman, J. P., Chapman, L. J., & Kwapil, T. R. (1995). Scales for the measurement of schizotypy. In A. Raine, T. Lencz & S. A. Mednick (Eds.), *Schizotypal Personality* (pp. 79-106). Cambridge: Cambridge University Press.

Chapman, L. J., & Chapman, J. P. (1973). Problems in the measurement of cognitive deficit. *Psychological Bulletin, 79*, 380-385.

Chapman, L. J., & Chapman, J. P. (1980). Scales for rating psychotic and psychotic-like experiences as continua. *Schizophrenia Bulletin, 6*, 476-489.

Chapman, L. J., Chapman, J. P., Kwapil, T. R., Eckblad, M., & Zinser, M. C. (1994). Putatively psychosis-prone subjects 10 years later. *Journal of Abnormal Psychology, 103*, 171-183.

Chapman, L. J., Chapman, J. P., & Miller, E. N. (1982). Reliabilities and intercorrelations of eight measures of proneness to psychosis. *Journal of Consulting and Clinical Psychology, 50*, 187-195.

Chapman, L. J., Chapman, J. P., Numbers, J. S., Edell, W. S., Carpenter, B. N., & Beckfield, D. (1984). Impulsive non-conformity as a trait contributing to the prediction of psychotic-like and schizophrenic symptoms. *Journal of Nervous and Mental Disease, 172*, 681-691.

Chapman, L. J., Chapman, J. P., & Raulin, M. L. (1976). Scales for physical and social anhedonia. *Journal of Abnormal Psychology, 85*, 374-382.

Chapman, L. J., Chapman, J. P., & Raulin, M. L. (1978). Body image aberration in schizophrenia. *Journal of Abnormal Psychology, 87*, 399-407.

Chequers, J., Joseph, S., & Diduca, D. (1997). Belief in extraterrestrial life, UFO-related beliefs, and schizotypal personality. *Personality and Individual Differences, 23*, 519-521.

Chua, S. E., & McKenna, P. J. (1995). Schizophrenia - a brain disease? A critical review of structural and functional cerebral abnormality in the disorder. *British Journal of Psychiatry, 166*, 563-582.

Chubb, C., Sperling, G., & Solomon, J. A. (1989). Texture interactions determine perceived contrast. *Proceedings of the National Academy of Sciences of the United States of America, 86*, 9631-9635.

Claridge, G. (1983). The Eysenck Psychoticism Scale. In J. N. Butcher & C. D. Spielberger (Eds.), *Advances in Personality Assessment* (Vol. 2). Hillsdale: Lawrence Erlbaum.

Claridge, G. (1997a). Theoretical background and issues. In G. Claridge (Ed.), *Schizotypy: Implications for Illness and Health* (pp. 3-19). Oxford: Oxford University Press.

Claridge, G. (Ed.). (1997b). *Schizotypy: Implications for Illness and Health*. Oxford: Oxford University Press.

Claridge, G., & Beech, T. (1995). Fully and quasi-dimensional constructions of schizotypy. In A. Raine, T. Lencz & S. A. Mednick (Eds.), *Schizotypal Personality*. Cambridge: Cambridge University Press.

Claridge, G., & Broks, P. (1984). Schizotypy and hemisphere function: I. Theoretical considerations and the measurement of schizotypy. *Personality and Individual Differences, 5*, 633-648.

Claridge, G., McCreery, C., Mason, O., Bentall, R., Boyle, G., Slade, P., et al. (1996). The factor structure of 'schizotypal' traits: a large replication study. *British Journal of Clinical Psychology, 35*, 103-115.

Clark, D. M., & Fairburn, C. G. (1997). *Science and Practice of Cognitive Behaviour Therapy*. Oxford: Oxford University Press.

Clark, D. M., & Wells, A. (1995). A cognitive model of social phobia. In R. G. Heimberg, M. R. Liebowitz, D. A. Hope & F. R. Schneier (Eds.), *Social Phobia: Diagnosis, Assessment and Treatment* (pp. 41-68). New York: Guilford Press.

Clarke, D. (1991). Belief in the paranormal: a New Zealand study. *Journal of the Society for Psychical Research, 57*, 412-425.

Cohen, J. (1988). *Statistical Power Analysis for the Behavioural Sciences*. Hillsdale, NJ: Erlbaum Associates.

Cohen, J., Cohen, P., West, S. G., & Aiken, L. S. (2003). *Applied Multiple Regression/Correlation Analysis for the Behavioural Sciences* (3rd ed.). Mahwah, NJ: Lawrence Earlbaum Associates.

Coldham, E. L., Addington, J., & Addington, D. (2002). Medication adherence of individuals with a first episode of psychosis. *Acta Psychiatrica Scandinavica, 106*, 286-290.

Cole, J. O., Klerman, C. L., & Goldberg, S. C. (1969). Phenothiazine treatment of acute schizophrenia. *Archives of General Psychiatry, 10*, 246-261.

Combs, D. R., & Penn, D. L. (2004). The role of subclinical paranoia on social perception and behaviour. *Schizophrenia Research, 69*, 93-104.

Corlett, P. R., Honey, G. D., & Fletcher, P. C. (2007). From prediction error to psychosis: ketamine as a pharmacological model of delusions. *Journal of Psychopharmacology, 21*, 235-252.

Corrigan, P. W., Giffort, D., Rashid, F., Leary, M., & Okeke, I. (1999). Recovery as a psychological construct. *Community Mental Health Journal, 35*, 231-239.

Cosoff, S. J., & Hafner, R. J. (1998). The prevalence of comorbid anxiety in schizophrenia, schizoaffective disorder and bipolar disorder. *Australian and New Zealand Journal of Psychiatry, 32*, 67-72.

Costello, A. B., & Osborne, J. W. (2005). Best practices in exploratory factor analysis: four recommendations for getting the most from your analysis. *Practical Assessment, Research and Evaluation, 10*, Available online at: http://pareonline.net/getvn.asp?v=10&n=17.

Cosway, R., Byrne, M., Clafferty, R., Hodges, A., Grant, E., Abukmeil, S. S., et al. (2000). Neuropsychological change in young people at high risk for schizophrenia: results from the first two neuropsychological assessments of the Edinburgh High Risk Study. *Psychological Medicine, 30*, 1111-1121.

Cougnard, A., Marcelis, M., Myin-Germeys, I., de Graaf, R., Vollebergh, W., Krabbendam, L., et al. (2007). Does normal developmental expression of psychosis combine with environmental risk to cause persistence of psychosis? *Psychological Medicine, 37*, 513-527.

Couper, M. P., Blair, J., & Triplett, T. (1999). A comparison of mail and email for a survey of employees in federal statistics agencies. *Journal of Official Statistics, 15*, 39-56.

Cramer, J. A., Rosenheck, R., Xu, W., Thomas, J., Henderson, W., & Charney, D. S. (2000). Quality of life in schizophrenia: a comparison of instruments. Department of Veterans Affairs cooperative study group on clozapine in refactory schizophrenia. *Schizophrenia Bulletin, 26*, 659-666.

Crawford, J. R., & Henry, J. D. (2003). The Depression Anxiety Stress Scales (DASS): normative data and latent structure in a large non-clinical sample. *British Journal of Clinical Psychology, 42*, 111-131.

Crow, T. J. (1980). Molecular pathology of schizophrenia: more than one disease process? *British Medical Journal, 280*, 66-68.

Cummings, J. L. (1988). Organic psychosis. *Psychosomatics, 29*, 16-26.

Curran, P. J., West, S. G., & Finch, J. F. (1996). The robustness of test statistics to nonnormality and specification error in confirmatory factor analysis. *Psychological Methods, 1*, 16-29.

Cutting, J. (1985). *The Psychology of Schizophrenia*. Edinburgh: Churchill-Livingstone.

Cutting, J. (2003). Descriptive psychopathology. In S. R. Hirsch & D. R. Weinberger (Eds.), *Schizophrenia*. Oxford: Blackwell Science.

Dakin, S., Carlin, P., & Hemsley, D. (2005). Weak suppression of visual context in chronic schizophrenia. *Current Biology, 15*, 822-824.

Data Protection Act. (1998). London: Stationery Office.

Davidson, L. (2003). *Living Outside Mental Illness: Qualitative Studies of Recovery in Schizophrenia*. New York: New York University Press.

Davidson, L., & McGlashan, T. H. (1997). The varied outcomes of schizophrenia. *Canadian Journal of Psychiatry, 42*, 34-43.

Davis, K. L., Kahn, R. S., Ko, G., & Davidson, M. (1991). Dopamine in schizophrenia: a review and reconceptualization. *American Journal of Psychiatry, 148*, 1474-1486.

De Jong, P. J. (2002). Implicit self-esteem and social anxiety: differential self-favouring effects in high and low anxious individuals. *Behaviour Research and Therapy, 40*, 501-508.

Deegan, P. (1988). Recovery: the lived experience of rehabilitation. *Psychosocial Rehabilitation Journal, 11*(4), 11-19.

Deegan, P. (1997). Recovery and empowerment for people with psychiatric disabilities. *Social Work in Health Care, 25*(3), 11-24.

Delis, D. C., Jacobson, M., Bondi, M. W., Hamilton, J. M., & Salmon, D. P. (2003). The myth of testing construct validity using factor analysis or correlations with normal or mixed clinical populations: lessons from memory assessment. *Journal of the International Neuropsychological Society, 9*, 936-946.

Department of Health. (2001). *The Journey to Recovery - the Government's Vision for Mental Health Care*. London: Department of Health.

Detre, T. P., & Jarecki, H. (1971). *Modern Psychiatric Treatment*. Philadelphia: Lippincott.

Dinn, W. M., Harris, C. L., Aycicegi, A., Greene, P., & Andover, M. S. (2002). Positive and negative schizotypy in a student sample: neurocognitive and clinical correlates. *Schizophrenia Research, 56*, 171-185.

Dodwell, P. C., & Humphrey, G. K. (1990). A functional theory of the McCollough effect. *Psychological Review, 97*, 78-89.

Dodwell, P. C., & Humphrey, G. K. (1993). What is important about McCollough effects? A reply to Allan and Siegel. *Psychological Review, 100*, 347-350.

Dohrewend, B. P., Levav, I., Shrout, P. E., Schwartz, S., Naveh, G., Link, B. G., et al. (1992). Socioeconomic status and psychiatric disorders: the causation-selection issue. *Science, 225*, 946-951.

Dorman, P. J., Waddell, F., Slattery, J., Dennis, M., & Sandercock, P. (1997). Is the EuroQol a valid measure of health-related quality of life after stroke? *Stroke, 28*, 1876-1882.

Drayton, M., Birchwood, M., & Trower, P. (1998). Early attachment experience and recovery from psychosis. *British Journal of Clinical Psychology, 37*, 269-284.

Drury, V. (1992). Monitoring recovery from acute psychosis. In M. Birchwood & N. Tarrier (Eds.), *Innovations in the Psychological Management of Schizophrenia*. Chichester: John Wiley & Sons Ltd.

Dunn, G., & Bentall, R. (2007). Modelling treatment-effect heterogeneity in randomised controlled trials of complex interventions (psychological treatments). *Statistics in Medicine, 26*, 4719-4745.

Eaton, W. W., Romanoski, A., Anthony, J. C., & Nestadt, G. (1991). Screening for psychosis in the general population with a self-report interview. *Journal of Nervous and Mental Disease, 179*, 689-693.

Eckblad, M., & Chapman, L. J. (1983). Magical ideation as an indicator of schizotypy. *Journal of Consulting and Clinical Psychology, 51*, 215-225.

Eckblad, M., & Chapman, L. J. (1986). Development and validation of a new scale for hypomanic personality. *Journal of Abnormal Psychology, 95*, 214-222.

Eckblad, M., Chapman, L. J., Chapman, J. P., & Mishlove, M. (1982). *The Revised Social Anhedonia Scale*. University of Wisconsin - Madison: Unpublished Test.

Ehlers, A., & Clark, D. M. (2000). A cognitive model of posttraumatic stress disorder. *Behaviour Research and Therapy, 38*, 319-345.

Ellenbroek, B. A., & Cools, A. R. (2000). Animal models for the negative symptoms of schizophrenia. *Behavioural Pharmacology, 11*, 223-233.

Endicott, J., & Spitzer, R. L. (1978). A diagnostic interview: The Schedule for Affective Disorders and Schizophrenia. *Archives of General Psychiatry, 35*, 837.

Endicott, J., Spitzer, R. L., Fleiss, J. L., & Cohen, J. (1976). The global assessment scale. A procedure for measuring overall severity of psychiatric disturbance. *Archives of General Psychiatry, 33*, 766-771.

Enns, M., Inayatulla, M., Cox, B., & Cheyne, L. (1997). Prediction of suicide intent in Aboriginal and non-Aboriginal adolescent inpatients: a research note. *Suicide and Life Threatening Behavior, 27*, 218-224.

Enns, M., Larsen, D. K., & Cox, B. J. (2000). Discrepancies between self and observer ratings of depression. The relationship to demographic, clinical and personality variables. *Journal of Affective Disorders, 60*, 33-41.

Erickson, D. H., Beiser, M., & Iacono, W. G. (1999). Social support predicts 5-year outcome in first-episode schizophrenia. *Journal of Abnormal Psychology, 107*, 681-685.

Estroff, S. E. (1989). Self, identity, and subjective experiences of schizophrenia: in search of the subject. *Schizophrenia Bulletin, 15*, 189-196.

Estroff, S. E. (1995). Brokenhearted lifetimes: ethnography, subjectivity, and psychosocial rehabilitation. *International Journal of Mental Health, 24*(1), 82-92.

EuroQol Group. (1996). *EQ-5D User Guide: A measure of health-related quality of life developed by the EuroQol Group*. Rotterdam, Netherlands: EuroQol Group.

Eysenck, H. J., & Eysenck, S. G. B. (1975). *Manual of the Eysenck Personality Questionnaire*. London: Hodder and Stoughton.

Eysenck, H. J., & Eysenck, S. G. B. (1976). *Psychoticism as a Dimension of Personality*. London: Hodder and Stoughton.

Eysenck, S. B., Eysenck, H. J., & Barrett, P. (1985). A revised version of the Psychoticism scale. *Personality and Individual Differences, 6*, 21-29.

Fabrigar, L. R., Wegener, D. T., MacCallum, R. C., & Strahan, E. J. (1999). Evaluating the use of exploratory factor analysis in psychological research. *Psychological Methods, 4*, 272-299.

Farrell, M. (1992). Personality and anti-social behaviour among emotionally/behaviourally disturbed boys. *Personality and Individual Differences, 13*, 511-517.

Fava, G. A. (1999). Sub-clinical symptoms in mood disorders: pathophysiological and therapeutic implications. *Psychological Medicine, 29*, 47-61.

Feelgood, S. R., & Rantzen, A. J. (1994). Auditory and visual hallucinations in university students. *Personality and Individual Differences, 17*, 293-296.

Fenigstein, A., & Vanable, P. A. (1992). Paranoia and self-consciousness. *Journal of Personality and Social Psychology, 62*, 129-138.

Fialko, L., Freeman, D., Bebbington, P. E., Kuipers, E., Garety, P. A., Dunn, G., et al. (2006). Understanding suicidal ideation in psychosis: findings from the Psychological Prevention of Relapse in Psychosis (PRP) trial. *Acta Psychiatrica Scandinavica, 114*, 177-186.

Figueroa, E. F., Silk, K. R., Huth, A., & Lohr, N. E. (1997). History of childhood sexual abuse and general psychopathology. *Comprehensive Psychiatry, 38*, 23-30.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1996). *Structured Clinical Interview for DSM-IV Axis I Disorders, Clinical Version (SCID-CV)*. Washington D. C.: American Psychiatric Press, Inc.

Foa, E. B., Ehlers, A., Clark, D. M., Tolin, D. F., & Orsillo, E. M. (1999). The Posttraumatic Cognitions Inventory (PTCI): development and validation. *Psychological Assessment, 11*, 303-314.

Forchuk, C., Jewell, J., Tweedell, D., & Steinnagel, L. (2003). Reconnecting: the client experience of recovery from psychosis. *Perspectives in Psychiatric Care, 39*, 141-150.

Fowler, D. (2000a). Cognitive behaviour therapy for psychosis: from understanding to treatment. *Psychiatric Rehabilitation Skills, 4*, 199-215.

Fowler, D. (2000b). Psychological Formulation of Early Episodes of Psychosis: A Cognitive Model. In M. Birchwood, D. Fowler & C. Jackson (Eds.), *Early Intervention in Psychosis: A Guide to Concepts, Evidence and Interactions*. Chichester: John Wiley & Sons.

Fowler, D. (2002). Psychological Formulation of Early Episodes of Psychosis: A Continuum Model. In M. Birchwood, D. Fowler & C. Jackson (Eds.), *Early Intervention in Psychosis: A Guide to Concepts, Evidence and Interactions*. Chichester: Wiley.

Fowler, D., Freeman, D., Smith, B., Kuipers, E., Bebbington, P., Bashforth, H., et al. (2006). The Brief Core Schema Scales (BCSS): psychometric properties and associations with paranoia and grandiosity in non-clinical and psychosis samples. *Psychological Medicine, 36*, 1-11.

Fowler, D., Freeman, D., Steel, C., Hardy, A., Smith, B., Hackmann, C., et al. (2006). The catastrophic interaction hypothesis: how do stress, trauma, emotion, and information processing abnormalities lead to psychosis? In W. Larkin & A. P. Morrison (Eds.), *Trauma and Psychosis: New Directions for Theory and Therapy*: John Wiley and Sons.

Fowler, D., Garety, P., & Kuipers, E. (1998). Cognitive therapy for psychosis: formulation, treatment effects and service implications. *Journal of Mental Health, 7*, 123-133.

Fowler, D., Garety, P. A., & Kuipers, E. (1995). *Cognitive Behaviour Therapy for Psychosis: Theory and Practice*. Chichester: Wiley.

Freeman, D. (2007). Suspicious minds: the psychology of persecutory delusions. *Clinical Psychology Review, 27*, 425-457.

Freeman, D., Dunn, G., Garety, P. A., Bebbington, P., Slater, M., Kuipers, E., et al. (2005). The psychology of persecutory ideation I: a questionnaire survey. *Journal of Nervous and Mental Disease, 193*, 302-308.

Freeman, D., Garety, P., Fowler, D., Kuipers, E., Dunn, G., Bebbington, P., et al. (1998). The London-East Anglia randomized controlled trial of cognitive-behaviour therapy for psychosis. IV: Self-esteem and persecutory delusions. *British Journal of Clinical Psychology, 37*, 415-430.

Freeman, D., Garety, P., Kuipers, E., Fowler, D., & Bebbington, P. (2002). A cognitive model of persecutory delusions. *British Journal of Clinical Psychology, 41*, 331-347.

Freeman, D., & Garety, P. A. (1999). Worry, worry processes and dimensions of delusions: an exploratory investigation of a role for anxiety processes in the maintenance of delusional distress. *Behavioural and Cognitive Psychotherapy, 27*, 47-52.

Freeman, D., & Garety, P. A. (2003). Connecting neurosis and psychosis: the direct influence of emotion on delusions and hallucinations. *Behaviour Research and Therapy, 41*, 923-947.

Freeman, D., Garety, P. A., Bebbington, P. E., Smith, B., Rollinson, R., Fowler, D., et al. (2005). Psychological investigation of the structure of paranoia in a non-clinical population. *British Journal of Psychiatry, 186*, 427-435.

Freeman, D., Gittins, M., Pugh, K., Antley, A., Slater, M., & Dunn, G. (2008). What makes one person paranoid and another person anxious? The differential prediction of social anxiety and persecutory ideation in an experimental situation. *Psychological Medicine, 38*, 1121-1132.

Fricker, S., Galesic, M., Tourangeau, R., & Yan, T. (2005). An experimental comparison of web and telephone surveys. *Public Opinion Quarterly, 69*, 370-392.

Frith, C. D. (1979). Consciousness, information processing and schizophrenia. *British Journal of Psychiatry, 134*, 225-235.

Frith, C. D. (1992). *The Cognitive Neuropsychology of Schizophrenia*. Hove: Lawrence-Erlbaum.

Ganev, K. (2000). Long-term trends of symptoms and disability in schizophrenia and related disorders. *Social Psychiatry and Psychiatric Epidemiology, 35*, 389-395.

Garety, P. A., Fowler, D., & Kuipers, E. (2000). Cognitive-behavioural therapy for medication resistant symptoms. *Schizophrenia Bulletin, 26*, 73-76.

Garety, P. A., & Hemsley, D. (1994). *Delusions: Investigations into the Psychology of Delusional Reasoning*. Maudsley Monograph, vol. 36: Psychology Press.

Garety, P. A., Kuipers, E., Fowler, D., Freeman, D., & Bebbington, P. E. (2001). A cognitive model of the positive symptoms of psychosis. *Psychological Medicine, 31*, 189-195.

Giersch, A., & Rhein, V. (2008). Lack of flexibility in visual grouping in patients with schizophrenia. *Journal of Abnormal Psychology, 117*, 132-142.

Gilbert, P., Boxall, M., Cheung, M., & Irons, C. (2005). The relation of paranoid ideation and social anxiety in a mixed clinical population. *Clinical Psychology and Psychotherapy, 12*, 124-133.

Gitlin, M., Nuechterlein, K., Subotnik, K. L., Ventura, J., Mintz, J., Fogelson, D. L., et al. (2001). Clinical outcome following neuroleptic discontinuation in patients with remitted recent-onset schizophrenia. *American Journal of Psychiatry, 158*, 1835-1842.

Goff, D. C., Brotman, A. W., Kindlon, D., Waites, M., & Amico, E. (1991). Self-reports of childhood abuse in chronically psychotic patients. *Psychiatry Research, 37*, 73-80.

Goldman, H. H., Skodol, A. E., & Lave, T. R. (1992). Revising axis V for DSM-IV: a review of measures of social functioning. *American Journal of Psychiatry, 149*, 1148-1156.

Gooding, D. C., Tallent, K. A., & Hegyi, J. V. (2001). Cognitive slippage in schizotypic individuals. *Journal of Nervous and Mental Disease, 189*, 750-756.

Gooding, D. C., Tallent, K. A., & Matts, C. W. (2005). Clinical status of at-risk individuals 5 years later: further validation of the psychometric high-risk strategy. *Journal of Abnormal Psychology, 114*, 170-175.

Goodman, L. A., Rosenberg, S. D., Mueser, K. T., & Drake, R. E. (1997). Physical and sexual assault history in women with serious mental illness: prevalence, correlates, treatment, and future research directions. *Schizophrenia Bulletin, 23*, 685-696.

Gorsuch, R. L. (1993). *Factor Analysis, 2nd edition*. Hillsdale, NJ: Erlbaum.

Gottesman, I., & Shields, J. (1982). *Schizophrenia: The Epigenetic Puzzle*. Cambridge: Cambridge University Press.

Goulding, A. (2005). Healthy schizotypy in a population of paranormal believers and experients. *Personality and Individual Differences, 38*, 1069-1083.

Gourevitch, R., Abbadi, S., & Guelfi, J. D. (2004). Quality of life in schizophrenics with and without the deficit syndrome. *European Psychiatry, 19*, 172-174.

Gracie, A., Freeman, D., Green, S., Garety, P. A., Kuipers, E., Hardy, A., et al. (2007). The association between traumatic experience, paranoia and hallucinations: a test of the predictions of psychological models. *Acta Psychiatrica Scandinavica, 116*, 280-289.

Gray, J. A. (1982). *The Neuropsychology of Anxiety: an Enquiry into the Functions of the Septohippocampal System*. Oxford: Oxford University Press.

Gray, J. A. (1993). Consciousness, schizophrenia and scientific theory. *Ciba Foundation Symposium, 174*, 263-273; discussion 273-281.

Gray, J. A. (1995). The contents of consciousness: a neuropsychological conjecture. *Behavioural and Brain Sciences, 18*, 617-680.

Gray, J. A. (1998a). Abnormal contents of consciousness: the transition from automatic to controlled processing. *Advances in Neurology, 77*, 195-208; discussion 208-211.

Gray, J. A. (1998b). Integrating schizophrenia. *Schizophrenia Bulletin, 24*, 249-266.

Gray, J. A., Feldon, J., Rawlins, J. N. P., Hemsley, D., & Smith, A. D. (1991). The neuropsychology of schizophrenia. *Behavioural and Brain Sciences, 14*, 1-19; discussion 20-84.

Gray, N. S., Pilowsky, L. S., Gray, J. A., & Kerwin, R. W. (1995). Latent inhibition in drug naive schizophrenics: relationship to duration of illness and dopamine D2 binding using SPET. *Schizophrenia Research, 17*, 95-107.

Green, B. L. (1996). Psychometric review of Trauma History Questionnaire (self-report). In B. H. Stamm & E. M. Varra (Eds.), *Measurement of Stress, Trauma and Adaptation*. Lutherville, MD: Sidran.

Green, C., Garety, P. A., Freeman, D., Fowler, D., Bebbington, P., Dunn, G., et al. (2006). Content and affect in persecutory delusions. *British Journal of Clinical Psychology, 45*, 561-577.

Green, J. (2006). The evolving randomised controlled trial in mental health: studying complexity and treatment process. *Advances in Psychiatric Treatment, 12*, 268-279.

Green, J., & Dunn, G. (2008). Using intervention trials in developmental psychiatry to illuminate basic science. *British Journal of Psychiatry, 192*, 323-325.

Green, M. F. (1992). Information processing in schizophrenia. In D. J. Kavanagh (Ed.), *Schizophrenia: An Overview and Practical Handbook* (pp. 45-58). London: Chapman & Hall.

Green, M. F. (2001). *Schizophrenia Revealed*. New York: Norton.

Green, M. J., & Phillips, M. (2004). Social threat perception and the evolution of paranoia. *Neuroscience and Biobehavioral Reviews, 28*, 333-342.

Green, M. J., Williams, L. M., & Davidson, D. (2003). Visual scanpaths and facial affect recognition in delusion-prone individuals: Increased sensitivity to threat? *Cognitive Neuropsychiatry, 8*, 19-41.

Greenfield, S. F., Strakowski, S. M., Tohen, M., Batson, S. C., & Kolbrener, M. L. (1994). Childhood abuse in first-episode psychosis. *British Journal of Psychiatry, 164*, 831-834.

Gregory, R. L. (1998). *Eye and Brain: The Psychology of Seeing* (5th ed.). Oxford: Oxford University Press.

Gross, G., Huber, G., & Klosterkötter, J. (1987). *Bonn Scale for the Assessment of Basic Symptoms - BSABS*. Berlin: Springer.

Grove, W. M., Lebow, B. S., Clementz, B. A., Cerri, A., Modus, C., & Iacono, W. G. (1991). Familial prevalence and coaggregation of schizotypy indicators: a multitrait family study. *Journal of Abnormal Psychology, 100*, 115-121.

Grube, B. S., Bilder, R. M., & Goldman, R. S. (1998). Meta-analysis of symptom factors in schizophrenia. *Schizophrenia Research, 31*, 113-120.

Gruzelier, J. H. (1996). The factorial structure of schizotypy: Part I. Affinities with syndromes of schizophrenia. *Schizophrenia Bulletin, 22*, 611-620.

Gschwandtner, U., Aston, J., Borgwardt, S., Drewe, M., Feinendegen, C., Lacher, D., et al. (2003). Neuropsychological and neurophysiological findings in individuals suspected to be at risk for schizophrenia: preliminary findings from the Basel early detection of psychosis study - Fruherkennung von Psychosen (FEPSY). *Acta Psychiatrica Scandinavica, 108*, 152-155.

Guillem, F., Pampoulova, T., Stip, E., Lalonde, P., & Todorov, C. (2005). The relationships between symptom dimensions and dysphoria in schizophrenia. *Schizophrenia Research, 75*, 83-96.

Gumley, A., O'Grady, M., McNay, L., Reilly, J., Power, K., & Norrie, J. (2003). Early intervention for relapse in schizophrenia: results of a 12-month randomised controlled trial of cognitive behavioural therapy. *Psychological Medicine, 33*, 419-431.

Gumley, A., O'Grady, M., Power, K., & Schwannauer, M. (2004). Negative beliefs about self and illness: a comparison of individuals with psychosis with or without comorbid social anxiety disorder. *Australian and New Zealand Journal of Psychiatry, 38*, 960-964.

Gumley, A., & Schwannauer, M. (2006). *Staying Well After Psychosis: A Cognitive Interpersonal Approach to Recovery and Relapse Prevention*. Chichester: Wiley.

Gumley, A., White, C. A., & Power, K. (1999). An interacting cognitive subsystems model of relapse and the course of psychosis. *Clinical Psychology and Psychotherapy, 6*, 261-279.

Gunderson, J. G., Siever, L. J., & Spaulding, E. (1983). The search for the schizotype: crossing the border again. *Archives of General Psychiatry, 40*, 15-22.

Gureje, O., Harvey, C., & Herrman, H. (2004). Self-esteem in patients who have recovered from psychosis: profile and relationship to quality of life. *Australian and New Zealand Journal of Psychiatry, 38*, 334-338.

Häfner, H., Löffler, W., Maurer, K., Hambrecht, M., & an der Heiden, W. (1999). Depression, negative symptoms, social stagnation and social decline in the early course of schizophrenia. *Acta Psychiatrica Scandinavica, 100*, 105-118.

Häfner, H., Maurer, K., Ruhrmann, S., Bechdolf, A., Klosterkötter, J., Wagner, M., et al. (2004). Early detection and secondary prevention of psychosis: facts and visions. *European Archives of Psychiatry and Clinical Neuroscience, 254*, 117-128.

Hamilton, M. (1960). A rating scale for depression. *Journal of Neurology, Neurosurgery and Psychiatry, 23*, 56-62.

Hammersley, P., Dias, A., Todd, G., Bowen-Jones, K., Reilly, B., & Bentall, R. P. (2003). Childhood trauma and hallucinations in bipolar affective disorder: preliminary investigation. *British Journal of Psychiatry, 182*, 543-547.

Hanssen, M., Bak, M., Bijl, R., Vollebergh, W., & van Os, J. (2005). The incidence and outcome of subclinical psychotic experiences in the general population. *British Journal of Clinical Psychology, 44*, 181-191.

Harb, G. C., Heimberg, R. G., Fresco, D. M., Schneier, F. R., & Liebowitz, M. R. (2002). The psychometric properties of the Interpersonal Sensitivity Measure in social anxiety disorder. *Behaviour Research and Therapy, 40*, 961-979.

Harding, C. M. (1988). Course types in schizophrenia: an analysis of European and American studies. *Schizophrenia Bulletin, 14*, 633-643.

Harding, C. M., Brooks, G. W., Ashikaga, T., Strauss, J. S., & Breier, A. (1987). The Vermont longitudinal study of persons with severe mental illness, I. Methodology, study sample, and overall status 32 years later. *American Journal of Psychiatry, 144*, 718-726.

Hardy, A., Fowler, D., Freeman, D., Smith, B., Steel, C., Evans, J., et al. (2005). Trauma and hallucinatory experience in psychosis. *Journal of Nervous and Mental Disease, 193*, 501-507.

Harrison, G., Croudace, T., Mason, P., Glazebrook, C., & Medley, I. (1996). Predicting the long-term outcome of schizophrenia. *Psychological Medicine, 26*, 697-705.

Harrison, G., Hopper, K., Craig, T., Laska, E., Siegel, C., Wanderling, J., et al. (2001). Recovery from psychotic illness: a 15- and 25- year international follow-up study. *British Journal of Clinical Psychiatry, 178*, 506-517.

Harrison, P. J., & Owen, M. J. (2003). Genes for schizophrenia? Recent findings and their pathophysiological implications. *Lancet, 361*, 417-419.

Hathaway, S. R., & McKinley, J. C. (1951). *The MMPI Manual*. New York: The Psychological Corporation.

Hawkins, K. A., McGlashan, T. H., Quinlan, D., Miller, T. J., Perkins, D. O., Zipursky, R. B., et al. (2004). Factorial structure of the Scale of Prodromal Symptoms. *Schizophrenia Research, 68*, 339-347.

Hazlett, E. A., Dawson, M. E., Filion, D. L., Schell, A. M., & Nuechterlein, K. (1997). Autonomic orienting and the allocation of processing resources in schizophrenia patients and putatively at-risk individuals. *Journal of Abnormal Psychology, 106*, 171-181.

Heinimaa, M., Salokangas, R., Ristkari, T., Plathin, M., Huttunen, J., Ilonen, T., et al. (2003). PROD-screen - a screen for prodromal symptoms of psychosis. *International Journal of Methods in Psychiatric Research, 12*, 92-104.

Heinrichs, D. W., Hanlon, T. E., & Carpenter, B. N. (1984). The Quality of Life Scale: an instrument for rating the schizophrenic deficit syndrome. *Schizophrenia Bulletin, 10*, 388-398.

Helson, H. (1964). *Adaptation-level Theory: An Experimental and Systematic Approach to Behavior*. New York: Harper and Row.

Hemsley, D. (1987). An experimental psychological model for schizophrenia. In H. Häfner, W. Gattaz & W. Janzarik (Eds.), *Search for the Causes of Schizophrenia* (Vol. 1, pp. 179-188). New York: Springer.

Hemsley, D. (1992). Cognitive abnormalities and schizophrenic symptoms. *Psychological Medicine, 22*, 839-842.

Hemsley, D. (1993). A simple (or simplistic?) cognitive model for schizophrenia. *Behaviour Research and Therapy, 31*, 633-646.

Hemsley, D. (1998). The disruption in the sense of self in schizophrenia: potential links with disturbances of information processing. *British Journal of Medical Psychology, 71*, 115-124.

Hemsley, D. (2005a). The development of a cognitive model of schizophrenia: placing it in context. *Neuroscience and Biobehavioral Reviews, 29*, 977-988.

Hemsley, D. (2005b). The schizophrenic experience: taken out of context? *Schizophrenia Bulletin, 31*, 43-53.

Henquet, C., Murray, R., Linszen, D., & van Os, J. (2005). The environment and schizophrenia: the role of cannabis use. *Schizophrenia Bulletin, 31*, 608-612.

Hirsch, S. R., & Weinberger, D. R. (2003). *Schizophrenia*. Oxford: Blackwell Science.

Ho, B., Nopoulos, P., Flaum, M., Arndt, S., & Andreasen, N. C. (1998). Two-year outcome in first-episode schizophrenia: predictive value of symptoms for quality of life. *American Journal of Psychiatry, 155*, 1196-1201.

Hodges, A., Byrne, M., Grant, E., & Johnstone, E. (1989). People at risk of schizophrenia: sample characteristics of the first 100 cases in the Edinburgh high-risk study. *British Journal of Psychiatry, 174*, 547-553.

Hoffmann, H., & Kupper, Z. (2002). Facilitators of psychosocial recovery from schizophrenia. *International Review of Psychiatry, 14*, 293-302.

Hoffmann, H., Kupper, Z., & Kunz, B. (2000). Hopelessness and its impact on rehabilitation outcome in schizophrenia - an exploratory study. *Schizophrenia Research, 43*, 147-158.

Hogarty, G. E., Goldberg, S. C., Schooler, N. R., & Ulrich, R. F. (1974). Drug and sociotherapy in the aftercare of schizophrenic patients, II: two-year relapse rates. *Archives of General Psychiatry, 31*, 603-608.

Hogarty, G. E., Kornblith, S. J., Greenwald, P., DiBarry, A. L., Cooley, S., Ulrich, R. F., et al. (1997). Three-year trials of personal therapy with schizophrenics living with or independent of family, I: description of study and effects on relapse rates. *American Journal of Psychiatry, 154*, 1504-1513.

Holmes, E. A., & Steel, C. (2004). Schizotypy: a vulnerability factor for traumatic intrusions. *Journal of Nervous and Mental Disease, 192*, 28-34.

Howitt, D., & Cramer, D. (2005). *Introduction to Research Methods in Psychology*. Harlow: Pearson Prentice Hall.

Humphrey, G. K. (1998). The McCollough effect: misperception and reality. In V. Walsh & J. J. Kulikowski (Eds.), *Perceptual Constancy: Why Things Look as They Do* (pp. 31-68). Cambridge: Cambridge University Press.

Humphrey, G. K., & Goodale, M. A. (1998). Probing unconscious visual processing with the McCollough effect. *Consciousness and Cognition, 7*, 494-519.

Iancu, I., Poren, A., Lehman, B., Shamir, E., & Kotler, M. (2005). The Positive and Negative Symptoms Questionnaire: a self-report scale in schizophrenia. *Comprehensive Psychiatry, 46*, 61-66.

Jablensky, A., McGrath, J., Herrman, H., Castle, D., Gureje, O., Morgan, V., et al. (1999). *People living with psychotic illness: An Australian Study 1997-1998*. Canberra: Commonwealth of Australia.

Jablensky, A., Sartorious, N. E., G., Anker, M., Korten, A., Cooper, J., & Bertelson, A. (1992). Schizophrenia: manifestations, incidence and course in different cultures. A World Health Organisation Ten Country Study. *Psychological Medicine, 20*, 1-97.

Jackson, C., & Iqbal, Z. (2002). Psychological adjustment to early psychosis. In M. Birchwood, D. Fowler & C. Jackson (Eds.), *Early Intervention in Psychosis: A Guide to Concepts, Evidence and Interventions*. Chichester: Wiley.

Jackson, H. J., & Edwards, J. (1992). Social networks and social support in schizophrenia: correlates and assessment. In D. J. Kavanagh (Ed.), *Schizophrenia: An Overview and Practical Handbook*. London: Chapman & Hall.

Jackson, H. J., McGorry, P. D., & Dudgeon, P. (1995). Prodromal symptoms of schizophrenia in first-episode psychosis: prevalence and specificity. *Comprehensive Psychiatry, 36*, 241-250.

Jackson, M. (1997). Benign schizotypy? The case of spiritual experience. In G. Claridge (Ed.), *Schizotypy. Implications for Illness and Health*. Oxford: Oxford University Press.

Jacobson, N., & Greenley, D. (2001). What is recovery? A conceptual model and explication. *Psychiatric Services, 52*, 482-485.

Janssen, I., Krabbendam, L., Bak, M., Hanssen, M., Vollebergh, W., Graaf, R., et al. (2004). Childhood abuse as a risk factor for psychotic experiences. *Acta Psychiatrica Scandinavica, 109*, 38-45.

Johannesen, J. K., Bodkins, M., O'Donnell, B. F., Shekhar, A., & Hetrick, W. P. (2008). Perceptual anomalies in schizophrenia co-occur with selective impairments in the gamma frequency component of midlatency auditory ERPs. *Journal of Abnormal Psychology, 117*, 106-118.

Johns, L. C., Cannon, M., Singleton, N., Murray, R. M., Farrell, M., Brugha, T., et al. (2004). Prevalence and correlates of self-reported psychotic symptoms in the British population. *British Journal of Psychiatry, 185*, 298-305.

Johns, L. C., Nazroo, J. Y., Bebbington, P., & Kuipers, E. (1998). Occurrence of hallucinations in a community sample. *Schizophrenia Research, 29*, 23.

Johns, L. C., & van Os, J. (2001). The continuity of psychotic experiences in the general population. *Clinical Psychology Review, 21*, 1125-1141.

Johnstone, E. C., Crow, T. J., Frith, C. D., Carney, M. W., & Price, J. (1978). Mechanism of the antipsychotic effect in the treatment of acute schizophrenia. *Lancet, 1*, 848-851.

Johnstone, E. C., Ebmeier, K. P., Miller, P., Owens, D. G., & Lawrie, S. M. (2005). Predicting schizophrenia: findings from the Edinburgh High-Risk Study. *British Journal of Psychiatry, 186*, 18-25.

Johnstone, E. C., Macmillan, J. F., Frith, C. D., Benn, D. K., & Crow, T. J. (1990). Further investigation of the predictors of outcome following first schizophrenic episodes. *British Journal of Psychiatry, 157*, 182-189.

Jolley, A. G., Hirsch, S. R., Morrison, E., McRink, A., & Wilson, L. (1990). Trial of brief intermittent neuroleptic prophylaxis for selected schizophrenic outpatients: clinical and social outcome at two years. *British Medical Journal, 301*, 837-842.

Jolley, S., Garety, P. A., Ellett, L., Kuipers, E., Freeman, D., Bebbington, P. E., et al. (2006). A validation of a new measure of activity in psychosis. *Schizophrenia Research, 85*, 288-295.

Jones, L. A., Cardno, A. G., Murphy, K. C., Sanders, R. D., Gray, M. Y., McCarthy, G., et al. (2000). The Kings Schizotypy Questionnaire as a quantitative measure of schizophrenia liability. *Schizophrenia Research, 45*, 213-221.

Jones, P., Rodgers, B., Murray, R. M., & Marmot, M. (1994). Child development risk factors for adult schizophrenia in the British 1946 birth cohort. *Lancet, 344*, 1398-1402.

Jones, S., & Bentall, R. (2006). *The Psychology of Bipolar Disorder: New Developments and Research Strategies*. Oxford: Oxford University Press.

Jorgensen, P. (1998). Early signs of psychotic relapse in schizophrenia. *British Journal of Psychiatry, 172*, 372-330.

Joseph, S., & Diduca, D. (2001). Schizotypy and religiosity in 13-18 year old school pupils. *Mental Health, Religion and Culture, 4*, 63-69.

Kampman, O., Caippala, P., Vaananen, J., Koivisto, E., Kiviniemi, P., Kilkku, N., et al. (2002). Indicators of medication compliance in first-episode psychosis. *Psychiatry Research, 110*, 39-48.

Kane, J. M., Honigfield, G., Singer, J., & Meltzer, H. (1988). Clozapine for the treatment resistant schizophrenic: a double blind comparison with chlorpromazine. *Archives of General Psychiatry, 45*, 789-796.

Kapur, S. (2003). Psychosis as a state of aberrant salience: a framework linking biology, phenomenology, and pharmacology in schizophrenia. *American Journal of Psychiatry, 160*, 13-23.

Kapur, S., Arenovich, T., Agid, O., Zipursky, R. B., Lindborg, S., & Jones, B. (2005). Evidence for onset of antipsychotic effects within the first 24 hours of treatment. *American Journal of Psychiatry, 162*, 939-946.

Kapur, S., & Mamo, D. C. (2004). Why antipsychotics are anti-'psychotic'. In C. McDonald, K. Schulze, R. Murrary & P. Wright (Eds.), *Schizophrenia: Challenging the Orthodox* (pp. 113-126). London: Taylor and Francis.

Kay, S. R. (1991). *Positive and Negative Syndromes in Schizophrenia: Assessment and Research*. New York: Brunner/Mazel.

Kay, S. R., Fiszbein, A., & Opler, L. A. (1987). The Positive and Negative Syndrome Scale (PANSS) for schizophrenia. *Schizophrenia Bulletin, 13*, 261-276.

Kaye, B. K., & Johnson, T. J. (1999). Research methodology: taming the cyber frontier. *Social Science Computer Review, 17*, 323-337.

Keitner, G. I., Soloman, D. A., Ryan, C. E., Miller, I. W., Mallinger, A., Kupfer, D. J., et al. (1996). Prodromal and residual symptoms in bipolar I disorder. *Comprehensive Psychiatry, 37*, 362-367.

Kendler, K. S., Gallagher, T. J., Abelson, J. M., & Kessler, R. C. (1996). Lifetime prevalence, demographic risk factors, and diagnostic validity of nonaffective psychosis as assessed in a US community sample: The national co-morbidity survey. *Archives of General Psychiatry, 53*, 1022-1031.

Kendler, K. S., Liebermann, J. A., & Walsh, D. (1989). The Structured Interview for Schizotypy (SIS): a preliminary report. *Schizophrenia Bulletin, 15*, 559-571.

Keshavan, M. S., Anderson, S., & Pettegrew, J. W. (1994). Is schizophrenia due to excessive synaptic pruning in the prefrontal cortex? The Feinberg hypothesis revisited. *Journal of Psychiatric Research, 28*, 239-265.

Kety, S., Rosenthal, D., Wender, P. H., Schulsinger, F., & Jacobsen, B. (1974). Mental illness in the biological and adoptive families of adopted individuals who have become schizophrenic: a preliminary report based on psychiatric interviews. In R. Fieve, D. Rosenthal & H. Brill (Eds.), *Genetic Research in Psychiatry*. Baltimore, MD: Johns Hopkins University Press.

Khouri, P. J., Haier, R. J., Rieder, R. O., & Rosenthal, D. (1980). A symptom schedule for the diagnosis of borderline schizophrenia: a first report. *British Journal of Psychiatry, 137*, 140.

Kirkpatrick, B., & Buchanan, R. W. (1990). Anhedonia and the deficit syndrome of schizophrenia. *Psychiatry Research, 31*, 25-30.

Kline, P. (1993). *An Easy Guide to Factor Analysis*. New York: Routledge.

Klosterkötter, J., Hellmich, M., Steinmeyer, E. M., & Schultze-Lutter, F. (2001). Diagnosing schizophrenia in the initial prodromal phase. *Archives of General Psychiatry, 58*, 158-164.

Klosterkötter, J., Schultze-Lutter, F., Wieneke, A., Picker, H., & Steinmeyer, E. M. (2001). Introduction and reliability of the first version of the Schizophrenia Prediction Instrument (SPI-A). *Schizophrenia Research, 49*, 4.

Knight, R. A. (1984). Converging models of cognitive deficit in schizophrenia. In W. D. Spaulding & J. K. Cole (Eds.), *Theories of Schizophrenia and Psychosis* (pp. 93-156). London: University of Nebraska Press.

Kolb, J. E., & Gunderson, J. G. (1980). Diagnosing borderline patients with a semi-structured interview. *Archives of General Psychiatry, 37*, 37.

König, H. H., Roick, C., & Angermeyer, M. C. (2007). Validity of the EQ-5D in assessing and valuing health status in patients with schizophrenic, schizotypal or delusional disorders. *European Psychiatry, 22*, 177-187.

Konings, M., Bak, M., Hanssen, M., van Os, J., & Krabbendam, L. (2006). Validity and reliability of the CAPE: a self-report instrument for the measurement of psychotic experiences in the general population. *Acta Psychiatrica Scandinavica, 114*, 55-61.

Kontaxakis, V. P., Kollias, C. T., Havaki-Kotaxaki, B. J., Margariti, M. M., Stamouli, S. S., Petridou, E., et al. (2006). Physical anhedonia in the acute phase of schizophrenia. *Annals of General Psychiatry, 5*, 1.

Koreen, A. R., Siris, S. G., Chakos, M., Alvir, J., Mayerhoff, D., & Lieberman, J. (1993). Depression in first-episode schizophrenia. *American Journal of Psychology, 150*, 1643-1648.

Krabbendam, L., Myin-Germeys, I., & van Os, J. (2004). The expanding psychosis phenotype. *International Journal of Psychology and Psychological Therapy, 4*, 411-421.

Kraemer, H. C., Fairburn, C. G., & Agras, W. S. (2002). Mediators and moderators of treatment effects in randomised clinical trials. *Archives of General Psychiatry, 59*, 877-883.

Kraepelin, E. (1919). *Dementia Praecox and Paraphrenia*. Reprinted 1971, Huntingdon, NY: Robert E. Krieger.

Kremen, W. S., Faraone, S. V., Toomey, R., Seidman, L. J., & Tsuang, M. T. (1998). Sex differences in self-reported schizotypal traits in relatives of schizophrenic probands. *Schizophrenia Research, 34*, 27-37.

Krstev, H., Jackson, H., & Maude, D. (1999). An investigation of attributional style in first-episode psychosis. *British Journal of Clinical Psychology, 38*, 181-194.

Kubany, E. S., Hanes, S. N., Leisen, M. B., Owens, J. A., Kaplan, A. S., Watson, S. B., et al. (2000). Development and preliminary validation of a brief and broad-spectrum measure of trauma exposure: the Traumatic Life Events Questionnaire. *Psychological Assessment, 12*, 210-224.

Kuipers, E., Garety, P., Fowler, D., Freeman, D., Dunn, G., & Bebbington, P. (2006). Cognitive, emotional, and social processes in psychosis: refining cognitive behaviour therapy for persistent positive symptoms. *Schizophrenia Bulletin, 32*(suppl.), S24-S31.

Kuipers, E., Garety, P. A., Fowler, D., Dunn, G., Bebbington, P., Freeman, D., et al. (1997). London-East Anglia randomised controlled trial of cognitive-behavioural therapy for psychosis I: effects of treatment phase. *British Journal of Psychiatry, 171*, 319-327.

Kwapil, T. R., Mann, M. C., & Raulin, M. L. (2002). Psychometric properties and concurrent validity of the schizotypal ambivalence scale. *Journal of Nervous and Mental Disease, 190*, 290-295.

Kwapil, T. R., Miller, M. B., Zinser, M. C., Chapman, J., & Chapman, L. J. (1997). Magical ideation and social anhedonia as predictors of psychosis proneness: a partial replication. *Journal of Abnormal Psychology, 106*, 491-495.

Kwapil, T. R., Raulin, M. L., & Midthun, J. C. (2000). A ten-year longitudinal study of intense ambivalence as a predictor of risk for psychopathology. *Journal of Nervous and Mental Disease, 188*, 402-408.

Laing, R. D. (1960). *The Divided Self*. London: Tavistock Press.

Laing, R. D. (1961). *Self and Others*. London: Tavistock Press.

Laithwaite, H. M., Gumley, A., Benn, A., Scott, E., Downey, K., Black, K., et al. (2007). Self-esteem and psychosis: a pilot study investigating the effectiveness of a self-esteem programme on the self-esteem and positive symptomatology of

mentally disordered offenders. *Behavioural and Cognitive Psychotherapy, 35*, 569-578.

Launay, G., & Slade, P. (1981). The measurement of hallucinatory predisposition in male and female prisoners. *Personality and Individual Differences, 2*, 221-234.

Leete, E. (1989). How I perceive and manage my illness. *Schizophrenia Bulletin, 15*, 197-200.

Leff, J., Tress, K., & Edwards, B. (1988). The clinical course of depressive symptoms in schizophrenia. *Schizophrenia Research, 1*, 25-30.

Lester, H., & Gask, L. (2006). Delivering medical care for patients with serious mental illness or promoting a collaborative model of recovery? *British Journal of Psychiatry, 188*, 401-402.

Leucht, S., Kane, J. M., Kissling, W., Hamaan, J., Etschel, E., & Engel, R. R. (2005). What does the PANSS mean? *Schizophrenia Research, 79*, 231-238.

Liberman, R. P. (2002). Future directions for research studies and clinical work on recovery from schizophrenia: questions with some answers. *International Review of Psychiatry, 14*, 337-342.

Liberman, R. P., & Kopelowicz, A. (2002). Recovery from schizophrenia: a challenge for the 21st century. *International Review of Psychiatry, 14*, 245-255.

Liberman, R. P., Kopelowicz, A., Ventura, J., & Gutkind, D. (2002). Operational criteria and factors related to recovery from schizophrenia. *International Review of Psychiatry, 14*, 256-272.

Liddle, P. F. (1987). Symptoms of chronic schizophrenia. A re-examination of the positive-negative dichotomy. *British Journal of Psychiatry, 151*, 145-151.

Lieberman, J., Drake, R., Sederer, L., Belger, A., Keefe, R., Perkins, D., et al. (2008). Science and recovery in schizophrenia. *Psychiatric Services, 59*, 487-496.

Lieberman, J. A., Kane, J. M., & Alvir, J. (1987). Provocative tests with psychostimulant drugs in schizophrenia. *Psychopharmacology, 91*, 415-433.

Liebowitz, M. R. (1987). Social phobia. *Modern Problems of Pharmacopsychiatry, 22*, 141-173.

Lincoln, T. M. (2007). Relevant dimensions of delusions: continuing the continuum versus category debate. *Schizophrenia Research, 93*, 211-220.

Liraud, F., Droulout, T., Parrot, M., & Verdoux, H. (2004). Agreement between self-rated and clinically assessed symptoms in subjects with psychosis. *Journal of Nervous and Mental Disease, 192*, 352-356.

Lobban, F., Barrowclough, C., & Jones, S. (2003). A review of the role of illness models in severe mental illness. *Clinical Psychology Review, 23*, 171-196.

Loewy, R. L., Bearden, C. E., Johnson, J. K., Raine, A., & Cannon, T. D. (2005). The prodromal questionnaire (PQ): preliminary validation of a self-report screening measure for prodromal and psychotic syndromes. *Schizophrenia Research, 77*, 141-149.

Logue, N. A., & Byth, W. (1993). Extraversion and the McCollough effect. *British Journal of Psychology, 84*, 67-84.

Lovejoy, M. (1984). Recovery from schizophrenia: a personal odyssey. *Hospital and Community Psychiatry, 35*, 809-812.

Lovibond, P. F., & Lovibond, S. H. (1995). The structure of negative emotional states: comparison of the depression anxiety stress scales (DASS) with the Beck depression and anxiety inventories. *Behaviour Research and Therapy, 33*, 335-343.

Lubow, R. E., & Gewirtz, J. C. (1995). Latent inhibition in humans: data, theory, and implications for schizophrenia. *Psychological Bulletin, 117*, 87-103.

Lubow, R. E., & Moore, A. U. (1959). Latent inhibition: the effect of nonreinforced pre-exposure to the conditional stimulus. *Journal of Comparative and Physiological Psychology, 52*, 415-419.

Lyon, H. M., Kaney, S., & Bentall, R. P. (1994). The defensive function of persecutory delusions. Evidence from attribution tasks. *British Journal of Psychiatry, 164*, 637-646.

Lyoo, I. K., Youn, T., Ha, T. H., Park, H. S., & Kwon, J. S. (2003). Classification of frequency distributions of diagnostic criteria scores in twelve personality disorders by the curve fitting method. *Psychiatry and Clinical Neurosciences, 57*, 417-423.

MacBeth, A., Schwannauer, M., & Gumley, A. (2008). The association between attachment style, social mentalities, and paranoid ideation: an analogue study. *Psychology and Psychotherapy: Theory, Research and Practice, 81*, 79-93.

Macmillan, I., Howells, L., Kale, K., Hackmann, C., Taylor, G., Hill, K., et al. (2007). Social and symptomatic outcomes of first-episode bipolar psychoses in an early intervention service. *Early Intervention in Psychiatry, 1*, 79-87.

Maher, B. A. (1983). A tentative theory of schizophrenic utterance. In B. A. Maher & W. B. Maher (Eds.), *Progress in Experimental Personality Research* (Vol. 12, pp. 1-52). New York: Academic Press.

Maher, B. A. (1988). Anomalous Experience and Delusional Thinking: The Logic of Explanations. In T. F. Oltmanns & B. A. Maher (Eds.), *Delusional Beliefs*. New York: Wiley.

Malla, A. K., Cortese, L., Shaw, T. S., & Ginsberg, B. (1990). Life events and relapse in schizophrenia. A one year prospective study. *Social Psychiatry and Psychiatric Epidemiology, 25*, 221-224.

Malla, A. K., Norman, R. M., & Williamson, P. (1993). Stability of positive and negative symptoms in schizophrenia. *Canadian Journal of Psychiatry, 38*, 617-621.

Malla, A. K., & Payne, J. (2005). First-episode psychosis: psychopathology, quality of life, and functional outcome. *Schizophrenia Bulletin, 31*, 650-671.

Marzillier, S. L., & Steel, C. (2007). Positive schizotypy and trauma-related intrusions. *Journal of Nervous and Mental Disease, 195*, 60-64.

Mason, O., & Claridge, G. (2006). The Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE): further description and extended norms. *Schizophrenia Research, 82*, 203-211.

Mason, O., Claridge, G., & Jackson, M. (1995). New scales for the assessment of schizotypy. *Personality and Individual Differences, 18*, 7-13.

Mason, O., Claridge, G., & Williams, L. (1997). Questionnaire measurement. In G. Claridge (Ed.), *Schizotypy: Implications for Illness and Health* (pp. 19-37). Oxford: Oxford University Press.

Mason, P., Harrison, G., Glazebrook, C., Medley, I., Dalkin, T., & Croudace, T. (1995). Characteristics of outcome in schizophrenia at 13 years. *British Journal of Psychiatry, 167*, 596-603.

Masters, K. J. (1995). Environmental trauma and psychosis. *Journal of the American Academy of Child and Adolescent Psychiatry, 34*, 1258-1259.

Matthews, G., & Deary, I. J. (1998). *Personality Traits*. Cambridge: Cambridge University Press.

Maurer, K., Hörrmann, F., Schmidt, M., Trendler, G., & Häfner, H. (2004). The Early Recognition Inventory ERIraos: a two-step procedure for the detection of "at-risk mental states". *Schizophrenia Research, 70*(suppl.), 76.

McCollough, C. (1965). Colour adaptation of edge-detectors in the human visual system. *Science, 149*, 1115-1116.

McCreery, C., & Claridge, G. (2002). Healthy schizotypy: the case of out-of-the-body experiences. *Personality and Individual Differences, 32*, 141-154.

McEwen, B. S., & Sapolsky, R. M. (1995). Stress and cognitive function. *Current Opinion in Neurobiology, 5*, 205-216.

McGlashan, T. H. (1987). Testing DSM-III symptoms criteria for schizotypal and borderline personality disorders. *Archives of General Psychiatry, 44*, 143-148.

McGlashan, T. H. (1996). Early detection and intervention in schizophrenia: research. *Schizophrenia Bulletin, 22*, 327-345.

McGlashan, T. H., Miller, T. J., Woods, S. W., Rosen, J. L., Hoffman, R. E., & Davidson, L. (2003). *Structured Interview for Prodromal Syndromes, Ver. 4.0*. New Haven, CT: Yale School of Medicine.

McGorry, P. (1992). The concept of recovery and secondary prevention in psychotic disorders. *Australian and New Zealand Journal of Psychiatry, 26*, 3-17.

McGorry, P., Chanen, A., McCarthy, E., Van Riel, R., McKenzie, D., & Singh, B. S. (1991). Posttraumatic stress disorder following recent-onset psychosis. An unrecognised postpsychotic syndrome. *Journal of Nervous and Mental Disease, 179*, 253-258.

McGorry, P. D., McFarlane, C. A., Patton, G., Bell, R. C., Hibbert, M., Jackson, H., et al. (1995). The prevalence of prodromal features of schizophrenia in adolescence: a preliminary survey. *Acta Psychiatrica Scandinavica, 92*, 241-249.

McGuffin, P., Farmer, A., & Harvey, I. (1991). A polydiagnostic application of operational criteria in studies of psychotic illness. Development and reliability of the OPCRIT system. *Archives of General Psychiatry, 48*, 764-770.

Meehl, P. E. (1962). Schizotaxia, schizotypy and schizophrenia. *American Psychologist, 17*, 827-838.

Meehl, P. E. (1990). Toward an integrated theory of schizotaxia, schizotypy, and schizophrenia. *Journal of Personality Disorders, 4*, 1-99.

Melzer, D., Tom, B. D., Brugha, T. S., Fryers, T., & Meltzer, H. (2002). Common mental disorder symptom counts in populations: are there distinct case groups above epidemiological cut-offs? *Psychological Medicine, 32*, 1195-1201.

Meng, Z., Rosenthal, R., & Rubin, D. (1992). Comparing correlated correlation coefficients. *Psychological Bulletin, 111*, 172-175.

Meyer, T. D., & Hautzinger, M. (1999). Two-year stability of psychosis proneness scales and their relations to personality disorder traits. *Journal of Personality Assessment, 73*, 472-488.

Miers, T. C., & Raulin, M. L. (1985). *The development of a scale to measure Cognitive Slippage.* Paper presented at the Fortieth Eastern Psychological Association Convention, Boston, MA.

Miles, J., & Shevlin, M. (2001). *Applying Regression and Correlation: A Guide for Students and Researchers*. London: Sage.

Miller, P., Byrne, M., Hodges, A., Lawrie, S. M., Owens, D. G. C., & Johnstone, E. C. (2002). Schizotypal components in people at high risk of developing schizophrenia: early findings from the Edinburgh High-Risk Study. *British Journal of Psychiatry, 180*, 179-184.

Miller, P., Lawrie, S. M., Byrne, M., Cosway, R., & Johnstone, E. C. (2002). Self-rated schizotypal cognitions, psychotic symptoms and the onset of schizophrenia in young people at high risk of schizophrenia. *Acta Psychiatrica Scandinavica, 105*, 341-345.

Miller, T. J., Cicchetti, D., Markovich, P. J., McGlashan, T. H., & Woods, S. W. (2004). The SIPS screen: a brief self-report screen to detect the schizophrenia prodrome. *Schizophrenia Research, 70*(suppl.), 78.

Miller, T. J., McGlashan, T. H., Woods, S. W., Stein, K., Driesen, N., Cocoran, C. M., et al. (1999). Symptom assessment in schizophrenic prodromal states. *Psychiatric Quarterly, 70*, 273-287.

Mishlove, M., & Chapman, L. J. (1985). Social anhedonia in the prediction of psychosis proneness. *Journal of Abnormal Psychology, 94*, 384-396.

Mizrahi, R., Bagby, R. M., Zipursky, R. B., & Kapur, S. (2005). How antipsychotics work: the patients' perspective. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 29*, 859-864.

Moghaddam, B., & Bunney, B. S. (1990). Acute effects of typical and atypical antipsychotic drugs and the release of dopamine from prefrontal cortex, nucleus accumbens and striatum of the rat: an in vivo microdialysis study. *Journal of Neurochemistry, 54*, 1755-1760.

Mohr, C., Landis, T., Sandor, P. S., Fathi, M., & Brugger, P. (2004). Nonstereotyped responding in positive schizotypy after a single dose of levodopa. *Neuropsychopharmacology, 29*, 1741-1751.

Morrison, A. P. (1998). Cognitive behaviour therapy for psychotic symptoms in schizophrenia. In N. Tarrier (Ed.), *Treating Complex Cases: The Cognitive Behavioural Therapy Approach* (pp. 195-216). Chichester: Wiley.

Morrison, A. P. (2001). The interpretation of intrusions in psychosis: an integrative cognitive approach to hallucinations and delusions. *Behavioural and Cognitive Psychotherapy, 29*, 257-276.

Morrison, A. P., Bentall, R. P., French, P., Kilcommons, A., Knight, A., Kreutz, M., et al. (2002). Randomised controlled trial of early detection and cognitive therapy for preventing transition to psychosis in high risk individuals. Study design and interim analysis of transition rate and psychological risk factors. *British Journal of Psychiatry, 181*(suppl.), S78-S84.

Morrison, A. P., Frame, L., & Larkin, W. (2003). Relationships between trauma and psychosis: A review and integration. *British Journal of Clinical Psychology, 42*, 331-353.

Morrison, A. P., Wells, A., & Nothard, S. (2000). Cognitive factors in predisposition to auditory and visual hallucinations. *British Journal of Clinical Psychology, 39*, 67-78.

Mueser, K. T., Corrigan, P. W., Hilton, D. W., Tanzman, B., Schaub, A., Gingerich, S., et al. (2002). Illness management and recovery: a review of the research. *Psychiatric Services, 53*, 1272-1284.

Mueser, K. T., Goodman, L. B., Trumbetta, S. L., Rosenberg, S. D., Osher, C., Vidaver, R., et al. (1998). Trauma and posttraumatic stress disorder in severe mental illness. *Journal of Consulting and Clinical Psychology, 66*, 493-499.

Mueser, K. T., Rosenberg, S. D., Goodman, L. A., & Trumbetta, S. L. (2002). Trauma, PTSD, and the course of severe mental illness: an interactive model. *Schizophrenia Research, 53*, 123-143.

Mueser, K. T., Salyers, M. P., & Mueser, P. R. (2001). A prospective analysis of work in schizophrenia. *Schizophrenia Bulletin, 27*, 281-296.

Mueser, K. T., Sayers, S. L., Schooler, N. R., Mance, R. M., & Haas, G. L. (1994). A multisite investigation of the reliability of the Scale for the Assessment of Negative Symptoms. *American Journal of Psychiatry, 151*, 1453-1462.

Muntaner, C., Garcia-Sevilla, L., Fernandez, A., & Torrubia, R. (1988). Personality dimensions, schizotypal and borderline personality traits and psychosis proneness. *Personality and Individual Differences, 9*, 257-268.

Murray, R. M., & Lewis, S. W. (1987). Is schizophrenia a neurodevelopmental disorder? *British Medical Journal, 295*, 681-682.

Muthén, L. K., & Muthén, B. O. (1998). *Mplus User's Guide. Fourth Edition.* Los Angeles, CA: Muthén & Muthén.

Myin-Germeys, I., Delespaul, P., & van Os, J. (2005). Behavioural sensitization to daily life stress in psychosis. *Psychological Medicine, 35*, 733-741.

Myin-Germeys, I., Krabbendam, L., Jolles, J., Delespaul, P., & van Os, J. (2002). Are cognitive impairments associated with sensitivity to stress in schizophrenia? An experience sampling study. *American Journal of Psychiatry, 159*, 443-449.

Myin-Germeys, I., Krabbendam, L., & van Os, J. (2003). Continuity of psychotic symptoms in the community. *Current Opinion in Psychiatry, 16*, 443-449.

Myin-Germeys, I., van Os, J., Schwartz, J. E., Stone, A. A., & Delespaul, P. A. (2001). Emotional reactivity to daily life stress in psychosis. *Archives of General Psychiatry, 58*, 1137-1144.

Narvaez, J. M., Twamley, E. W., McKibbin, C. L., Heaton, R. K., & Patterson, T. L. (2008). Subjective and objective quality of life in schizophrenia. *Schizophrenia Research, 98*, 201-208.

Nielsen, T. C., & Petersen, N. E. (1976). Electrodermal correlates of extraversion, trait anxiety, and schizophrenism. *Scandinavian Journal of Psychology, 17*, 73-80.

Nithsdale, V., Davies, J., & Croucher, P. (2008). Psychosis and the experience of employment. *Journal of Occupational Rehabilitation, 18*, 175-182.

Noordsy, D., Torrey, W., Mueser, K., Mead, S., O'Keefe, C., & Fox, L. (2002). Recovery from severe mental illness: an intrapersonal and functional outcome definition. *International Review of Psychiatry, 14*, 318-326.

Norman, R. M., & Malla, A. K. (1991). Dysphoric mood and symptomatology in schizophrenia. *Psychological Medicine, 21*, 897-903.

Norman, R. M., Malla, A. K., Cortese, L., & Diaz, F. (1998). Aspects of dysphoria and symptoms of schizophrenia. *Psychological Medicine, 28*, 1433-1441.

Norris, F. H., & Hamblen, J. L. (2004). Standardised self-report measures of civilian trauma and PTSD. In J. P. Wilson & T. M. Keane (Eds.), *Assessing Psychological Trauma and PTSD, 2nd edition*. London: Guilford Publications.

Nuechterlein, K. H., & Dawson, M. E. (1984). A heuristic vulnerability/stress model of schizophrenic episodes. *Schizophrenia Bulletin, 10*, 300-312.

O'Reilly, T., Dunbar, R., & Bentall, R. (2001). Schizotypy and creativity: An evolutionary connection? *Personality and Individual Differences, 31*, 1067-1078.

Office for National Statistics. (2003). The United Kingdom 2000 Time Use Survey - Technical Report. Available online at: http://www.statistics.gov.uk/timeuse/default.asp.

Ohayon, M. M. (2000). Prevalence of hallucinations and their pathological associations in the general population. *Psychiatry Research, 97*, 153-164.

Olfson, M., Lewis-Fernandez, R., Weissman, M. M., Feder, A., Gameroff, M. J., Pilowsky, D., et al. (2002). Psychotic symptoms in an urban general medicine practice. *American Journal of Psychiatry, 159*, 1412-1419.

Olsen, K. A., & Rosenbaum, B. (2006). Prospective investigations of the prodromal state of schizophrenia: review of studies. *Acta Psychiatrica Scandinavica, 113*, 247-272.

Oltmanns, T. F., & Maher, B. A. (1988). *Delusional Beliefs*. New York: Wiley.

Ord, L. M., & Myles-Worsley, M. (2004). Screening prodromal adolescents in an isolated high-risk population. *Schizophrenia Research, 71*, 507-508.

Osman, A., Jones, K., & Osman, J. R. (1990). Psychometric properties of the social fear scale. *Psychological Reports, 67*, 1367-1373.

Overall, J. E., & Gorham, D. R. (1962). The brief psychiatric rating scale. *Psychological Reports, 10*, 799-812.

Pallanti, S., Quercieli, L., & Hollander, E. (2004). Social anxiety in outpatients with schizophrenia: a relevant cause of disability. *Psychological Medicine, 31*, 1293-1306.

Parnas, J., Møller, P., Kircher, T., Thalbitzer, J., Jansson, L., Handest, P., et al. (2005). EASE: Examination of Anomalous Self-Experience. *Psychopathology, 38*, 236-258.

Peralta, V., & Cuesta, M. J. (1994). Psychometric properties of the Positive and Negative Syndrome Scale (PANSS) in schizophrenia. *Psychiatry Research, 53*, 31-40.

Peralta, V., & Cuesta, M. J. (1999). Dimensional structure of psychotic symptoms: an item-level analysis of SAPS and SANS symptoms in psychotic disorders. *Schizophrenia Research, 38*, 13-26.

Peralta, V., de Leon, J., & Cuesta, M. J. (1992). Are there more than two syndromes in schizophrenia? A critique of the positive-negative dichotomy. *British Journal of Psychiatry, 161*, 335-343.

Perry, W., & Braff, D. L. (1994). Information processing deficits and thought disorder in schizophrenia. *American Journal of Psychiatry, 151*, 363-367.

Persons, J. B. (1986). The advantages of studying psychological phenomena rather than psychiatric diagnosis. *American Psychologist, 41*, 1252-1260.

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

Peters, E. R., Joseph, S. A., & Garety, P. A. (1999). Measurement of delusional ideation in the normal population: Introducing the PDI (Peters et al. Delusions Inventory). *Schizophrenia Bulletin, 25*, 553-576.

Phillips, L., Francey, S., Edwards, J., & McMurray, N. (2007). Stress and psychosis: towards the development of new models of investigation. *Clinical Psychology Review, 27*, 307-317.

Pilling, S., Bebbington, P., Kuipers, E., Garety, P. A., Geddes, J., Martindale, B., et al. (2002). Psychological treatments in schizophrenia, II: Meta-analyses of randomised controlled trial of social skills training and cognitive remediation. *Psychological Medicine, 32*, 783-791.

Pilling, S., Bebbington, P., Kuipers, E., Garety, P. A., Geddes, J., Orbach, G., et al. (2002). Psychological treatments in schizophrenia, I: Meta-analysis of family intervention and cognitive behaviour therapy. *Psychological Medicine, 32*, 763-782.

Pini, S., Cassano, G. B., Dell'Oso, L., & Amador, X. F. (2001). Insight into illness in schizophrenia, schizoaffective disorder and mood disorders with psychotic features. *American Journal of Psychiatry, 158*, 122-125.

Pitt, L., Kilbride, M., Nothard, S., Welford, M., & Morrison, A. P. (2007). Researching recovery from psychosis: a user-led project. *Psychiatric Bulletin, 31*, 55-60.

Pope, H. G., Jonas, J. M., Hudson, J. I., Cohen, B. M., & Tohen, M. (1985). An empirical study of psychosis in borderline personality disorder. *American Journal of Psychiatry, 142*, 1285-1290.

Poulton, R., Caspi, A., Moffitt, T. E., Cannon, M., Murray, R., & Harrington, H. (2000). Children's self-reported psychotic symptoms and adult schizophreniform disorder: a 15-year longitudinal study. *Archives of General Psychiatry, 57*, 1053-1058.

Power, P. J., Bell, R. J., Mills, R., Herrman-Doig, T., Davern, M., Henry, L., et al. (2003). Suicide prevention in first episode psychosis: the development of a randomised controlled trial of cognitive therapy for acutely suicidal patients with early psychosis. *Australian and New Zealand Journal of Psychiatry, 37*, 414-420.

Prescott, H. M. (2002). Using the student body: college and university students as research subjects in the United States during the twentieth century. *Journal of the History of Medicine and Allied Sciences, 57*, 3-38.

Rado, S. (1953). Dynamics and classification of disordered behaviour. *American Journal of Psychiatry, 110*, 406-416.

Rahman, A. (1992). Psychological factors in criminality. *Personality and Individual Differences, 13*, 483-485.

Raine, A. (1991). The SPQ: a scale for the assessment of schizotypal personality based on DSM-III-R criteria. *Schizophrenia Bulletin, 17*, 555-564.

Raine, A., & Allbutt, J. (1989). Factors of schizoid personality. *British Journal of Clinical Psychology, 28*, 31-40.

Raine, A., & Benishay, D. (1995). The SPQ-B: a brief screening instrument for schizotypal personality disorder. *Journal of Personality Disorders, 9*, 346-355.

Raine, A., & Lencz, T. (1995). Conceptual and methodological issues in schizotypal personality research. In A. Raine, T. Lencz & S. A. Mednick (Eds.), *Schizotypal Personality Disorder*. Cambridge: Cambridge University Press.

Raine, A., Lencz, T., & Mednick, S. A. (1995). *Schizotypal Personality*. Cambridge: Cambridge University Press.

Raine, A., & Manders, D. (1988). Schizoid personality, inter-hemispheric transfer, and left-hemisphere over-activation. *British Journal of Clinical Psychology, 27*, 333-347.

Raine, A., Reynolds, C., Lencz, T., Scerbo, A., Triphon, N., & Kim, D. (1994). Cognitive-perceptual, interpersonal and disorganised features of schizotypal personality disorder. *Schizophrenia Bulletin, 20*, 191-201.

Raine, A., Sheard, C., Reynolds, G. P., & Lencz, T. (1992). Pre-frontal structural and functional deficits associated with individual differences in schizotypal personality. *Schizophrenia Research, 7*, 237-247.

Raulin, M. L. (1984). Development of a scale to measure intense ambivalence. *Journal of Consulting and Clinical Psychology, 52*, 63-72.

Raulin, M. L., & Wee, J. L. (1984). The development and initial validation of a scale of social fear. *Journal of Clinical Psychology, 40*, 780-784.

Rawlings, D., & Freeman, J. L. (1996). A questionnaire for the measurement of paranoia/suspiciousness. *British Journal of Clinical Psychology, 35*, 451-461.

Read, J., Agar, K., Argyle, N., & Aderhold, V. (2003). Sexual and physical abuse during childhood and adulthood as predictors of hallucinations, delusions and thought disorder. *Psychology and Psychotherapy: Theory, Research and Practice, 76*, 1-22.

Read, J., & Argyle, N. (1999). Hallucinations, delusions, and thought disorder among adult psychiatric inpatients with a history of child abuse. *Psychiatric Services, 50*, 1467-1472.

Read, J., Goodman, L., Morrison, A. P., Ross, C., & Aderhold, V. (2004). Childhood trauma, loss and stress. In J. Read, L. Mosher & R. Bentall (Eds.), *Models of Madness: Psychological, Social and Biological Approaches to Schizophrenia* (pp. 223-252). Hove, UK: Brunner-Routledge.

Read, J., Perry, B., Moskowitz, A., & Connolly, J. (2001). The contribution of early traumatic events to schizophrenia in some patients: a traumagenic neurodevelopmental model. *Psychiatry: Interpersonal and Biological Processes, 64*, 319-345.

Read, J., & Ross, C. A. (2003). Psychological trauma and psychosis: another reason why people diagnosed schizophrenic must be offered psychological therapies. *Journal of the American Academy of Psychoanalysis and Dynamic Psychiatry, 31*, 247-268.

Read, J., van Os, J., Morrison, A. P., & Ross, C. A. (2005). Childhood trauma, psychosis and schizophrenia: a literature review with theoretical and clinical implications. *Acta Psychiatrica Scandinavica, 112*, 330-350.

Rector, N. A., & Beck, A. T. (2001). Cognitive behavioral therapy for schizophrenia: an empirical review. *Journal of Nervous and Mental Disease, 189*, 278-287.

Reich, T., James, J., & Morris, C. (1972). The use of multiple thresholds in determining the mode of transmission of semi-continuous traits. *Annals of Human Genetics, 36*, 163-184.

Reith, J., Benkelfat, C., Sherwin, A., Yasuhara, Y., Kuwabara, H., Andermann, F., et al. (1994). Elevated dopa decarboxylase activity in living brain of patients with psychosis. *Proceedings of the National Academy of Sciences, USA, 91*, 11651-11654.

Resnick, S. G., Rosenheck, R., & Lehman, A. (2004). An exploratory analysis of correlates of recovery. *Psychiatric Services, 55*, 540-547.

Riecher-Rössler, A., Aston, J., Ventura, J., Merlo, M., Borgwardt, S., Gschwandtner, U., et al. (2008). The Basel Screening Instrument for Psychosis (BSIP): development, structure, reliability and validity. *Fortschritte der Neurologie-Psychiatrie, 76*, 207-216.

Riecher-Rössler, A., Gschwandtner, U., Borgwardt, S., Aston, J., Pfluger, M., & Rossler, W. (2006). Early detection and treatment of schizophrenia: how early? *Acta Psychiatrica Scandinavica, 113*(suppl.), S73-S80.

Riecher, A., Maurer, K., Löffler, W., Fätkenheuer, B., an der Heiden, W., & Häfner, H. (1989). Schizophrenia: a disease of young single males? Preliminary results from an investigation on a representative cohort admitted to hospital for the first time. *European Archives of Psychiatry and Neurological Sciences, 239*, 210-212.

Robins, L. N., & Regier, D. A. (1991). *Psychiatric Disorders in America: The Epidemiologic Catchment Area Study*. New York: Free Press.

Rooske, O., & Birchwood, M. (1998). Loss, humiliation and entrapment as appraisals of schizophrenic illness: a prospective study of depressed and non-depressed patients. *British Journal of Clinical Psychology, 37*, 259-268.

Rosen, K., & Garety, P. (2005). Predicting recovery from schizophrenia: a retrospective comparison of characteristics at onset of people with single and multiple episodes. *Schizophrenia Bulletin, 31*, 735-750.

Rosenberg, M. (1965). *Society and the Adolescent Self Image*. Princeton: Princeton University Press.

Ross, C. A., Anderson, G., & Clark, P. (1994). Childhood abuse and the positive symptoms of schizophrenia. *Hospital and Community Psychiatry, 45*, 489-491.

Rust, J. (1987). The Rust Inventory of Schizoid Cognitions (RISC): a psychometric measure of psychoticism in the normal population. *British Journal of Clinical Psychology, 26*, 151-152.

Rust, J. (1988). The Rust Inventory of Schizotypal Cognitions (RISC). *Schizophrenia Bulletin, 14*, 317-322.

Sacks, M. H., Carpenter, W. T. J., & Strauss, J. S. (1974). Recovery from delusions: three phases documented by patients' interpretations of research procedures. *Archives of General Psychiatry, 30*, 117-120.

Sahakian, B. J., & Owen, A. M. (1992). Computerised assessment in neuropsychiatry using CANTAB: discussion paper. *Journal of the Royal Society of Medicine, 85*, 399-402.

Salkovskis, P. M. (1991). The importance of behaviour in the maintenance of anxiety and panic: a cognitive account. *Behavioural Psychotherapy, 19*, 6-19.

Santor, D. A., Ascher-Svanum, H., Lindenmayer, J. P., & Obenchain, R. L. (2007). Item response analysis of the Positive and Negative Syndrome Scale. *BMC Psychiatry, 7*, 66.

Sartorious, N., Jablensky, A., & Shapiro, R. (1977). Two-year follow-up of the patients included in the WHO International Pilot Study of Schizophrenia: preliminary communication. *Psychological Medicine, 7*, 529-541.

Schenkel, L. S., Spaulding, W. D., DiLillo, D., & Silverstein, S. M. (2005). Histories of childhood maltreatment in schizophrenia: relationships with premorbid functioning, symptomatology, and cognitive deficits. *Schizophrenia Research, 76*, 273-286.

Schmidt, M., Blanz, B., Dippe, A., Koppe, T., & Lay, B. (1995). Course of patients diagnosed as having schizophrenia during first episode occurring under age 18 years. *European Archives of Psychiatry and Clinical Neuroscience, 245*, 93-100.

Schneider, K. (1959). *Clinical Psychopathology*. New York: Grune & Stratton.

Schultz, W. (1999). The reward signal of midbrain dopamine neurons. *News in Physiological Sciences, 14*, 249-255.

Seeman, P. (1987). Dopamine receptors and the dopamine hypothesis of schizophrenia. *Synapse, 1*, 133-152.

Seeman, P., & Kapur, S. (2000). Schizophrenia: more dopamine, more D2 receptors. *Proceedings of the National Academy of Sciences of the United States of America, 97*, 7673-7675.

Seeman, P., & Lee, T. (1975). Antipsychotic drugs: direct correlation between clinical potency and presynaptic action on dopamine neurons. *Science, 188*, 1217-1219.

Selten, J. P., Gernaat, H. B., Nolen, W. A., Wiersma, D., & van den Bosch, R. J. (1998). Experience of negative symptoms: comparison of schizophrenic patients to patients with a depressive disorder and to normal subjects. *American Journal of Psychiatry, 155*, 350-354.

Shaner, A., & Eth, S. (1989). Can schizophrenia cause posttraumatic stress disorder? *American Journal of Psychotherapy, 43*, 588-597.

Shean, G., & Wais, A. (2000). Interpersonal behaviour and schizotypy. *Journal of Nervous and Mental Disease, 188*, 842-846.

Shepherd, G., Boardman, J., & Slade, M. (2008). *Making Recovery a Reality. Policy Paper*. London: The Sainsbury Centre for Mental Health.

Shepherd, M. (1987). Formulation of new research strategies on schizophrenia. In H. Häfner, W. Gattaz & W. Janzarik (Eds.), *Search for the Causes of Schizophrenia* (pp. 75-87). Heidelberg: Springer.

Shepherd, M., Watt, D., Falloon, I. R. H., & Smeeton, N. (1989). *The Natural History of Schizophrenia: A Five-Year Follow-up Study of Outcome and Prediction in a Representative Sample of Schizophrenics*. Cambridge, UK: Cambridge University Press.

Short, S. (2006). *Review of the UK 2000 Time Use Survey*. London: Office for National Statistics.

Shute, C. C. D. (1979). *The McCollough Effect: An Indicator of Central Neurotransmitter Activity*. Cambridge: Cambridge University Press.

Silverstone, P. H. (1991). Low self-esteem in different psychiatric conditions. *British Journal of Clinical Psychology, 30*, 185-188.

Sims, A. (2002). *Symptoms in the Mind: An Introduction to Descriptive Psychopathology* (3rd ed.). Edinburgh: Elsevier Science Ltd.

Siris, S. G. (1995). Depression and schizophrenia. In S. R. Hirsch & D. R. Weinberger (Eds.), *Schizophrenia* (pp. 128-145). Oxford: Blackwell.

Skosnik, P. D., Spatz-Glenn, L., & Park, S. (2001). Cannabis use is associated with schizotypy and attentional disinhibition. *Schizophrenia Research, 48*, 83-92.

Skowbo, D., Timney, B. N., Gentry, T. A., & Morant, R. B. (1975). McCollough effects: experimental findings and theoretical accounts. *Psychological Bulletin, 82*, 497-510.

Slade, P. D. (1976). An investigation of psychological factors involved in the predisposition to auditory hallucinations. *Psychological Medicine, 6*, 123-132.

Slade, P. D., & Bentall, R. (1988). *Sensory Deception: Towards a Scientific Analysis of Hallucinations*. London: Croom Helm.

Smári, J., Stefánsson, S., & Thorgilsson, H. (1994). Paranoia, self-consciousness and social cognition in schizophrenics. *Cognitive Therapy and Research, 18*, 387-399.

Smith, B., Fowler, D., Freeman, D., Bebbington, P., Bashforth, H., Garety, P., et al. (2006). Emotion and psychosis: links between depression, self-esteem, negative schematic beliefs and delusions and hallucinations. *Schizophrenia Research, 86*, 181-188.

Soanes, C., & Stevenson, A. (Eds.). (2005). *Oxford Dictionary of English*. Oxford: Oxford University Press.

Spataro, J., Mullen, P. E., Burgess, P. M., Wells, D. L., & Moss, S. A. (2004). Impact of child sexual abuse on mental health: prospective study in males and females. *British Journal of Psychiatry, 184*, 416-421.

Spauwen, J., Krabbendam, L., Lieb, R., Wittchen, H. U., & van Os, J. (2003). Sex differences in psychosis: normal or pathological? *Schizophrenia Research, 62*, 45-49.

Spauwen, J., Krabbendam, L., Lieb, R., Wittchen, H. U., & van Os, J. (2004). Does urbanicity shift the population expression of psychosis? *Journal of Psychiatric Research, 38*, 613-618.

Spauwen, J., Krabbendam, L., Lieb, R., Wittchen, H. U., & van Os, J. (2006). Impact of psychological trauma on the development of psychotic symptoms: relationship with psychosis proneness. *British Journal of Psychiatry, 188*, 527-533.

Spitzer, R. L., Endicott, J., & Gibbon, M. (1979). Crossing the border into borderline personality and borderline schizophrenia. *Archives of General Psychiatry, 36*, 17-24.

Spitzer, R. L., Williams, J. B. W., & Gibbon, M. (1987). *Structured Clinical Interview for DSM-III-R Personality Disorders: SCID-II*. New York: New York State Psychiatric Institute.

SPSS. (2005). *SPSS Base 14.0 User's Guide*. Chicago, IL: SPSS Inc.

Steel, C., Fowler, D., & Holmes, E. A. (2005). Trauma-related intrusions and psychosis: an information processing account. *Behavioural and Cognitive Psychotherapy, 33*, 139-152.

Steel, C., Hemsley, D., & Pickering, A. D. (2002). Distractor cueing effects on choice reaction time and their relationship with schizotypal personality. *British Journal of Clinical Psychology, 41*, 143-156.

Steel, C., Mahmood, M., & Holmes, E. A. (2008). Positive schizotypy and trait dissociation as vulnerability factors for post-traumatic stress. *British Journal of Clinical Psychology, 47*, 245-249.

Stefanis, N. C., Hanssen, M., Smirnis, N. K., Avramopoulos, D. A., Evdokimidis, I. K., Stefanis, C. N., et al. (2002). Evidence that three dimensions of psychosis have a distribution in the general population. *Psychological Medicine, 32*, 347-358.

Strakowski, S. M., Keck, P. E., McElroy, S. L., West, S. A., Sax, K. W., Hawkins, J. M., et al. (1998). Twelve-month outcome after a first hospitalisation for affective psychosis. *Archives of General Psychiatry, 55*, 49-55.

Strauss, J. S. (1969). Hallucinations and delusions as points on continua function. *Archives of General Psychiatry, 21*, 581-586.

Strauss, J. S. (1989). Subjective experiences of schizophrenia: Toward a new dynamic psychiatry, II. *Schizophrenia Bulletin, 15*, 179-187.

Strauss, J. S., & Carpenter, W. T. (1977). Prediction of outcome in schizophrenia. III: Five year outcome and its predictors. *Archives of General Psychiatry, 34*, 159-163.

Strauss, J. S., Carpenter, W. T., & Bartko, J. (1974). The diagnosis and understanding of schizophrenia, part III. Speculations on the processes that underlie schizophrenic symptoms and signs. *Schizophrenia Bulletin, 11*, 61-69.

Streiner, D. L., & Norman, G. R. (2003). *Health Measurement Scales: A Practical Guide to their Development and Use. 3rd edition.* Oxford: Oxford University Press.

Subotnik, K. L., & Nuechterlein, K. H. (1988). Prodromal signs and symptoms of schizophrenic relapse. *Journal of Abnormal Psychology, 97*, 405-412.

Suhr, J. A., & Spitznagel, M. B. (2001). Factor versus cluster models of schizotypal traits. I: A comparison of unselected and highly schizotypal samples. *Schizophrenia Research, 52*, 231-239.

Tabachnick, B. G., & Fidell, L. S. (2001). *Using Multivariate Statistics*. Boston: Allyn and Bacon.

Tait, A., McNay, L., Gumley, A., & O'Grady, M. (2002). The development and implementation of an individualised early signs monitoring system in the prediction of relapse in schizophrenia. *Journal of Mental Health, 11*, 141-153.

Tait, L., Birchwood, M., & Trower, P. (2004). Adapting to the challenge of psychosis: personal resilience and the use of sealing-over (avoidant) coping strategies. *British Journal of Psychiatry, 185*, 410-415.

Tarrier, N., Barrowclough, C., & Bamrah, J. S. (1991). Prodromal signs of relapse in schizophrenia. *Social Psychiatry and Psychiatric Epidemiology, 26*, 157-161.

Tarrier, N., Khan, S., Cater, J., & Picken, A. (2007). The subjective consequences of suffering a first episode psychosis: trauma and suicide behaviour. *Social Psychiatry and Psychiatric Epidemiology, 42*, 29-35.

Tauscher-Wisniewski, S., & Zipursky, R. B. (2002). The role of maintenance pharmacotherapy in achieving recovery from a first episode of schizophrenia. *International Review of Psychiatry, 14*, 284-292.

Tek, C., Kirkpatrick, B., & Buchanan, R. W. (2001). A five-year follow-up study of deficit and nondeficit schizophrenia. *Schizophrenia Research, 49*, 253-260.

Ten Have, T. R., Joffe, M. M., Lynch, K. G., Brown, G., Maisto, S. A., & Beck, A. T. (2007). Causal mediation analyses with rank preserving models. *Biometrics, 63*, 926-934.

Thewissen, V., Bentall, R. P., Lecomte, T., van Os, J., & Myin-Germeys, I. (2008). Fluctuations in self-esteem and paranoia in the context of daily life. *Journal of Abnormal Psychology, 117*, 143-153.

Thewissen, V., Myin-Germeys, I., Bentall, R., de Graaf, R., Vollebergh, W., & van Os, J. (2007). Instability in self-esteem and paranoia in a general population sample. *Social Psychiatry and Psychiatric Epidemiology, 42*, 1-5.

Thiemann, S., Csernansky, J. G., & Berger, P. A. (1987). Rating scales in research: the case of negative symptoms. *Psychiatry Research, 20*, 47-55.

Thompson, P., & Hodgekins, J. (2004). After-effects and the schizotypal personality. *Perception, 33*(suppl.), S38.

Thornicroft, G., Bisoffi, G., de Salva, D., & Tansella, M. (1993). Urban-rural differences in the associations between social deprivation and psychiatric service utilization in schizophrenia and all diagnoses: a case-register study in northern Italy. *Psychological Medicine, 23*, 487-496.

Tien, A. Y. (1991). Distributions of hallucinations in the population. *Social Psychiatry and Psychiatric Epidemiology, 26*, 287-292.

Tien, A. Y., Costa, P. T., & Eaton, W. W. (1992). Covariance of personality, neurocognition, and schizophrenia spectrum traits in the community. *Schizophrenia Research, 7*, 149-158.

Tien, A. Y., & Eaton, W. W. (1992). Psychopathologic precursors and sociodemographic risk factors for the schizophrenia syndrome. *Archives of General Psychiatry, 49*, 37-46.

Tohen, M., Strakowski, S. M., Zarate, C., Hennen, J., Stoll, A. L., & Suppes, T. (2000). The McLean-Harvard first episode project: 6-month symptomatic and functional outcome in affective and non-affective psychosis. *Biological Psychiatry, 48*, 467-476.

Torgersen, S. (1985). Relationship of schizotypal personality disorder to schizophrenia: Genetics. *Schizophrenia Bulletin, 11*, 554-563.

Torgersen, S., Kringlen, E., & Cramer, V. (2001). The prevalence of personality disorders in a community sample. *Archives of General Psychiatry, 58*, 590-596.

Trower, P., & Chadwick, P. (1995). Pathways to defense of the self: a theory of two types of paranoia. *Clinical Psychology: Science and Practice, 2*, 263-278.

Trower, P., & Gilbert, P. (1989). New theoretical conceptions of social anxiety and social phobia. *Clinical Psychology Review, 9*, 19-35.

Tsai, S. M., Chen, C., Kuo, C., Lee, J., Lee, H., & Strakowski, S. M. (2001). 15 year outcome of treated bipolar affective disorder. *Journal of Affective Disorders, 63*, 215-220.

Tsuang, M. T., Stone, W. S., Tarbox, S. I., & Faraone, S. V. (2002). An integration of schizophrenia with schizotypy: identification of schizotaxia and implications for research on treatment and prevention. *Schizophrenia Research, 54*, 169-175.

Turnbull, G., & Bebbington, P. (2001). Anxiety and the schizophrenic process: clinical and epidemiological evidence. *Social Psychiatry and Psychiatric Epidemiology, 36*, 235-243.

Turner-Crowson, J., & Wallcraft, J. (2002). The recovery vision for mental health services and research: a British perspective. *Psychiatric Rehabilitation Journal, 25*, 245-255.

Unzicker, R. (1989). On my own: a personal journey through madness and re-emergence. *Psychosocial Rehabilitation Journal, 13*(1), 71-77.

van Os, J. (2003). Is there a continuum of psychotic experiences in the general population? *Epidemiol e Psichiatria Sociale, 12*, 242-252.

van Os, J., Gilvarry, C. M., Bale, R., van Horn, W., Tattan, T., White, I., et al. (1999). A comparison of the utility of dimensional and categorical representations of psychosis. *Psychological Medicine, 29*, 595-606.

van Os, J., Hanssen, M., Bijl, R., & Ravelli, A. (2000). Strauss (1969) revisited: a psychosis continuum in the general population? *Schizophrenia Research, 45*, 11-20.

van Os, J., Jones, P., Sham, P., Bebbington, P., & Murray, R. M. (1998). Risk factors for onset and persistence of psychosis. *Social Psychiatry and Psychiatric Epidemiology, 33*, 596-605.

van Os, J., Linscott, R. J., Myin-Germeys, I., Delespaul, P., & Krabbendam, L. (2009). A systematic review and meta-analysis of the psychosis continuum: evidence for

a psychosis-proneness-persistence-impairment model of psychotic disorder. *Psychological Medicine, 39*, 179-195.

van Os, J., & Sham, P. (2003). Gene-environment correlation and interaction in schizophrenia. In R. M. Murray, P. B. Jones, E. Susser, J. van Os & M. Cannon (Eds.), *The Epidemiology of Schizophrenia* (pp. 235-253). Cambridge: Cambridge University Press.

van Os, J., Verdoux, H., Maurice-Tison, S., Gay, B., Liraud, F., Salamon, R., et al. (1999). Self-reported psychosis-like symptoms and the continuum of psychosis. *Social Psychiatry and Psychiatric Epidemiology, 34*, 459-463.

Venables, P. H. (1984). Cerebral mechanisms automatic responsiveness and attention in schizophrenia. In W. D. Spaulding & J. K. Cole (Eds.), *Theories of Schizophrenia and Psychosis* (pp. 47-91). Lincoln: University of Nebraska Press.

Venables, P. H. (1995). Schizotypal personality as a developmental stage in studies of risk for schizophrenia. In A. Raine, T. Lencz & S. A. Mednick (Eds.), *Schizotypal Personality* (pp. 107-131). Cambridge: Cambridge University Press.

Venables, P. H., & Bailes, K. (1994). The structure of schizotypy, its relation to subdiagnoses of schizophrenia and to sex and age. *British Journal of Clinical Psychology, 33*, 277-294.

Venables, P. H., & Rector, N. A. (2000). The content and structure of schizotypy: a study using confirmatory factor analysis. *Schizophrenia Bulletin, 26*, 587-602.

Venables, P. H., Wilkins, S., Mitchell, D., Raine, A., & Bailes, K. (1990). A scale for the measurement of schizotypy. *Personality and Individual Differences, 11*, 481-495.

Ventura, J., Nuechterlein, K. H., Lukoff, D., & Hardesty, J. P. (1989). A prospective study of stressful life events and schizophrenic relapse. *Journal of Abnormal Psychology, 98*, 407-411.

Verdoux, H., Maurice-Tison, S., Gay, B., Van Os, J., Salamon, R., & Bourgeois, M. (1998). A survey of delusional ideation in primary care patients. *Psychological Medicine, 28*, 127-134.

Verdoux, H., & van Os, J. (2002). Psychotic symptoms in non-clinical populations and the continuum of psychosis. *Schizophrenia Research, 54*, 59-65.

Verdoux, H., van Os, J., Maurice-Tison, S., Gay, B., Salamon, R., & Bourgeois, M. (1999). Increased occurrence of depression in psychosis-prone subjects: a follow-up study in primary care settings. *Comprehensive Psychiatry, 40*, 462-468.

Voges, M., & Addington, J. (2005). The association between social anxiety and social functioning in first episode psychosis. *Schizophrenia Research, 76*, 287-292.

Vohs, J. L., Hetrick, W. P., Kieffaber, P. D., Bodkins, M., Bismark, A., Shekhar, A., et al. (2008). Visual event-related potentials in schizotypal personality disorder and schizophrenia. *Journal of Abnormal Psychology, 117*, 119-131.

Vollema, M. G., & Hoijtink, H. (2000). The multidimensionality of self-report schizotypy in a psychiatric population: an analysis using multidimensional Rasch models. *Schizophrenia Bulletin, 26*, 565-575.

Vollema, M. G., Sitskoorn, M. M., Appels, M. C. M., & Kahn, R. S. (2002). Does the Schizotypal Personality Questionnaire reflect the biological-genetic vulnerability to schizophrenia? *Schizophrenia Research, 54*, 39-45.

Vollema, M. G., & van den Bosch, R. J. (1995). The multidimensionality of schizotypy. *Schizophrenia Bulletin, 21*, 19-31.

Waddell, G., & Burton, A. (2006). *Is work good for your health and well-being?* London: TSO.

Walden, J., & Grunze, H. (2004). *Bipolar Affective Disorder: Aetiology and Treatment*. New York: Thieme Medical Publishers.

Wallace, C. J., Liberman, R. P., Tauber, R., & Wallace, J. (2000). The Independent Living Skills Survey: a comprehensive measure of the community functioning of severely and persistently mentally ill individuals. *Schizophrenia Bulletin, 26*, 631-658.

Warner, R. (1985). *Recovery from Schizophrenia: Psychiatry and Political Economy*. London: Routledge.

Weiser, M., Reichenberg, A., Rabinowitz, J., Kaplan, Z., Mark, M., Bodner, E., et al. (2001). Association between nonpsychotic psychiatric diagnoses in adolescent males and subsequent onset of schizophrenia. *Archives of General Psychiatry, 58*, 959-964.

Whitaker, R. (2004). The case against antipsychotic drugs: a 50-year record of doing more harm than good. *Medical Hypotheses, 62*, 5-13.

Whitehorn, D., Brown, J., Richard, J., Rui, Q., & Kopala, L. (2002). Multiple dimensions of recovery in early psychosis. *International Review of Psychiatry, 14*, 273-283.

Whitehorn, D., Lazier, L., & Kopala, L. (1998). Psychosocial rehabilitation early after the onset of psychosis. *Psychiatric Services, 49*, 1135-1137.

Widiger, T. A., Frances, A., & Trull, T. J. (1987). A psychometric analysis of the social-interpersonal and cognitive-perceptual items for schizotypal personality disorder. *Archives of General Psychiatry, 44*, 741-745.

Wiersma, D., Nienhuis, F. J., Slooff, C. J., & Giel, R. (1998). Natural course of schizophrenic disorders: a 15-year followup of a Dutch incidence cohort. *Schizophrenia Bulletin, 24*, 75-85.

Wiles, N. J., Zammit, S., Bebbington, P., Singleton, N., Meltzer, H., & Lewis, G. (2006). Self-reported psychotic symptoms in the general population: results from the longitudinal study of the British National Psychiatric Morbidity Survey. *British Journal of Psychiatry, 188*, 519-526.

Wing, J. K., Babor, T., Brugha, T., Burke, J., Cooper, J. E., Giel, R., et al. (1990). SCAN: Schedules for Clinical Assessment in Neuropsychiatry. *Archives of General Psychiatry, 47*, 589-593.

Wing, J. K., & Brown, G. (1970). *Institutionalism and schizophrenia: a comparative study of three mental health hospitals 1960-1968*. London: Cambridge University Press.

Wing, J. K., Cooper, J. E., & Sartorius, N. (1974). *The Measurement and Classification of Psychiatric Symptoms*. Cambridge: Cambridge University Press.

Winkelman, N. W., Jr. (1954). Chlorpromazine in the treatment of neuropsychiatric disorders. *Journal of the American Medical Association, 155*, 18-21.

Woods, S. W. (2000). The economic burden of bipolar disease. *Journal of Clinical Psychiatry, 61*, 38-41.

Woods, S. W. (2003). Chlorpromazine equivalent doses for the newer atypical antipsychotics. *Journal of Clinical Psychiatry, 64*, 663-667.

World Health Organisation. (1990). *Composite International Diagnostic Interview (CIDI), Version 1.0*. Geneva: World Health Organisation.

Wykes, T., Reeder, C., Landau, S., Everitt, B., Knapp, M., Patel, A., et al. (2007). Cognitive remediation therapy in schizophrenia: randomised controlled trial. *British Journal of Psychiatry, 190*, 421-427.

Wykes, T., Steel, C., Everitt, B., & Tarrier, N. (2008). Cognitive Behavior Therapy for schizophrenia: effect sizes, clinical models, and methodological rigor. *Schizophrenia Bulletin, 34*, 523-537.

Young, J. E. (1998). The Young Schema Questionnaire: short form. Available in electronic form at: http://home.sprynet/schema/ysqsl.htm.

Yue, C., Bidwell, L. C., & Norton, D. (2006). Trait vs. state markers for schizophrenia: Identification and characterisation through visual processes. *Current Psychiatry Reviews, 2*, 431-438.

Yung, A. R., Buckby, J. A., Cotton, S. M., Cosgrave, E. M., Killackey, E. J., Stanford, C., et al. (2006). Psychotic-like experiences in non-psychotic help-seekers: associations with distress, depression and disability. *Schizophrenia Bulletin, 32*, 352-359.

Yung, A. R., & McGorry, P. D. (1996). The initial prodrome in psychosis: descriptive and qualitative aspects. *Australian and New Zealand Journal of Psychiatry, 30*, 587-599.

Yung, A. R., Phillips, L., McGorry, P., Ward, J., Donovan, K., & Thompson, K. (2002). *Comprehensive assessment of at risk mental state (CAARMS)*. Melbourne: PACE Clinic, Department of Psychiatry, University of Melbourne.

Zubin, J., & Spring, B. (1977). Vulnerability - a new view of schizophrenia. *Journal of Abnormal Psychology, 86*, 103-126.

Zuckerman, M. (1989). Personality in the third dimension: a psychobiological approach. *Personality and Individual Differences, 10*, 391-418.

APPENDICES

APPENDIX A

Web Study Information

A1 Circular e-mail for web study recruitment

This e-mail was sent to all students at the University of East Anglia and King's College London.

A2 Web study information sheet

This information sheet was displayed on the first page of the website. Participants were asked to endorse a tick box after reading the information sheet if they agreed to take part in the study.

Hodgekins Joanne Ms (MED)

From: Hodgekins Joanne Ms (MED) **Sent:** Mon 01/09/2008 17:51
To: 'Hodgekins Joanne Ms (MED)'
Cc:
Subject: Web-based study investigating the influence of emotion on personality characteristics - circular
Attachments:

You are invited to take part in a student project investigating how different aspects of emotion are associated with characteristics of personality. The study is also interested in how traumatic events during an individual's lifetime can influence their later experiences and views.

You are under no obligation to reply to this email, however if you choose to, participation in this research is voluntary and you may withdraw at any time.

The study involves completing six questionnaires on a web-page we have set up (link below) and will take about 30 minutes. The first questionnaire lists unusual experiences and asks whether you have experienced them and if so, how often. The questionnaire also asks about your feelings in certain situations and about your views on certain issues including clairvoyancy and telepathy. The other questionnaires ask about: your emotions and how you have been feeling recently; the key beliefs that you hold about yourself and others; how you feel that other people think of you in social situations; how you feel about yourself and other people in your life. The final questionnaire asks about upsetting experiences that you may have encountered at any point in your life.

To read the information sheet about the study and to complete the questionnaires, please go to the web-page we have set up:

http://web1.iop.kcl.ac.uk/IoPDepts/EmotionAndPerson.nsf/Welcome?OpenPage

Please contact Joanne Hodgekins if you would like any further information.
(Joanne Hodgekins, Research Associate, School of Medicine, Health Policy and Practice, University of East Anglia, Norwich, NR4 7TJ. Tel: 01603 591232, e-mail: j.hodgekins@uea.ac.uk)

Thank you very much for your help.

Further Information:

The study has been reviewed and approved by the Institute of Health Ethics Committee at the University of East Anglia and the Institute of

Psychiatry
Ethical Committee (Research). It is not expected that participation in the
study has any risks or will cause any ill effects. However, you can stop
completing the questionnaires at any point if you feel upset by the survey.
If you continue to feel upset, please contact one of the research team members listed below. The UEA Health Centre (uhs@uea.ac.uk) and Dean of Students (dos@uea.ac.uk) are also aware of this research and have a variety
of services available should you wish to use them. More information about
these services can be found through the 'Student Links' section of the UEA
homepage.

Research Team Members:

Joanne Hodgekins, Research Associate, School of Medicine, Health Policy and
Practice, University of East Anglia, Norwich, NR4 7TJ. (Tel: 01603 591232,
e-mail: j.hodgekins@uea.ac.uk)

Prof. David Fowler, Professor of Social Psychiatry, School of Medicine, Health Policy and Practice, University of East Anglia, Norwich, NR4 7TJ. (Tel: 01603 593601, e-mail: d.fowler@uea.ac.uk)

Dr Daniel Freeman, Lecturer in Clinical Psychology, Department of Psychology, Institute of Psychiatry, Denmark Hill, London, SE5 8AF. (Tel:
020 7848 5003, e-mail: d.freeman@iop.kcl.ac.uk)

Prof. Philippa Garety, Professor of Clinical Psychology. Based at the Department of Psychology, Institute of Psychiatry, Denmark Hill, London, SE5 8AF.

Information Sheet
Student Survey: The Influence of Emotion on Personality Characteristics

You are invited to take part in a student project investigating how different aspects of emotion are associated with characteristics of personality. The study is also interested in how traumatic events in childhood can affect experiences and views in later life.

Who we would like to take part:
The study is open to everyone. In order to understand the influence of emotion on personality, we need to look at the views, feelings and experiences of all individuals, even if you have not experienced any of the items asked about. Participation is voluntary and you can withdraw from the study at any time if you do not wish to continue.

What the study involves:
The study involves filling in six questionnaires on the internet and will take about 30 minutes.

Anonymity and confidentiality:
The questionnaires are filled in on a web-page which has in-built security. Each participant is allocated a number and we do not record your name. Thus all responses to the questionnaires are completely anonymous and cannot be traced back to the participant's email address. Any information provided is also completely confidential. The information collected in the study will be used only for research and publication in research papers.

What you will be asked in the questionnaires:

Questionnaire 1: Lists unusual experiences and asks whether you have experienced them and if so, how often. The questionnaire also asks about your feelings in certain situations and about your views on certain issues including clairvoyancy and telepathy.

Questionnaire 2: Asks about your emotions and how you have been feeling recently.

Questionnaire 3: Asks about the key beliefs you hold about yourself and others.

Questionnaire 4: Asks about how you feel that other people think of you in social situations.

Questionnaire 5: Lists statements relating to how you may feel about yourself and other people in your life. You will be asked to state how much each item applies to you.

Questionnaire 6: Asks about some of your experiences growing up as a child and a teenager.

Ethical considerations:
The study has been reviewed by the local research ethics committee. It is not expected that participation in the study has any risks or will cause any ill effects. However you can stop completing the questionnaires at any point if you feel upset by the survey. If you continue to feel upset, please contact one of the research team members listed below.

Thank you very much for your help.

For further information, please contact Joanne Hodgekins (Tel: 01603 593584)

Research Team Members

- **University of East Anglia, Norwich**

David Fowler, Senior Lecturer in Clinical Psychology, School of Medicine, Health Policy and Practice, University of East Anglia, Norwich, NR4 7TJ. (Tel: 01603 593601; e-mail: d.fowler@uea.ac.uk)

Joanne Hodgekins, Research Associate, School of Medicine, Health Policy and Practice, University of East Anglia, Norwich, NR4 7TJ. (Tel: 01603 593898; e-mail: j.hodgekins@uea.ac.uk)

- **King's College London**

Dr Daniel Freeman, Lecturer in Clinical Psychology, Department of Psychology, Institute of Psychiatry, Denmark Hill, London, SE5 8AF. (Tel: 0207 848 5003; e-mail: d.freeman@iop.kcl.ac.uk)

Professor Philippa Garety, Professor of Clinical Psychology. Based at the Department of Psychology, Institute of Psychiatry, Denmark Hill, London, SE5 8AF. (Tel: 020 7848 5046; e-mail: p.garety@iop.kcl.ac.uk)

APPENDIX B

Trial outcome paper for ISREP study
(manuscript submitted to Psychological Medicine)

Cognitive behaviour therapy for improving social recovery in psychosis: a report from the ISREP MRC Trial Platform study (Improving Social Recovery in Early Psychosis)

D. Fowler[1†], J. Hodgekins[1], M. Painter[2], T. Reilly[3], C. Crane[2,4], I. Macmillan[3], M. Mugford[1], T. Croudace[4], P.B. Jones[2,4]

[1]School of Medicine, Health Policy and Practice, University of East Anglia, Norwich, UK

[2]CAMEO, Cambridgeshire and Peterborough Mental Health Partnership NHS Trust

[3]Norfolk Early Intervention Service, Norfolk and Waveney Mental Health Partnership NHS Trust

[4]Department of Psychiatry, University of Cambridge,, Cambridge, UK

Word count: Abstract 248, Text 4508

[†] Author for correspondence:
Professor David Fowler, School of Medicine, Health Policy & Practice, Elizabeth Fry Building, University of East Anglia, Norwich UK, NR4 7TJ
Email: d.fowler@uea.ac.uk
Tel: 44 1603 593 601 Fax: 44 1603 593 604

ABSTRACT

Background

This study reports on a preliminary evaluation of a cognitive behavioural intervention to improve social recovery in psychosis. The study was a single-blind RCT with two arms, 35 participants receiving CBT plus TAU, and 42 participants receiving TAU alone. Participants were assessed at baseline and post-treatment.

Method

Seventy-seven participants were recruited from secondary mental health teams after presenting with a history of unemployment and poor social outcome. The cognitive behavioural intervention was delivered over a nine-month period with a mean of 12 sessions. The primary outcomes were weekly hours spent in constructive economic and structured activity. A range of secondary and tertiary outcomes were also assessed.

Results

Intention to treat analysis on the combined affective and non-affective psychosis sample showed no significant impact of treatment on primary or secondary outcomes. Analysis of interactions by diagnostic subgroup approached significance for secondary symptomatic outcomes (PANSS ($F(1,69)=3.99$, $p=0.05$). Exploratory analyses within diagnostic subgroups revealed significant improvements in weekly hours in constructive and structured activity, PANSS scores, and beliefs about self and others amongst people with non-affective psychosis. In the affective psychosis group there were striking improvements in activity and symptoms but these occurred in both treatment and control groups.

Conclusion

The primary study comparison provided no clear evidence for the benefit of CBT in on a combined sample of patients. However, secondary analyses showed benefits for CBT amongst people with non-affective psychosis who have social recovery problems. These promising results need to be independently replicated in a larger, *multi-centre* RCT.

INTRODUCTION

Poor social outcome is often reported in psychosis. Long term follow-up studies suggest that less than 50% of people with non-affective psychosis achieve a social recovery, and only 10-20% of people return to competitive employment (Johnstone *et al.*, 1990, Jablensky *et al.*, 1992, Harrison *et al.*, 1996), despite the majority suggesting that they wish to work (Mueser *et al.*, 2001). Around 50% of people with severe affective psychosis also fail to return to work and remain disabled (Tsai *et al.*, 2001). Long term follow-up studies indicate that poor social outcomes in psychosis tend to emerge early, often become stable, and are closely associated with long term social course (Strauss and Carpenter, 1977, Carpenter and Strauss, 1991). The development of an effective intervention to improve social recovery in affective and non-affective psychosis could potentially have important long term benefits, especially if applied to cases who have developed poor social functioning in the early course of the disorder.

Effective interventions to improve psychosocial recovery in psychosis may need to consider factors associated with impairments in a sophisticated manner. These effects may include residual psychotic symptoms, sensitivity to stress and underlying cognitive deficits. In particular, care needs to be taken not to over stimulate. Past clinical trials of interventions which have attempted to promote social activity, without taking careful account of sensitivity to psychosis and anxiety, have shown increased risk of relapse, especially amongst people who still show psychotic symptoms (Hogarty *et al.*, 1974, Hogarty *et al.*, 1997). Cognitive behaviour therapy (CBT) may provide a useful basis for developing such an intervention. Several recent studies have reported evidence for the efficacy of CBT on depression and negative symptoms where these have been assessed as secondary outcomes (Sensky *et al.*, 2000, Turkington *et al.*, 2002, Durham *et al.*, 2003, Gumley *et al.*, 2003, Wykes *et al.*, 2007). However, these trials used relatively insensitive measures of social functioning and no trial to date has directly targeted changes in social recovery as primary outcome. An optimal intervention for people with psychosis who wish to work but have some degree of residual problems may be for therapists to combine techniques of cognitive behaviour therapy with those of vocational case management (Mueser *et al.*, 2001).

This study used a Trial Platform to evaluate the implementation feasibility and initial efficacy of a psychosocial intervention to improve social recovery in psychosis. The intervention was specifically focused on improving constructive social behaviour while managing sensitivity to stress, social anxiety, and psychotic symptoms. Social recovery is a complex construct probably best assessed across a number of domains. While engagement in full-time competitive work will always represent a key marker of social recovery (Mueser *et al.*, 2001), it is not the only marker of social improvement. Engagement in other domains of activity such as education, household chores, constructive voluntary work and structured social activities, reflect realistic and meaningful recovery goals for many service users and carers and also have wider economic benefits. In this study we therefore used time spent engaged in structured social and constructive economic activity as our primary measure of outcome. We were also interested in assessing the impact of the intervention on a range of secondary and tertiary outcomes including hopelessness, psychotic symptoms, depression and anxiety, illness cognitions, and beliefs about self and others. These reflect common psychological responses to the experience of psychosis and associated social adversity, which are important in their own right (Birchwood, 2003), but which also have important associations with symptomatic outcomes and withdrawn and amotivated social behaviour (Fowler *et al.*, 2006). We also planned to explore as a secondary hypothesis, the possibility of a differential effect of CBT on affective versus non-affective psychosis.

METHOD

Design

The ISREP study was a single blind randomised controlled treatment trial comparing cases who received SRCBT in addition to treatment as usual (treatment arm) with those receiving treatment as usual alone (control arm). Participants were randomised to CBT or control following a baseline assessment and initial screening for suitability. Randomisation was stratified for diagnosis (affective/non-affective psychosis was considered a prognostic factor) and administrative centre (Norfolk/Cambridgeshire). Post-treatment

assessments were conducted at the end of the intervention phase (nine months following randomisation). The primary outcomes were weekly hours spent in constructive economic activity and structured activity. Secondary outcomes included symptoms, anxiety, depression, hopelessness, and schema. Baseline and post-treatment assessments were conducted by research assistants who were blind to group allocation.

Participants

Inclusion criteria were:

- Current diagnosis of affective or non-affective psychosis (including schizophrenia, schizoaffective disorder, bipolar disorder, and psychotic depression).
- Illness duration of 8 years or less. Onset of illness was defined as the first contact with psychiatric services for psychotic symptoms. This was checked by research assistants from information in case notes.
- Positive psychotic symptoms (hallucinations and delusions) in relative remission (less than moderate severity, scoring 4 or less, on individual symptoms on the PANSS).
- Unemployed status or currently engaged in less than 16 hours paid employment or education.

Participants were excluded if:

- the psychotic disorder was thought to have an organic basis
- acute psychosis was present
- the primary diagnosis was drug dependency on opiates or cocaine

The study protocol was approved by local ethics committees and all participants gave written consent to participate following a formal explanation of the study.

Participant flow and characteristics

Participants were recruited from secondary mental health services in the East Anglia region of the UK, localised around two sites. The site based in Norfolk (Centre 1) recruited from cases in the Norfolk and Waveney Mental Health Partnership. A site based in Cambridgeshire (Centre 2) recruited from cases in two mental health trusts: Cambridgeshire and Peterborough Mental Health Partnership, and West Suffolk Hospital NHS Trust. Together the two centres

recruited from a catchment area with a semi-rural population of around two million people, living in small cities, towns and rural areas.

The CONSORT flow diagram in Figure 1 shows the initial referral rate, allocation by centre and diagnosis, and the level of drop out from the main outcome assessment. A total of 200 suitable participants were identified of which 77 participants consented to participate were recruited into the study. The average age was 29 (range = 18-52). Participants had been in contact with services for an average of five years, and average length of unemployment was 209 weeks. Fifty-five participants were male (71%). The majority of the sample had a diagnosis of non-affective psychosis (65%).

Thirty-five participants were randomised to the treatment condition and 42 to treatment as usual, the control condition. Key clinical and social characteristics of the sample are summarised in Table 1. This shows that randomisation resulted in balanced groups in terms of demographics, diagnosis, illness length, and social characteristics.

Treatments

Social Recovery Cognitive Behavioural Therapy

Therapy consisted of three stages and combined techniques of CBT with vocational case management. Stage 1 involved developing a formulation of the person in social recovery. This consisted of assessment and history taking with respect to personal motivation, premorbid hopes/expectations and goals which had either been changed or altered with respect to the impact of illness. The focus was on identifying meaningful personal goals which could be linked with achievable day-to-day activity targets. This often involved validation and acceptance of real barriers, threats and difficulties, while focusing on promoting hope for social recovery.

Stage Two involved identifying and working towards medium to long term goals. A particularly important aspect of this was identifying specific pathways to meaningful new activities. Where relevant this included referral to relevant vocational agencies, or alternatively direct liaison with employers or education providers. Cognitive work at this stage involved promoting a sense of agency and addressing feelings of stigma and negative beliefs about self and others.

Stage Three involved the active promotion of social activity, work, education and leisure linked to meaningful goals. This involved promotion of activity by behavioural experiments, while managing symptoms of anxiety and low level psychotic symptoms. Mastery and pleasure with respect to achieving goals was reviewed with respect to real gains achieved in social opportunities in work, education and leisure.

Specific therapeutic procedures used in the study were drawn from existing cognitive behaviour therapy manuals. Prominent amongst these were procedures to focus on self-regulation of psychotic symptoms and improve social recovery from psychosis (e.g. chapters 11 and 15 of Fowler *et al.*, 1995). Therapists were also encouraged to use techniques of activity scheduling and reviewing mastery and pleasure, as described in Beck *et al.* (1979); and behavioural experiment approaches to manage social anxiety, as described in Butler (1999). Therapists were also encouraged to combine therapist role with case management roles typical of individual placement and support working practices. For example, by adopting an assertive outreach worker style of contact, most frequently visiting people at home or in the workplace. Therapists were also encouraged to adopt a pragmatic and problem-solving approach in assisting people to overcome work-related problems. This often involved setting up joint interviews with clients and employment and education providers to discuss potential problems.

Therapy in Norfolk was carried out by case managers who had no previous formal training in CBT, but who had over two years experience working in an early intervention in psychosis team, under the supervision of expert CBT therapists. Therapy in the Cambridge based centre was carried out by CBT therapists who had attended approved courses prior to working on the trial. Therapy in both centres was supervised by experienced CBT specialists. Adherence and competence were monitored using tape recordings and individual and group supervision. Participants received a mean of 12 sessions (SD = 7).

Treatment as usual

Both sites provided active case management by multidisciplinary secondary care mental health teams. The services provided by Norfolk and Waveney

Mental Health Partnership Trust (Centre 1) had a pre-existing, active policy of promoting social recovery in case management. This consisted of multidisciplinary case management, and was backed by the availability of services to provide supported employment for people with severe and enduring mental health problems. Such an approach was consistently available for all cases. The Cambridgeshire site (Centre 2) also had active multidisciplinary case management, although supported employment agencies were less consistently available as part of generic services

Measures

Primary Outcome

Time Use Survey (adapted from UK 2000 Time Use Survey; Short, 2006)

This measure consists of a semi-structured interview in which the participant is asked about how they have spent their time over the last month. Activities enquired about include: work, education, voluntary work, leisure, sports, hobbies, socialising, resting, housework/chores, childcare, and sleep. Time spent on each of the activities is calculated in terms of the number of hours per week allocated to that activity over the last month. Two summary measures were derived from the Time Use Survey: hours in 'Constructive Economic Activity' and hours in 'Structured Activity'. Constructive economic activity is calculated as the sum of hours per week over the last month spent in work, education, voluntary work, housework and chores, and childcare. The constructive economic activity assessment could be undertaken by telephone contacts and triangulated with carer reports as well as from face-to-face interviews, thus maximising available data at post-treatment. Hours in 'Structured Activity' is calculated as the sum of hours per week over the last month spent in constructive economic activity, but also includes voluntary and structured leisure activities, sports, and hobbies. The structured activity assessment required a face-to-face interview with the participant.

Secondary Outcomes

Positive and Negative Syndrome Scale (PANSS; Kay et al., 1987)

The PANSS is a 30-item rating scale developed to assess symptoms associated with psychosis. Symptoms occurring over the last week are rated. PANSS total scores were used.

Brief Core Schema Scales (BCSS; Fowler et al., 2006)

The BCSS is a 24-item self-report scale designed to assess the type of extreme positive and negative evaluations of self and others that have been observed clinically to be typical of people with psychosis. Items are rated on a five point scale (0-4). Four scores are obtained: negative self (6 items), positive self (6 items), negative other (6 items) and positive other (6 items).

Beck Hopelessness Scale (BHS; Beck and Steer, 1988)

The BHS is a 20-item self-report scale designed to assess the way an individual perceives the future. Items are rated using a dichotomous true/false response format. Total scores from the BHS were used.

Schizotypal Symptoms Inventory (SSI; in preparation)

The SSI is a 20-item self-report scale designed to assess the presence and severity of low-level, residual psychotic symptoms occurring in the past two weeks. Items are rated on a five point scale (0-4). Four scores can be obtained: Total (20 items), Social Anxiety (6 items), Paranoia (6 items), and Anomalous Experiences (8 items).

Quality of Life Scale (QLS; Heinrichs et al., 1984)

The QLS is a 21-item semi-structured interview designed to assess the functional impairments associated with psychosis, including problems with interpersonal relationships and occupational role functioning. Two scores were used: total QLS score and the score on the Instrumental Role Functioning subscale (e.g. employment, accomplishment, role satisfaction).

Tertiary Assessments

Tertiary outcomes and other measures included the Beck Depression Inventory (BDI-II; Beck *et al.*, 1996); the Beck Anxiety Inventory (BAI; Beck and Steer, 1987); the Personal Beliefs about Illness Questionnaire (PBIQ; Birchwood *et al.*, 1993); the EuroQol visual analogue scale (Brooks, 1996); the Global Assessment of Symptoms Scale (GAS; American Psychiatric Association,

2000); the Social and Occupational Functioning Assessment Scale (SOFAS; Goldman *et al.*, 1992); the Client Service Receipt Inventory (CSRI; Beecham and Knapp, 1992); and the Camberwell Assessment of Needs (CAN; Slade *et al.*, 1996). All self-reports were completed independently by participants. The GAS, CAN, CSRI and SOFAS were completed with case managers where appropriate.

Reliability of Research Assessments and Blinding Procedures

Baseline and post-treatment assessments were conducted by research assistants who were independent of treatment delivery and randomisation. Every effort was made to ensure they were kept blind to allocation. Formal training in all measures was provided and interviews were audio-taped for reliability and quality control. Research assistants met regularly throughout the trial to maintain reliability of procedures and ratings. Where blindness was broken, another research assistant conducted the post-treatment assessment. Ninety-three percent of the post-treatment assessments were completed blind. The research assistants made allocation guesses after post-treatment assessments. These were 58% correct for CBT and 64% correct for TAU. This is within the levels that be expected by chance.

Statistical Analyses

Hypotheses

Primary Hypothesis: It was predicted that the provision of Social Recovery Cognitive Behaviour Therapy (SRCBT) added to case management (TAU) would improve levels of constructive economic and structured activity in comparison to cases receiving TAU alone.

Secondary Hypotheses: i) We predicted that SRCBT added to TAU would improve on secondary outcomes of symptoms of psychosis and emotional disorder, negative beliefs about self and others, and hopelessness. ii) We aimed to explore the impact of diagnosis on outcome.

Sample size and power of the study

The purpose of the study was to conduct exploratory efficacy research on a new intervention to improve social recovery in psychosis. The sample size was predicated on testing for an effect of SRCBT on activity with an effect size of around 0.6. Sample sizes with a minimum of 30 in each group would then be sufficient to detect such an effect with 90% power. Our trial platform legitimised limited investigation of some secondary hypotheses particularly regarding interactions with diagnostic group and centre. However, we understood that these would be underpowered. Secondary hypotheses were undertaken to inform the design of future research e.g. a larger, multicentre randomised controlled trial for independent replication/extension.

Analysis Plan

We first report descriptive statistics for each primary and secondary outcome at baseline and post-treatment for the combined study sample, and then the sample split by diagnosis. These estimates provide the basis for a provisional estimate of effect size, albeit biased by drop-outs and potential non-random differences at baseline.

Primary analyses and significance testing were conducted on an intention to treat basis. Following the protocol, ANCOVA models were used to test the significance of differences between the treatment and control groups. For each ANCOVA, outcome at the end of treatment (e.g. hours in structured activity at post-treatment) was used as the dependent variable; allocation to treatment, centre, and diagnosis were used as fixed factors; and three key variables assumed to be associated with outcome and predictive of drop out were used as covariates. The covariates were: baseline outcome (e.g. hours in structured activity at baseline); baseline schizotypal symptoms score; and length of unemployment. Non-significant interactions were removed before final testing for main effects. Where initial testing indicated the presence of an interaction between treatment and diagnosis, we planned to undertake a series of further ANCOVAs for each diagnostic group (affective/non-affective psychosis). These were similar to the whole group ANCOVAs but used allocation to treatment and location as fixed factors, thus allowing assessment of treatment effect independently of the diagnosis by treatment interaction.

These analyses allow for the presence of missing outcome data under the assumption that the data are missing at random (MAR) conditional on the covariates included in the regression model (i.e. allocation, schizotypal symptoms, length of unemployment, and baseline values of the outcome variables).

RESULTS

Primary outcome data (constructive economic activity) was available for 92% of the recruited sample. Eighty percent of the sample completed post-treatment face-to-face interviews, providing structured activity and secondary outcome assessments. Questionnaire assessments for secondary outcomes (e.g. BDI, BAI, BHS, BCSS) were available for around 75% of the sample. Descriptive statistics for all outcome and mediating variables are given in Table 2. These are broken down by treatment and diagnostic group at baseline and post-treatment and derive from data available at post-treatment assessment (i.e. completers).

Contacts with secondary mental health services

There were no differences in the level of support given to treated cases and controls at baseline or the number of contacts available for participants between the two sites. However, the TAU group received more contacts with secondary mental health services than the treatment group over the course of the trial (mean = 11.9, SD = 11.3 versus mean = 9.7, SD = 18.8) respectively; t=2.02, p=0.05). The difference in the mean number of contacts with voluntary services was not significant.

Outcomes for the combined group (non-affective and affective psychosis)

Table 2 shows that all participants made large improvements in most domains, including activity and symptoms, as a result of both CBT and TAU conditions. Analyses of the main effects of CBT treatment for the combined group using ANCOVAs showed significant effects on the hypothesised mediating variables of positive beliefs about self (F (1, 70) =5.1, p=0.03) and positive beliefs about others (F(1, 70)=5.61, p=0.02). There were strong trends suggesting treatment

by diagnosis interactions for PANSS ($F(1,69)=3.99$, $p=0.05$); and CAN ($F(1, 69)=3.27$, $p=0.08$).

Non-affective psychosis group

The non-affective group consisted of 50 cases (23 treatment, 27 controls) for whom 43 post-treatment assessments were available. Descriptive results are reported in table 2. Table 3 reports the results of significance testing for the main outcome variables in the non-affective subgroup. The ANCOVAs for the non-affective psychosis group showed significant benefits for treatment (CBT) on constructive economic activity, structured activity, PANSS, and positive beliefs about self; and trends for improvements in hopelessness and instrumental role functioning. There were also significant treatment by centre interactions for structured activity, depression, hopelessness, negative beliefs about self, and both positive and negative beliefs about others. The treatment by centre interactions were consistent with a relatively large treatment effect on activity favouring the expert therapist centre (Centre 2). However, effects on hopelessness and depression tended to favour the non-expert therapist centre (Centre 1).

Affective psychosis group

There were 27 cases in the affective psychosis group who were predominantly people with bipolar disorder. Results for nine cases in the treatment group and 12 in the control group were available at post-treatment. The descriptive statistics in table 2 show suggestions of effects favouring CBT on anxiety and beliefs about self but few indications of effects on activity or other outcomes. However, there were no significant effects for treatment on any of the outcome variables. The main observation is of striking improvements in activity levels for the affective psychosis group in both the treatment and control conditions.

Admissions to hospital

Ten participants had admissions into hospital during the trial. Six of these were in the treatment group and four were in the control group. Average days spent in hospital for the whole sample over the course of the trial was 3.8 (SD = 17.2). In the six months prior to participating in the trial there had been 15 admissions

in the sample. Seven of these were in the group allocated to TAU, and eight were in the group allocated to receive treatment. Average days spent in hospital for the whole sample in the six months preceding the trial was 5.8 (SD = 14.4). Thus, participating in the trial did not appear to have an adverse effect on relapse rates.

DISCUSSION

The trial was designed to refine methods and estimate the effect size of the use of Social Recovery-oriented Cognitive Behaviour Therapy (SRCBT) on the primary outcome of hours in constructive social activity; and secondary outcomes of symptoms, schematic beliefs about self and others, illness cognitions, and hopelessness. The primary study comparison provided no clear evidence for the benefit of CBT on a combined sample of patients with both affective and non affective psychosis. However, a planned secondary analysis revealed some evidence for the potential of CBT to improving constructive and structured activity amongst a more homogeneous sample of patients non affective psychosis with poor social outcomes, relatively early in the course of disorder

The indications of benefits for the cognitive behavioural intervention in non-affective psychosis are promising but require replication in a large multi-centre trial. These gains were large and clinically meaningful. There was an average gain of twelve hours per week in structured activity for CBT in comparison to four hours for TAU in the non-affective psychosis group. This was achieved in association with clinically meaningful and significant improvements in symptoms, hopelessness and beliefs about self and others. The affective psychosis cases (mainly bipolar disorder) also showed large gains in both symptoms and activity but as this occurred in both treatment and control groups it is likely to be the result of a response to treatment as usual conditions and possibly the placebo effect of being involved in a trial.

The study provided a relatively strict evaluation of efficacy as large improvements also occurred in the control group on most of the target variables of outcome, including activity, symptoms and depression. These gains were unexpected as we had deliberately recruited a group of patients who had stable poor social outcome at recruitment and may be the result of a good response to

the treatment as usual provided. The affective psychosis group made particularly large gains in activity and depression in both control and treatment conditions. These observations may be consistent with our recent observations that bipolar disorder cases respond rapidly and with good social recovery outcomes to early intervention services compared with non-affective psychosis (Macmillan *et al.*, 2007). It was certainly the case that there was an active treatment factor in the treatment as usual condition. All cases were in receipt of active treatment from secondary mental health teams. In both centres the control group received more than 20 contacts from these teams over the course of the trial, with some interventions aiming to improve social recovery as well as providing generic case management. Informal observations also suggested that involvement in the therapy trial may have acted as a catalyst for those providing treatment as usual to focus attention on the social recovery needs of cases in both the therapy and control groups. Furthermore involvement in the trial assessment procedures for all cases provided several sessions of discussing, reviewing and monitoring social and symptomatic outcomes which may have had a beneficial effect. It is therefore important to interpret the impact of the study in terms of the effect size of providing an additional focused cognitive behavioural intervention over and above a good existing community mental health service.

Improvements in beliefs about self and others and depression could be taken as support for the cognitive model underpinning the intervention, which had a deliberate focus on deliberately fostering positive self-esteem and hope while working toward adopting new social activities. The aim of the study was also to develop an intervention which deliberately linked improvements in meaningful activities with improvements in psychological well-being and self-esteem, while also managing risk of sensitivity to stress. In this regard it is important to note that there was no indication of any worsening of psychotic symptoms, as has been observed in other studies (Hogarty *et al.*, 1974, Hogarty *et al.*, 1997). Indeed the findings suggest that symptoms improved. Clinical observations by therapists suggested the need to take particular care regarding initial increases in social anxiety symptoms associated with involvement in new activities. However, there was no significant increase in anxiety symptoms over the course of the intervention. We intend to explore the association between

changes in beliefs about self and others, anxiety and activity in future mediational analyses.

This study has highlighted that it was possible for case managers to provide hope and to manage many aspects of cognitive therapy work associated with SRCBT, within their existing case management style of work and skill base. However, there were suggestions that those therapists in the trial who had received more formal prior training (mainly in Centre 1) achieved stronger effects, especially on activity. Supervision discussions and analysis of case notes suggest these differences may have arisen from those therapists who had less formal training in CBT feeling less confident about using more structured active behavioural interventions, particularly in cases where assisting people to engage in new activities may lead to short term increases in anxiety. At the present time, trained CBT therapists may be best placed to deliver the behavioural experiment aspects of this intervention with rigorous levels of adherence and competence. However, this study clearly shows that case managers can deliver an intervention which accrues many significant benefits (particularly in terms of increasing hope); and that it may be possible to develop specific programmes of training focusing on improving their skills to apply the intervention in day-to-day practice at some stage in the future.

The results of this study need to be regarded with caution and as indicative of an effect size useful for researchers undertaking further research. The study was designed to be exploratory rather than confirmatory and lacks power. Results for the non-affective group are therefore suggestive, and those for the affective group are too small to warrant any formal conclusion. The study has been useful in indicating that the key outcome assessments are sensitive to change and, in the case of activity assessment, are relatively independent of other dimensions of outcome. The results also indicate the possible promise of undertaking further research on what appears to be a highly feasible intervention to improve activity in non-affective psychosis. A further large scale trial of this type of intervention is warranted.

ACKNOWLEDGEMENTS

We appreciate the involvement of the trial therapists who included Dorothy O'Connor, Annabella Houlden, Neil Harmer, Cas Wright, Mark Wright, Ian Bell, Nick Whitehouse, Patrick Wymbs, Sarah Newman and Marie Alexander. UK Mental Health Research Network staff including Angela Browne, Freya Mellor and Barbara Dickson provided assistance with recruitment and assessment.

Funding/Declaration of Interest:

Funding for the trial was provided by an MRC trial platform grant.

REFERENCES

American Psychiatric Association (2000). Global Assessment of Functioning. In *DSM-IV-TR*, p. 32. American Psychiatric Association.

Beck, A. T., Rush, A. J., Shaw, B. F. & Emery, G. (1979). *Cognitive Therapy for Depression*. Guilford Press: New York.

Beck, A. T. & Steer, R. A. (1987). *Beck Anxiety Inventory*. The Psychological Corporation: San Antonio, TX.

Beck, A. T. & Steer, R. A. (1988). *Beck Hopelessness Scale Manual*. The Psychological Corporation: San Antonio, TX.

Beck, A. T., Steer, R. A. & Brown, G. K. (1996). *BDI-II Manual*. The Psychological Corporation: San Antonio, TX.

Beecham, J. & Knapp, M. (1992). Costing psychiatric interventions. In *Measuring Mental Health Needs* (ed. G. Thornicroft, C. Brewin and J. Wing), pp. 163-183. Gaskell: London.

Birchwood, M. (2003). Pathways to emotional dysfunction in first episode psychosis. *British Journal of Psychiatry* **182**, 373-375.

Birchwood, M., Mason, R., MacMillan, F. & Healy, J. (1993). Depression, demoralization and control over psychotic illness: a comparison of depressed and non-depressed patients with a chronic psychosis. *Psychological Medicine* **23**, 387-395.

Brooks, R. (1996). EuroQol: the current state of play. *Health Policy* **37**, 53-72.

Butler, G. (1999). *Overcoming Social Anxiety: A Self-Help Guide using Cognitive-Behavioural Techniques*. Constable and Robinson.

Carpenter, W. T. & Strauss, J. S. (1991). The prediction of outcome in schizophrenia. IV: Eleven-year follow-up of the Washington IPSS cohort. *Journal of Nervous and Mental Disease* **179**, 517-525.

Durham, R. C., Guthrie, M., Morton, V., Reid, D. A., Treliving, R. L., Fowler, D. & MacDonald, R. (2003). Tayside-Fife clinical trial of cognitive-behavioural therapy for medication-resistant psychotic symptoms. *British Journal of Psychiatry* **182**, 303-311.

Fowler, D., Freeman, D., Smith, B., Kuipers, E., Bebbington, P., Bashforth, H., Coker, S., Hodgekins, J., Gracie, A., Dunn, G. & Garety, P. (2006). The Brief Core Schema Scales (BCSS): psychometric properties and associations with paranoia and grandiosity in non-clinical and psychosis samples. *Psychological Medicine* **36**, 1-11.

Fowler, D., Garety, P. A. & Kuipers, E. (1995). *Cognitive Behaviour Therapy for Psychosis: Theory and Practice*. Wiley: Chichester.

Goldman, H. H., Skodol, A. E. & Lave, T. R. (1992). Revising axis V for DSM-IV: a review of measures of social functioning. *American Journal of Psychiatry* **149**, 1148-1156.

Gumley, A., O'Grady, M., McNay, L., Reilly, J., Power, K. & Norrie, J. (2003). Early intervention for relapse in schizophrenia: results of a 12-month randomised controlled trial of cognitive behavioural therapy. *Psychological Medicine* **33**, 419-431.

Harrison, G., Croudace, T., Mason, P., Glazebrook, C. & Medley, I. (1996). Predicting the long-term outcome of schizophrenia. *Psychological Medicine* **26**, 697-705.

Heinrichs, D. W., Hanlon, T. E. & Carpenter, B. N. (1984). The Quality of Life Scale: an instrument for rating the schizophrenic deficit syndrome. *Schizophrenia Bulletin* **10**, 388-398.

Hogarty, G. E., Goldberg, S. C., Schooler, N. R. & Ulrich, R. F. (1974). Drug and sociotherapy in the aftercare of schizophrenic patients, II: two-year relapse rates. *Archives of General Psychiatry* **31**, 603-608.

Hogarty, G. E., Kornblith, S. J., Greenwald, P., DiBarry, A. L., Cooley, S., Ulrich, R. F., Carter, M. & Flesher, S. (1997). Three-year trials of personal therapy with schizophrenics living with or independent of family, I: description of study and effects on relapse rates. *American Journal of Psychiatry* **154**, 1504-1513.

Jablensky, A., Sartorious, N. E., G., Anker, M., Korten, A., Cooper, J. & Bertelson, A. (1992). Schizophrenia: manifestations, incidence and course in different cultures. A World Health Organisation Ten Country Study. *Psychological Medicine* **20**, 1-97.

Johnstone, E. C., Macmillan, J. F., Frith, C. D., Benn, D. K. & Crow, T. J. (1990). Further investigation of the predictors of outcome following first schizophrenic episodes. *British Journal of Psychiatry* **157**, 182-189.

Kay, S. R., Fiszbein, A. & Opler, L. A. (1987). The Positive and Negative Syndrome Scale (PANSS) for schizophrenia. *Schizophrenia Bulletin* **13**, 261-276.

Macmillan, I., Howells, L., Kale, K., Hackmann, C., Taylor, G., Hill, K., Bradford, S. & Fowler, D. (2007). Social and symptomatic outcomes of first-episode bipolar psychoses in an early intervention service. *Early Intervention in Psychiatry* **1**, 79-87.

Mueser, K. T., Salyers, M. P. & Mueser, P. R. (2001). A prospective analysis of work in schizophrenia. *Schizophrenia Bulletin* **27**, 281-296.

Sensky, T., Turkington, D., Kingdon, D., Scott, J. L., Scott, J., Siddle, R., O'Carroll, M. & Barnes, T. R. E. (2000). A randomised controlled trial of cognitive-behavioural therapy for persistent symptoms in schizophrenia resistant to medication. *Archives of General Psychiatry* **57**, 165-172.

Short, S. (2006). *Review of the UK 2000 Time Use Survey*. Office for National Statistics: London.

Slade, M., Phelan, M., Thornicroft, G. & Parkman, S. (1996). The Camberwell Assessment of Need (CAN): comparison of assessments by staff and patients of the needs of the severely mentally ill. *Social Psychiatry and Psychiatric Epidemiology* **31**, 109-113.

SPSS (2005). *SPSS Base 14.0 User's Guide*. SPSS Inc.: Chicago, IL.

Strauss, J. S. & Carpenter, W. T. (1977). Prediction of outcome in schizophrenia. III: Five year outcome and its predictors. *Archives of General Psychiatry* **34**, 159-163.

Tsai, S. M., Chen, C., Kuo, C., Lee, J., Lee, H. & Strakowski, S. M. (2001). 15 year outcome of treated bipolar affective disorder. *Journal of Affective Disorders* **63**, 215-220.

Turkington, D., Kingdon, D. & Turner, T. (2002). Effectiveness of a brief cognitive-behavioural therapy intervention in the treatment of schizophrenia. *British Journal of Psychiatry* **180**, 523-527.

Wykes, T., Steel, C., Everitt, B. & Tarrier, N. (2008). Cognitive Behavior Therapy for Schizophrenia: Effect Sizes, Clinical Models, and Methodological Rigor. *Schizophrenia Bulletin* **34**, 523-537.

Figure 1

CONSORT Diagram of Flow of Participants through the Trial

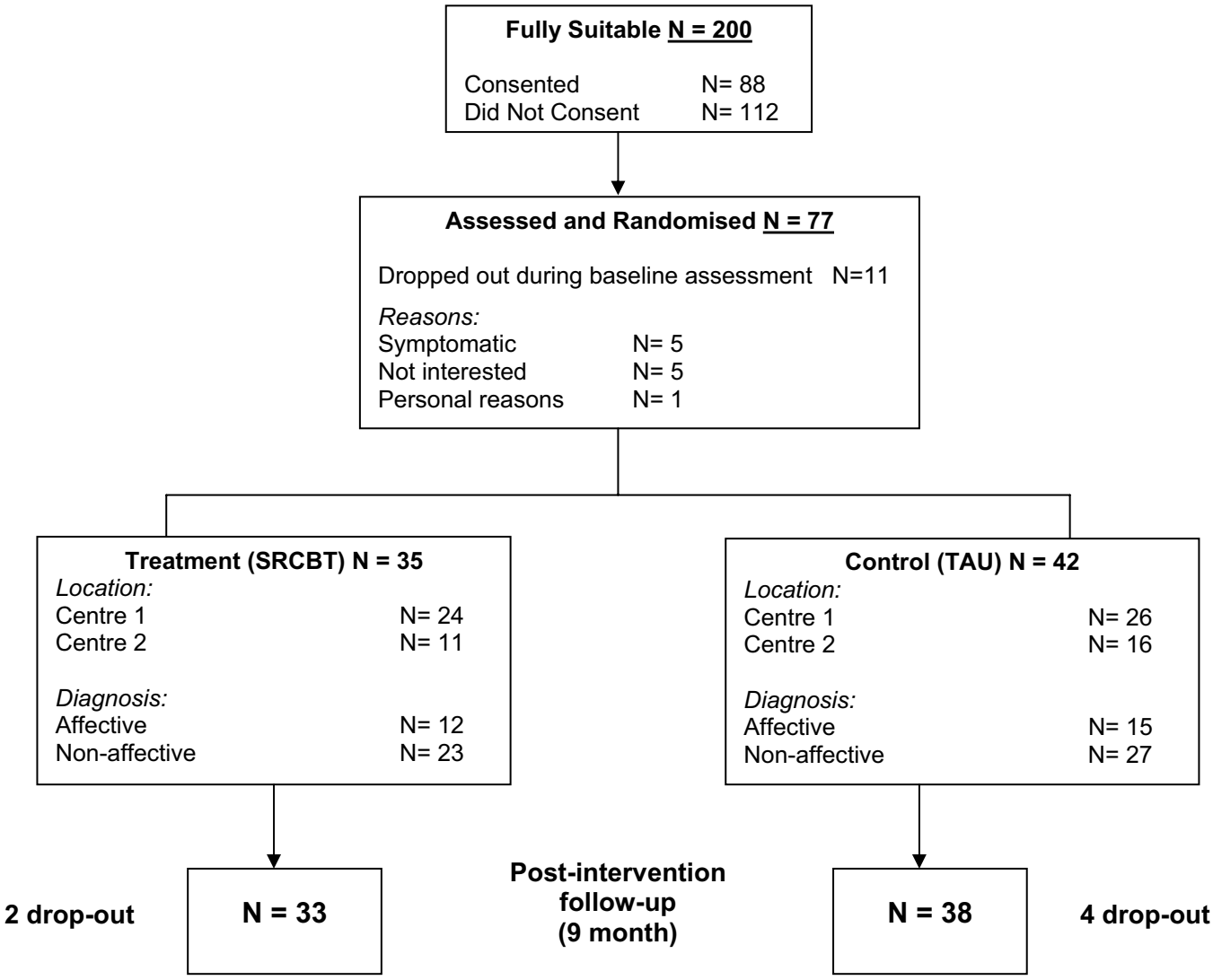


Table 1

Baseline characteristics of participants

	SRCBT (N = 35)	TAU (N = 42)	Total (N = 77)
Demographic characteristics			
Mean Age in yrs (St Dev)	27.8 (6.1)	30.0 (7.2)	29.0 (6.8)
Gender (% male)	71.4%	71.4%	71.4%
Ethnicity (% white)	85.7%	95.2%	90.9%
Diagnosis (% non-affective psychosis)	65.7%	64.3%	64.9%
Mean illness length in yrs (St Dev)	4.9 (2.2)	4.8 (2.4)	4.8 (2.3)
Medication level in mg (St Dev) (chlorpromazine equivalence)	265.1 (200.8)	223.7 (167.0)	242.2 (182.7)
Social and Clinical characteristics			
Mean unemployment length in wks	202.4 (146.0)	214.8 (209.2)	209.1 (182.2)
Time Use in hours per week:			
- Constructive Economic	14.8 (20.2)	10.4 (13.9)	12.4 (17.1)
- Structured	30.4 (19.9)	27.8 (19.2)	29.0 (19.4)
Current IQ	101.8 (11.3)	103.7 (11.3)	102.8 (11.3)
Contacts with Secondary Mental Health Services in last 6mths	32.1 (35.3)	25.9 (23.1)	32.1 (35.3)
Contacts with Voluntary Services in last 6mths	11.0 (18.3)	7.4 (14.4)	9.0 (16.2)

Table 2

Descriptive Statistics – Mean (St Dev) – for Primary, Secondary, and Mediator Variables by Treatment and Diagnosis

		Total Sample		Non-Affective		Affective	
		TAU	CBT	TAU	CBT	TAU	CBT
Primary Outcomes							
Structured Activity	0	27.9 (19.2)	30.4 (19.9)	27.7 (20.0)	25.1 (10.9)	28.2 (18.4)	40.6 (28.5)
	9	34.4 (20.6)	40.0 (22.8)	31.8 (21.3)	37.1 (17.2)	39.8 (18.9)	45.4 (31.2)
Constructive Economic Activity	0	10.4 (13.9)	14.8 (20.2)	8.7 (13.3)	10.3 (7.3)	13.6 (14.7)	23.6 (32.1)
	9	15.6 (15.9)	19.2 (21.0)	11.9 (13.6)	14.7 (12.9)	22.4 (18.1)	28.6 (30.6)
Secondary Outcomes							
PANSS Total	0	56.0 (10.3)	57.6 (11.6)	58.1 (9.4)	57.5 (10.8)	52.1 (11.0)	58.0 (13.4)
	9	50.4 (10.1)	50.5 (9.2)	53.2 (8.3)	50.3 (8.2)	44.5 (11.3)	50.7 (11.3)
Quality of Life	0	62.7 (14.8)	66.8 (14.8)	58.2 (11.0)	64.1 (10.2)	70.7 (17.5)	71.7 (20.5)
	9	72.5 (18.5)	76.1 (14.0)	67.1 (15.0)	72.8 (12.3)	83.8 (20.5)	82.3 (15.5)
Role Functioning	0	5.6 (3.8)	6.6 (4.1)	4.6 (2.9)	5.8 (3.5)	7.4 (4.6)	8.2 (4.9)
	9	7.2 (5.7)	9.0 (5.6)	6.1 (5.3)	8.3 (5.6)	9.5 (5.9)	10.5 (5.4)
Beck Hopelessness	0	8.7 (5.8)	8.9 (5.8)	8.0 (5.5)	8.3 (5.5)	10.2 (6.4)	10.2 (6.3)
	9	7.9 (5.8)	6.4 (4.7)	8.2 (5.9)	4.9 (2.3)	7.3 (5.9)	9.3 (6.6)
Schizotypal Symptoms	0	21.7 (14.5)	15.9 (17.5)	24.8 (16.5)	10.5 (9.3)	15.8 (6.7)	26.6 (24.8)
	9	24.8 (16.6)	18.6 (12.8)	26.0 (18.4)	18.0 (12.3)	22.8 (13.8)	19.7 (14.4)
BCSS – Negative Self	0	6.7 (6.3)	5.5 (5.1)	6.4 (6.7)	4.0 (3.5)	7.2 (5.8)	8.3 (6.5)
	9	4.9 (4.4)	4.0 (5.3)	4.2 (3.8)	2.7 (2.3)	5.9 (5.2)	6.2 (7.8)
BCSS – Positive Self	0	9.2 (6.4)	8.4 (5.6)	8.8 (6.8)	8.6 (5.5)	10.0 (5.8)	8.1 (6.1)
	9	10.6 (6.8)	11.9 (6.0)	10.0 (7.6)	11.6 (5.7)	11.5 (5.3)	12.3 (6.8)
BCSS – Negative Other	0	6.7 (6.6)	6.6 (6.3)	7.7 (7.1)	5.7 (5.7)	4.8 (5.2)	8.1 (7.2)
	9	4.9 (4.6)	3.5 (4.2)	4.3 (3.8)	2.9 (3.6)	5.8 (5.8)	4.3 (5.2)
BCSS – Positive Other	0	11.7 (6.5)	8.9 (5.5)	11.2 (6.9)	8.5 (5.4)	12.6 (5.6)	9.6 (5.8)
	9	10.0 (6.1)	11.9 (6.2)	9.5 (6.5)	12.4 (5.9)	10.8 (5.6)	11.2 (6.8)
Tertiary Outcomes							
SOFAS	0	48.9 (7.9)	51.5 (9.0)	47.3 (6.8)	50.1 (6.8)	51.8 (9.1)	54.2 (12.1)
	9	53.8 (12.3)	54.8 (9.4)	51.5 (11.3)	53.7 (9.2)	58.3 (13.3)	56.9 (10.1)
CAN Number of Needs	0	6.9 (3.4)	5.6 (2.3)	7.1 (3.5)	6.0 (2.4)	6.4 (3.2)	4.9 (2.2)
	9	5.5 (2.5)	5.3 (1.8)	6.2 (2.3)	5.5 (1.8)	4.1 (2.3)	5.0 (1.9)
EuroQol	0	57.5 (21.8)	52.9 (25.4)	55.7 (22.5)	58.4 (21.9)	60.5 (21.2)	42.3 (29.2)
	9	65.7 (18.2)	65.8 (19.8)	67.2 (16.9)	67.9 (13.9)	63.7 (20.4)	62.0 (27.9)
Personal Beliefs About Illness	0	21.8 (7.6)	21.2 (7.6)	22.5 (8.4)	19.8 (7.3)	20.4 (5.6)	23.9 (7.7)
	9	19.7 (6.8)	18.9 (6.2)	19.9 (7.2)	17.8 (4.0)	19.2 (6.4)	20.6 (8.8)
GAS	0	57.2 (8.8)	56.4 (10.9)	55.4 (8.6)	56.7 (8.9)	60.5 (8.4)	55.8 (14.4)
	9	60.2 (14.1)	59.2 (10.9)	56.6 (12.6)	58.5 (10.9)	67.9 (14.6)	60.7 (11.0)
Beck Depression	0	22.6 (13.8)	21.1 (13.9)	21.4 (14.4)	17.9 (11.3)	24.7 (12.8)	27.0 (16.5)
	9	14.4 (12.7)	13.6 (10.6)	14.3 (11.5)	11.3 (7.5)	14.7 (14.9)	17.2 (14.0)
Beck Anxiety	0	17.0 (11.8)	16.9 (13.5)	16.6 (13.0)	14.8 (12.8)	17.7 (9.8)	21.1 (14.5)
	9	13.2 (10.5)	13.0 (12.8)	12.3 (9.7)	11.6 (11.9)	14.7 (12.0)	15.3 (14.6)

Table 3

Results of model estimates of treatment effects within the non-affective psychosis group (using EM estimates for missing data)

	Main Effect (of CBT)	Interaction (CBT x centre)
Primary Outcome Variables		
Structured Activity	***F*(1,43)=11.73, *p*=0.001**	***F*(1,43)=5.44, *p*=0.02**
Constructive Economic Activity	***F*(1,44)=6.19, *p*=0.02**	*F*(1,43)=0.79, *p*=0.38
Secondary Outcome Variables		
PANSS Total	***F*(1,44)=4.56, *p*=0.04**	*F*(1,43)=0.05, *p*=0.82
Quality of Life	*F*(1,44)=1.54, *p*=0.22	*F*(1,43)=0.16, *p*=0.69
Instrumental Role Functioning	*F*(1,44)=3.32, *p*=0.08	*F*(1,43)=0.59, *p*=0.45
Beck Hopelessness Scale	*F*(1,44)=3.79, *p*=0.06	*F*(1,43)=3.60, *p*=0.07
Schizotypal Symptoms	*F*(1,45)=0.23, *p*=0.64	*F*(1,44)=2.73, *p*=0.11
BCSS – Negative Self	*F*(1,43)=0.01, *p*=0.93	***F*(1,43)=5.40, *p*=0.03**
BCSS – Positive Self	***F*(1,44)=5.52, *p*=0.02**	*F*(1,43)=2.38, *p*=0.13
BCSS – Negative Other	*F*(1,43)=0.001, *p*=0.98	***F*(1,43)=8.54, *p*=0.006**
BCSS – Positive Other	*F*(1,43)=0.18, *p*=0.67	***F*(1,43)=17.15, *p*=<0.0001**
Tertiary Outcome Variables		
Global Assessment of Symptoms	*F*(1,44)=0.48, *p*=0.49	*F*(1,43)=0.01, *p*=0.92
Beck Depression Inventory	*F*(1,43)=0.03, *p*=0.87	***F*(1,43)=9.95, *p*=0.003**
Beck Anxiety Inventory	*F*(1,44)=0.001, *p*=0.97	*F*(1,43)=0.08, *p*=0.78
Social and Occupational Functioning	*F*(1,44)=2.43, *p*=0.13	*F*(1,43)=0.75, *p*=0.39
CAN Number of Needs	*F*(1,44)=2.96, *p*=0.09	*F*(1,43)=0.30, *p*=0.58
EuroQol	*F*(1,44)=0.17, *p*=0.68	*F*(1,43)=2.61, *p*=0.11
Personal Beliefs About Illness	*F*(1,44)=0.20, *p*=0.62	*F*(1,43)=1.08, *p*=0.30

APPENDIX C

Letters of Approval from Ethics Committees

C1 Ethical approval for web study from University of East Anglia

C2 Ethical approval for web study from King's College London

C3 Ethical approval for ISREP study

C4 Ethical approval for EI study

School of Medicine, Health Policy and Practice
Institute of Health

University of East Anglia
Norwich NR4 7TJ England

Telephone
01603 456161
Direct dial 01603 593061
Fax
01603 593752
Email
www.med.uea.ac.uk

Joanne Hodgekins
School of Medicine, Health Policy and Practice
University of East Anglia
Norwich
NR4 7TJ

23 November 2004

Dear Joanne

RE: An investigation into the structure and underlying mechanisms of Schizotypy in a non-clinical sample.

Your letter dated 5 November 2004 was forwarded for 'Chairs Action', to consider a few minor concerns regarding your research proposal as discussed at the Institute of Health Ethics Committee meeting held on Thursday 28 October 2004.

These revisions have been approved and the Committee does not require any further amendments to be made.

It is necessary to stress however, the Committee's view that the content of the questionnaires themselves, could have been distressing. Although the original form of this study has been approved by the IOP/SLAM Research Ethics Office, it is felt that support structures at UEA should be kept informed about projects which may have an impact on the student body. It would therefore be advisable to make sure that both the Health Centre and the Dean of Students have had a chance to see all questionnaires so that they have an accurate idea about what students may be referring to.

Best wishes with your research.

Yours sincerely

Helen Dodgson
Notetaker
Institute of Health (MED, NAM, AHP) Ethics Committee
Tel: 01603 591258
Email: h.dodgson@uea.ac.uk

Institute of Psychiatry

at The Maudsley

Ethical Committee
(Research)
Research Ethics Co-ordinator
Margaret M Chambers Msc

P006, Room W109
De Crespigny Park
Denmark Hill
London SE5 8AF
Tel 020 7848 0797
Fax 020 7848 0147
Email m.chambers@iop.kcl.ac.uk

KING'S College LONDON

University of London

ETHICAL COMMITTEE (RESEARCH)

23 August 2004

Prof P Garety
PO77
Dept of Psychology
Institute of Psychiatry

Dear Prof Garety

Re: Structure in schizotypy (142/04)

The Chair of the Ethical Committee (Research) has taken action to approve this study from an ethical point of view. **Please note that this approval is subject to deletion of words 'and understand' from statement one of the consent form; and deletion of reference to medical care in statement two of the consent form as participants are non-clinical volunteers.**

Please note that this approval is subject to confirmation by the full Committee when it meets on 17 September 2004. Initial approval is given for one year. This will be extended automatically only on completion of annual progress reports on the study when requested by the EC(R). Please note that as Principal Investigator you are responsible for ensuring these reports are sent to us.

Please note that projects which have not commenced within two years of original approval must be re-submitted to the EC(R).

Any serious adverse events which occur in connection with this study should be reported to the Committee using the attached form.

Please quote Study No. 142/04 in all future correspondence.

Yours sincerely,

Margaret M Chambers
Research Ethics Coordinator

Norwich Local Research Ethics
Committee
c/o Norfolk & Norwich University
Hospital NHS Trust
Clinical Governance Department
Aldwych House
57 Bethel Street
Norwich
NR2 1NR
Tel: 01603 289813 / 286397

17 June 2004

Mr David Fowler
Reader in Clinical Psychology
School of Medicine Health Policy & Practice
University Plain
University of East Anglia
Norwich
NR4 7TJ

Dear Mr Fowler

Full title of study: Improving social recovery in early affective and non-affective psychosis: a randomised controlled trial of Social Recovery orientated Cognitive Behaviour (SRCBT)
REC reference number: 04/Q0101/1
Protocol number:

Thank you for your letter of 26 May 2004, responding to the Committee's request for further information on the above research and submitting revised documentation.

The further information has been considered on behalf of the Committee by the Chairman.

Ethical opinion

On behalf of the Committee, I am pleased to confirm a favourable ethical opinion for the above research on the basis described in the application form, protocol and supporting documentation as revised.

The favourable opinion applies to the research sites listed on the attached sheet. Confirmation of approval for other sites listed in the application will be issued as soon as local assessors have confirmed that they have no objection.

Conditions of approval

The favourable opinion is given provided that you comply with the conditions set out in the attached document. You are advised to study the conditions carefully.

Notification of other bodies

We shall notify Dr Mary Cubitt, Research Manager as the representative of the research sponsor (the Norfolk Mental Health Care NHS Trust) that the study has a favourable ethical opinion.

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

REC reference number: 04/Q0101/1 **Please quote this number on all correspondence**

Yours sincerely

Administrator

Enclosures *Standard approval conditions [SL-AC2]*

List of approved sites

CC: *Dr Mary Cubitt, Research Manager, Norfolk Mental Health Care NHS Trust*
East Norfolk and Waveney Research Governance Committee Ref 2004MH02

APPENDIX D

Information Sheet and Consent Forms for Clinical Sample

D1 Information sheet for ISREP study

D2 Consent form for ISREP study

D3 Information sheet for EI study

D4 Consent form for EI study

ISREP *improving social recovery in early psychosis*

Funded by the MRC

Norfolk and Waveney Mental Health Partnership NHS Trust

UEA NORWICH

ISREP Project Team
c/o Early Intervention Service
80 St Stephens Road
Norwich
NR1 3RE

Improving Social Recovery in Early Psychosis: Participant Information Sheet

Invitation Paragraph
You are being invited to take part in a research study. Before you decide, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with others if you wish. Ask us if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part. Thank you for reading this

What is the purpose of the study?
People who have episodes of worrying, distressing or unusual experiences or beliefs often recover from the worst of these experiences, but may continue to have difficulties in maintaining social contacts and social activities or in returning to or taking up employment or educational opportunities. We think people can be helped to make a better social recovery by working with a therapist using a therapy called social recovery oriented cognitive behaviour therapy (SRCBT). The study aims to see if working with a therapist helps to improve social recovery and to reduce symptoms of hopelessness and anxiety if present.

Why have I been chosen?
We are approaching people who have had a first episode of psychosis within the last eight years, who are unemployed, and who are in contact with their community mental health teams. The whole study will involve 100 patients in Norfolk and Cambridge.

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 given this information sheet to keep and will be asked to sign a consent form. If you decide to take part, you are still free to withdraw at any time and without giving a reason. A decision to withdraw at any time, or a decision not to take part, will not affect the standard of care you receive.

What will happen to me if I take part?
If you do agree to take part, you will meet with a researcher who will ask about your current problems and social situation, after this you will either be offered SRCBT and your usual treatment or your usual treatment with your team alone. After nine months you will then meet with the researcher again to repeat the assessments. This is a randomised trial in which, as we do not know which way of treating patients is best, we need to make comparisons. People will be put into groups and then compared. The groups will be selected by a computer which has no information about the individual i.e. by chance. Patients in each group then have a different treatment and these are compared. You will have a 50/50 chance of receiving SRCBT or treatment as usual.

What do I have to do?
SRCBT involves weekly or fortnightly meetings with a therapist for up to nine months which will be arranged at a time to suit you. Expenses for attending such appointments can be claimed

What is the therapy being tested?
The aims of SRCBT are: to carefully identify activities and occupations which are meaningful for the person; to understand any barriers people may have to undertaking the activity the person wants to do; and to help people prepare for work or leisure activities by practicing in safe and low stress environments. This kind of help is called Social Recovery oriented Cognitive Behaviour Therapy (SRCBT). Social recovery is the aim. CBT tries to help you to understand what you are experiencing and feeling, cope with it differently, and feel less worried when you are trying to do new things.

SRCBT is a relatively new treatment. We still do not know how exactly it helps people to improve. The main aim of the study is therefore to see if SRCBT works, but we also want to improve our understanding of this type of treatment so that we can develop it further to be more helpful.

What are the alternatives for treatment?
Many existing therapeutic approaches which sometimes form part of normal treatment in mental health services such as case management, occupational therapy, vocational therapy and rehabilitation aim to improve social recovery. These will be sometimes be available as part of normal treatment to all participants. Where these treatments are available SRCBT aims to enhance these treatments.

What are the possible disadvantages and risks of taking part?
If people feel pressurised into undertaking new activities they can sometimes have a recurrence of symptoms. However, the aim of SRCBT is to help people explore new activities they want to do while taking care to minimise the risk of symptom recurrence.

What are the possible benefits of taking part?
We hope that all the treatments will help you. However, this cannot be guaranteed. The information we get from this study may help us treat future patients better.

What happens when the research study stops?
When the research study finishes, all participants will receive normal care from local mental health services.

What happens if something goes wrong?
If you are harmed by taking part in a research project there are no special compensation arrangements. If you are harmed by someone's negligence you may have grounds for legal action but you may have to pay for it. Regardless of this, if you wish to complain, or have any concerns about any aspect of the way you have been approached or treated during the course of the study, the normal National Health Service complaints mechanisms should be available to you.

Will my taking part in this study be kept confidential?
If you consent to take part in the study we will check your medical records for details of your care and other treatment. All information that is collected about you during the course of the research will be kept strictly confidential. Any information about you that leaves the Hospital/Trust will have your name and address removed so that you cannot be recognised from it.

If you consent, we will inform your consultant psychiatrist and the team responsible for your care about your involvement in the study. We will send them a very brief summary of our assessment unless you do not wish us to do this.

Since we are trying to provide the very best treatment possible, we would like to audio tape sessions that you have with your therapist. The reason for this is to check that the therapy is carried out in the way that we expect it to be. We will ask you separately for your consent to this.

Where and how long will records be stored?
Data will be stored in locked cabinets in local health care or university premises. It will be kept for 5 years after the completion of the study and then destroyed.

What will happen to the results of the research study?
When the study is finished the results will be published. This is likely to be in 2007. We will ensure that copies of the report are available to local users groups, and we will arrange local talks to which participants will be invited. We will not identify you individually in any report or publication of the research.

Who is organising and funding the research?
The research is funded by the Medical Research Council. It is being carried out by researchers from University of East Anglia, University of Cambridge and staff working at Norfolk and Waveney Mental Health and Social Care Partnership and Cambridge University Teaching Hospitals Trust.

Who has reviewed the study?
The research has been considered and approved by the Norwich Local Research Ethics Committee.

Thank you for reading this. If you need further information, please contact a member of the research team. The names of people to contact are given below.

We will give you this information sheet to keep as well as a signed consent form if you agree to take part in the study.

Contact for further information:
David Fowler, Principal Investigator. School of Medicine, Health Policy and Practice, UEA, NR4 7TJ (Tel: 01603 593637).
Mark Wright, Trial Therapist. Early intervention Service, 80 St Stephens St, Norwich (Tel: 01603 201552).
Jo Hodgekins, Assistant Psychologist/Research Associate. School of Medicine, Health Policy and Practice, UEA, NR4 7TJ (Tel: 01603 591232).

ISREP *improving social recovery in early psychosis*

Funded by the MRC

Norfolk and Waveney Mental Health Partnership NHS Trust

UEA NORWICH

Centre Number:
Study Number:
Patient Identification Number for this trial:

CONSENT FORM

Title of Project: Improving social recovery in Early Psychosis

Name of Researcher:

Please initial box

1. I confirm that I have read and understand the information sheet dated 26/05/2004 (version 2) for the above study and have had the opportunity to ask questions. ☐

2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. ☐

3. I understand that sections of any of my medical notes may be looked at by responsible individuals working on the improving social recovery project or from regulatory authorities where it is relevant to my taking part in research. I give permission for these individuals to have access to my records. ☐

4. I am willing for my care team/consultant psychiatrist to be informed of my participation in this project, and for assessment information regarding my current problems and social circumstances to be shared with my care team/consultant psychiatrist. ☐

5. I give my consent for tape recordings of assessment and treatment sessions to be made. I understand that this is for the purposes of training and supervision, and that any person hearing the tape will sign a declaration of confidentiality and that recordings will be stored under locked conditions. ☐

4. I agree to take part in the above study. ☐

_____	_____	_____
Name of Patient	Date	Signature
_____	_____	_____
Name of Person taking consent (if different from researcher)	Date	Signature
_____	_____	_____
Researcher	Date	Signature

1 for patient; 1 for researcher; 1 to be kept with hospital notes

Trauma in Psychosis Study
In collaboration with
the Early Intervention
in Psychosis Service

Norfolk Mental Health Care NHS
NHS Trust

UEA
NORWICH

Early Intervention Service
80 St Stephens Road,
Norwich,
NR1 3RE.

Participant information sheet.

Study: **Trauma in Psychosis.**

You are being invited to take part in a research study. Before you decide, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and feel free to discuss it. Ask us if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part. Thank you for reading this.

What is the purpose of the study?
The aim of this study is to look at how much trauma there is in psychosis, and the possible effects of the trauma. Trauma means a frightening or threatening experience. Please feel free to ask us about what trauma means if you would like more information. This study is being carried out as part of a research degree.

Existing research suggests that many people who have problems associated with unusual experiences and beliefs have been exposed to threatening life events and trauma and may have problems associated with intrusive memories and emotions associated with these events. We need to know how often such problems occur. We also want to know to what degree problems associated with trauma relate to other symptoms, depression and problems in attention and memory.

What will happen to me if I take part in the study?
We are firstly asking if we could use the information which is collected to understand your needs as part of the Early Intervention Service for research purposes. The reason why you are being invited to take part in this research is that we feel that your answers to these routine interviews could benefit the understanding of the experiences and needs of people who have had problems like your own.

These assessments are carried out with everybody whether or not they consent to take part in the study. If you consent to take part this information would be entered and stored on a data

base. This data will be anonomised and confidential. No names or addresses will be entered on the data base and you could not be identified from the data.

Secondly, if you participate we may ask you to answer some extra questions. This will add up to 30 minutes to your routine assessment interviews. We will indicate which are the additional study specific questions during the interview. **Whether or not you consent to these additional questions is up to you.** As mentioned earlier all of the information you give us is confidential therefore, the information used for this study is anonymous and will not have your name or any personal details on it.

Do I have to take part in the study?

Whether or not you take part in the study is up to you. The answers that you give during the routine clinical assessments will not be used for research purposes without your consent. Furthermore, you do not have to consent to answer any additional questions that are not part of your routine clinical assessment. If you do not consent to take part in this study your routine clinical interviews will not be affected. If you do consent to take part in this study you can opt out at any time. Opting out of the study at any time will in no way be of detriment to your care.

Will my taking part in the study be kept confidential?

All the information collected about you in the course of the research will be kept strictly confidential, in accordance with requirements of the Data Protection Act. Information from all of the people who agree to take part will be stored using anonimised codes (as opposed to the use of names). The information that you give during the routine interviews for the Early Intervention Service will be available to the members of this service. The information that you give relating to additional questions associated with the research study will also be available to the Early Intervention team. Your name, however, will not be disclosed outside the Norfolk Mental Health Care Trust.

What will happen to the results of the research study?

When we have finished collecting information for our research we aim to prepare results for publication. We will also present the findings at local talks at which you can attend should you wish.

Is there anything else I should know?

If you consent to take part in the research your mental health records may be looked at by the research team for the purposes of analysing the results. These records are confidential therefore your name will not be disclosed.

If you consent, we will inform your consultant psychiatrist and the Early Intervention Team and your GP about your involvement in the study unless you do not wish us to do so. We will send them a very brief summary of our assessment unless you do not wish this.

It is important for us to have an accurate record of our interview. We would therefore like to ask you if it would be ok for us to audio tape the interview. You will be asked separately for your consent to do this. These tapes will be kept in a locked cabinet under the Data Protection Act. These tapes will be destroyed after a three year period.

This research has been reviewed and a favourable opinion given by the Norwich Local Research Ethics Committee.

If you decide to consent to this study you will be given this information sheet to keep and a signed consent form. If you decide to help us with our research you are still free to change your mind at ay time without giving a reason. A decision to withdraw at any time or not take part in the research will not affect your relationship with the Early Intervention team.

Thank you for taking the time to read this. If you need further information please feel free to ask any questions you have, or contact a member of the research team. The names of the people to contact are given below.

Contact for further information.

Research team:

Corinna Hackmann. Lead Researcher. School of Health Policy and Practice. UEA. NR4 7TJ. (01603) 593541.

Freya Mellor, Gavin Taylor, Helen Lockett, Sarah Fish & Katharina Joosen. Assistant Psychologists. Early Intervention Service. 80 St Stephens Rd. Norwich, NR1 3RE. (01603) 201550.

David Fowler. Principal Investigator. School of Health, Policy and Practice. UEA. NR4 7TJ. (01603) 593637.

Dr Iain Macmillan, Consultant Psychiatrist, Early Intervention Service, 80 St Stephens Rd, Norwich, NR1 3RE. (01603) 201550.

Trauma in Psychosis Study
In collaboration with
the Early Intervention
in Psychosis Service

Norfolk Mental Health Care NHS
NHS Trust

UEA
NORWICH

Centre Number:
Study Number:
Patient Identification Number for this study:

CONSENT FORM

Trauma in Psychosis Study

NAME OF RESEARCHER:

Please initial box

1. I confirm that I have read and understand the information sheet dated 17/06/2005 (version number 4) for the above study and have had the opportunity to ask questions. ☐

2. I understand that my participation is voluntary and that I am free to withdraw at any time without giving any reason, without my legal rights and medical care being affected ☐

3. I consent for the answers given during routine Early Intervention Service clinical and needs assessment interviews to be used for research purposes ☐

4. I understand that if I consent to take part in this study I may be asked additional questions for the specific purpose of research in this study. I give consent for these questions to be asked and the answers given to these questions to be used for research purposes. ☐

5. I understand that sections of my mental health notes may be looked at by responsible individuals from the research team at the Early Intervention Service (Norfolk mental health care trust) or regulatory authorities where it is relevant to my taking part in research. I give permission for these individuals to have access to my records. ☐

Please initial box

6. I give my consent for tape recordings of the interviews to be made, I understand that this is for research purposes, and that any person hearing the tape will sign a declaration of confidentially and that the recordings will be kept under locked conditions. ☐

7. I give consent for my GP, consultant psychiatrist and the Early Intervention Team to be informed that I am taking part in the study. ☐

8. I agree to take part in the above study. ☐

Name of participant	Date	Signature
Name of person taking consent (if different from researcher)	Date	Signature
Researcher	Date	Signature

1 for participant; 1 for researcher; 1 to be kept with hospital notes.

APPENDIX E

Study Measures

E1 Schizotypal Symptoms Inventory (Long version)

E2 Schizotypal Symptoms Inventory (Brief version)

E3 Trauma History Screen

E4 Time Use Survey

SSI (Long Version)

Please answer each item by choosing either "Yes" or "No". If you choose "Yes", please state how often this experience has occurred over the **past 2 weeks**. Please answer all of the questions honestly, even if you are unsure of your answer.

1.	Do you sometimes feel that things you see on the TV or read in the newspaper have a special meaning for you?		*Yes*			*No*
	Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
2.	I sometimes avoid going to places where there will be many people because I will get anxious.		*Yes*			*No*
	Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
3.	Have you had experiences with the supernatural?		*Yes*			*No*
	Has this happened in the **past 2 weeks?**	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
4.	Have you often mistaken objects or shadows for people, or noises for voices?		*Yes*			*No*
	Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
5.	Other people see me as slightly eccentric (odd).		*Yes*			*No*
	Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
6.	I have little interest in getting to know other people.		*Yes*			*No*
	Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
7.	People sometimes find it hard to understand what I am saying.		*Yes*			*No*
	Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
8.	People sometimes find me aloof and distant.		*Yes*			*No*
	Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*

9.	I am sure I am being talked about behind my back.		*Yes*		*No*	
	Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
10.	I am aware that people notice me when I go out for a meal or to see a film.		*Yes*		*No*	
	Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
11.	I get very nervous when I have to make polite conversation.		*Yes*		*No*	
	Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
12.	Do you believe in telepathy (mind-reading)?		*Yes*		*No*	
	Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
13.	Have you ever had the sense that some person or force is around you, even though you cannot see anyone?		*Yes*		*No*	
	Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
14.	People sometimes comment on my unusual mannerisms and habits.		*Yes*		*No*	
	Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
15.	I prefer to keep myself to myself.		*Yes*		*No*	
	Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
16.	I sometimes jump quickly from one topic to another when speaking.		*Yes*		*No*	
	Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
17.	I am poor at expressing my true feelings by the way I talk and look.		*Yes*		*No*	
	Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
18.	Do you often feel that other people have got it in for you?		*Yes*		*No*	
	Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*

19. Do some people drop hints about you or say things with a double meaning?	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
20. Do you ever get nervous when someone is walking behind you?	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
21. Are you sometimes sure that other people can tell what you are thinking?	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
22. When you look at a person, or yourself in a mirror, have you ever seen the face change right before your eyes?	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
23. Sometimes other people think that I am a little strange.	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
24. I am mostly quiet when with other people.	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
25. I sometimes forget what I am trying to say.	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
26. I rarely laugh and smile.	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
27. Do you sometimes get concerned that friends or co-workers are not really loyal or trustworthy?	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
28. Have you ever noticed a common event or object that seemed to be a special sign for you?	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*

29. I get anxious when meeting people for the first time.	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
30. Do you believe in clairvoyancy (psychic forces, fortune telling, etc)?	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
31. I often hear a voice speaking my thoughts aloud.	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
32. Some people think that I am a very bizarre person.	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
33. I find it hard to be emotionally close to other people.	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
34. I often ramble on too much when speaking.	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
35. My "non-verbal" communication (smiling and nodding during a conversation) is poor.	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
36. I feel I have to be on my guard, even with friends.	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
37. Do you sometimes see special meanings in advertisements, shop windows, or in the way things are arranged around you?	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
38. Do you often feel nervous when you are in a group of unfamiliar people?	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*

39. Can other people feel your feelings when they are not there?		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
40. Have you ever seen things invisible to other people?		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
41. Do you feel that there is no-one you are really close to outside of your immediate family, or people you can confide in or talk to about emotional problems?		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
42. Some people find me a bit vague and elusive during a conversation.		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
43. I am poor at returning social courtesies and gestures.		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
44. Do you often pick up hidden threats or put-downs from what people say or do?		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
45. When shopping, do you get the feeling that other people are taking notice of you?		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
46. I feel very uncomfortable in social situations involving unfamiliar people.		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
47. Have you had experiences with astrology, seeing the future, UFOs, ESP or a sixth sense?		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*

48. Do everyday things seem unusually large or small?		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
49. Writing letters to friends is more trouble than it is worth.		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
50. I sometimes use words in unusual ways.		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
51. I tend to avoid eye contact when conversing with others.		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
52. Have you found that it is best not to let other people know too much about you?		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
53. When you see people talking to each other, do you often wonder if they are talking about you?		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
54. I would feel very anxious if I had to give a speech in front of a large group of people.		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
55. Have you ever felt that you are communicating with another person telepathically (by mind-reading)?		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
56. Does your sense of smell sometimes become unusually strong?		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
57. I tend to keep in the background on social occasions.		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*

58. Do you tend to wander off the topic when having a conversation?	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
59. I often feel that others have it in for me.	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
60. Do you sometimes feel that other people are watching you?	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
61. Do you ever suddenly feel distracted by distant sounds that you are not normally aware of?	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
62. I attach little importance to having close friends.	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
63. Do you sometimes feel that people are talking about you?	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
64. Are your thoughts sometimes so strong that you can almost hear them?	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
65. Do you often have to keep an eye out to stop people from taking advantage of you?	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
66. Do you feel that you are unable to get “close” to people?	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
67. I am an odd unusual person.	*Yes*			*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*

68. I do not have an expressive and lively way of speaking.		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
69. I find it hard to communicate clearly what I want to say to people.		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
70. I have some eccentric (odd) habits.		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
71. I feel very uneasy talking to people I do not know well?		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
72. People occasionally comment that my conversation is confusing.		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
73. I tend to keep my feelings to myself.		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
74. People sometimes stare at me because of my odd appearance.		*Yes*		*No*	
Has this happened in the **past 2 weeks**?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*

SSI (Brief Version)

Please answer each item depending on how often (if at all) this experience has occurred over the **past 2 weeks**. Please answer all of the questions honestly, even if you are unsure of your answer.

1. I sometimes avoid going to places where there will be many people because I will get anxious.	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
2. Do you believe in telepathy (mind-reading)?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
3. I am sure I am being talked about behind my back.	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
4. I get very nervous when I have to make polite conversation.	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
5. Have you had the sense that some person or force is around you, even though you cannot see anyone?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
6. Do you often feel that other people have got it in for you?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
7. I feel very uneasy talking to people I do not know well.	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
8. Have you noticed a common event or object that seemed to contain a special sign for you?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
9. When you see people talking to each other, do you often wonder if they are talking about you?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
10. I often hear a voice speaking my thoughts aloud.	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
11. Do you often feel nervous when you are in a group of unfamiliar people?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
12. I often feel that others have it in for me.	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
13. Have you seen things invisible to other people?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*

14. I feel very uncomfortable in social situations involving unfamiliar people.	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
15. Do you sometimes feel that people are talking about you?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
16. Can other people feel your feelings when they are not there?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
17. I get anxious when meeting people for the first time.	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
18. Do you believe in clairvoyancy (psychic forces, fortune telling)?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
19. Do you sometimes feel that other people are watching you?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*
20. Have you felt that you are communicating with another person telepathically (by mind-reading)?	*Not at all*	*Occasionally*	*Sometimes*	*Often*	*All of the time*

Trauma Questions for Web Study (selected items taken from the Traumatic Life Events Questionnaire (TLEQ; Kubany et al, 2000) and the Trauma History Questionnaire (THQ; Green, 1996)

You will now be asked some questions about different types of stressful events that may have happened in your life. These kinds of events can be traumatic and disturbing.

Some of the questions are quite personal so only answer those you feel comfortable with. If you are comfortable with answering the question, all you will be asked is if the event has happened in your lifetime and how you feel about it now. You will not be asked for in-depth details.

1. Have you ever been in a serious car accident or serious accident at work, or somewhere else?

   If this has happened:
   - Did you ever think you might be killed or seriously injured as a result of the event? (yes/no)
   - Did you experience intense fear, helplessness or horror when it happened? (yes/no)
   - Does the event still affect you? (yes/no)
   - Do you still think about the event? (yes/no)
   - Do you avoid thinking about the event? (yes/no)
   - Please use the space below to give a brief indication of the severity of the event (e.g. minor burns, near death in hospital, etc)

2. Has anyone ever done anything particularly nasty or cruel to you? Has anyone ever been unnecessarily mean to you? Has anyone ever tormented you? Have you ever been teased, taunted or actually bullied by anyone?

   If this has happened:
   - Did you ever think you might be killed or seriously injured as a result of the event? (yes/no)
   - Did you experience intense fear, helplessness or horror when the event happened? (yes/no)
   - Does the event still affect you? (yes/no)
   - Do you still think about the event? (yes/no)
   - Do you avoid thinking about the event? (yes/no)
   - Please use the space below to give a brief indication of the severity of the event (e.g. single episode of taunting at school, continual emotional abuse, etc)

3. Did you ever have sexual contact with anyone who was at least five years older than you before you reached the age of thirteen?

   If this has happened:
   - Did you ever think you might be killed or seriously injured as a result of the event? (yes/no)
   - Did you experience intense fear, helplessness or horror when the event happened? (yes/no)

- Does the event still affect you? (yes/no)
- Do you still think about the event? (yes/no)
- Do you avoid thinking about the event? (yes/no)
- Please use the space below to give a brief indication of the severity of the event (e.g. touched by someone without your permission, rape involving penetration, etc)

4. Before you were age eighteen, did anyone ever use pressure, coercion, or non-physical threats to have sexual contact with you?

   If this has happened:
   - Did you ever think you might be killed or seriously injured as a result of the event? (yes/no)
   - Did you experience intense fear, helplessness or horror when the event happened? (yes/no)
   - Does the event still affect you? (yes/no)
   - Do you still think about the event? (yes/no)
   - Do you avoid thinking about the event? (yes/no)
   - Please use the space below to give a brief indication of the severity of the event (e.g. touched by someone without your permission, rape involving penetration, etc)

5. At any other time in your life, has anyone ever used physical force or threat of force to make you have some type of unwanted sexual contact with them?

   If this has happened:
   - Did you ever think you might be killed or seriously injured as a result of the event? (yes/no)
   - Did you experience intense fear, helplessness or horror when the event happened? (yes/no)
   - Does the event still affect you? (yes/no)
   - Do you still think about the event? (yes/no)
   - Do you avoid thinking about the event? (yes/no)
   - Please use the space below to give a brief indication of the severity of the event (e.g. touched by someone without your permission, rape involving penetration, etc)

6. At any time in your life, has anyone (including family members or friends) ever attacked you with a gun, knife or some other weapon, regardless of whether you ever reported it?

   If this has happened:
   - Did you ever think you might be killed or seriously injured as a result of the event? (yes/no)
   - Did you experience intense fear, helplessness or horror when the event happened? (yes/no)
   - Does the event still affect you? (yes/no)
   - Do you still think about the event? (yes/no)
   - Do you avoid thinking about the event? (yes/no)
   - Please use the space below to give a brief indication of the severity of the event (e.g. threatened with gun, stabbed by someone, etc)

7. At any time in your life, has anyone (including family members or friends) ever attacked you without a weapon, but with the intent to kill or seriously harm you?

   If this has happened:
   - Did you ever think you might be killed or seriously injured as a result of the event? (yes/no)
   - Did you experience intense fear, helplessness or horror when the event happened? (yes/no)
   - Does the event still affect you? (yes/no)
   - Do you still think about the event? (yes/no)
   - Do you avoid thinking about the event? (yes/no)
   - Please use the space below to give a brief indication of the severity of the event (e.g. cuts and bruises, near death in hospital, etc)

8. Have you ever experienced (or seen) any other events that were life threatening, caused serious injury, or were highly disturbing or distressing?

   If this has happened:
   - Did you ever think you might be killed or seriously injured as a result of the event? (yes/no)
   - Did you experience intense fear, helplessness or horror when the event happened? (yes/no)
   - Does the event still affect you? (yes/no)
   - Do you still think about the event? (yes/no)
   - Do you avoid thinking about the event? (yes/no)
   - Please use the space below to give a brief indication of the severity of the event.

TIME USE INTERVIEW

EMPLOYMENT

1. (a) Did you do any paid work in the last month, either as an employee or self-employed?

YES → GO TO QU 4
NO → ASK b

(b) Have you been on a government scheme for employment training?

YES → DETAILS

NO → GO TO QU 2

2. (a) Did you have a job or business you were away from?

YES → ASK b
NO → GO TO QU 3

(b) Why were you away? (Then ask QU 4 for typical work pattern when not away)

Holiday	
Sickness	
Studying	
Maternity/paternity leave	
Other reason (please state)	

3. (a) Did you do any unpaid work for any business that you or a relative own?

YES → GO TO QU 4
NO → ASK b

(b) Have you ever had a paid job?

YES → ASK c & questions 4-7 for **most recent** paid job
NO → GO TO QU 8

(c) When did you leave your last paid job?

4. What was your main job in the last month/most recent period of paid work?

What do/did you mainly do in your job? (check special qualifications, managerial duties, etc)

5. How many hours a week do you usually work in your main job or business? Include any overtime. How many hours have you worked in the last month?

6. What was your take-home monthly pay after all deductions the last time you were paid?

1	Less than £215	
2	£215 to less than £435	
3	£435 to less than £870	
4	£870 to less than £1305	
5	£1305 to less than £174	
6	£1740 to less than £2820	
7	£2820 to less than £3420	
8	£3420 to less than £3830	
9	£3830 to less than £4580	
10	£4580 to less than £6670	
11	£6670 or more	

7. In the last month, did you do any other paid work or have any other paid job or business, in addition to the one you have just told me about?

YES → DETAILS (e.g. how many, number of hours, type of job, wages)

NO → IF NO PAID WORK AT ALL IN LAST MONTH, GO TO QU 8
IF CURRENTLY WORKING, GO TO QU 11

8. Thinking of the last month, have you been looking for any kind of paid work government training schemes?

YES → ASK QU 9
NO → GO TO QU 10

9. In the last month, did you do any of these things?

Visited a Jobcentre/Jobmarket or Training and Employment Agency Office?	
Visited a Jobclub?	
Had your name on the books of an employment agency?	
Advertised for jobs in newspapers, etc?	
Looked for advertisements in newspapers, etc?	
Answered advertisements in newspapers, etc?	
Applied directly to employers?	

Asked friends, relatives, colleagues or trade unions about jobs?	
Waited for the results of a job application?	
Been to an interview?	
Done anything else to find work? Please state.	

How much time did you spend doing this?

10. May I just check, what was the main reason you did not look for work in the last month?

Waiting for the results of a job application/being assessed by training agent?	
Student?	
Looking after the family home?	
Temporarily sick or injured?	
Long-term sick or disabled?	
Believe no jobs available?	
Not yet started looking?	
Any other reason? Please state.	

11. Are you at present receiving any state benefits in your own right or on behalf of anyone in your household? If so, which ones? (show list)

EDUCATION AND TRAINING

1. (a) Do you have any qualifications from school, college or university, connected with work or from government schemes?

YES	→	ASK b onwards
NO	→	GO TO QU 2
Don't know	→	GO TO QU 2

(b) Which qualification do you have, starting with the highest qualification (show list)?

(c) When did you last study for any qualifications?

2. Are you studying for any qualifications at the moment (show list)?

YES → DETAILS (e.g. what, where, full/part time, hours, etc)

1	Degree level qualification including graduate membership of a professional institute or PGCE or higher (include undergraduate and postgraduate degrees)	
2	Diploma in higher education	
3	HNC/HND	
4	ONC/OND	
5	BTEC, BEC or TEC	
6	SCOTVEC, SCOTEC or SCOTBEC	
7	Teaching qualification excluding PGCE	
8	Nursing or other medical qualification not yet mentioned?	
9	Other higher education qualification below degree level	
10	A-level or equivalent	
11	SCE highers	
12	NVQ/SVQ	
13	GNVQ/GSVQ	
14	AS-level	
15	Certificate of sixth year studies (CSYS) or equivalent	
16	O-Level or equivalent	
17	SCE Standard or Ordinary (O) grade	
18	GCSE	
19	CSE	
20	RSA	
21	City and Guilds	
22	YT certificate/YTP	
23	Any other professional or vocational qualification or foreign qualifications (e.g. apprenticeship)	
666	Don't know	

NO → GO TO QU 3

3. (a) In the last month, have you been on any taught courses or undertaken learning of any of the following sorts:

Taught courses meant to lead to qualifications (even if you did not obtain them)	
Taught courses designed to help you develop skills that you might use in a job	
Courses or instruction or tuition in driving, in playing a musical instrument, in an art or craft, in a sport or in any practical skill	
Evening classes	
Learning which involved working on your own from a package of materials provided	

IF YES TO ANY OF THE ABOVE → ASK b
IF NONE OF THE ABOVE → GO TO QU 4

(b) How many taught courses have you been involved in in the last month?

4. In the last month, have you studied or received training in any of these ways:

Studied for a qualification without taking part in a taught course	
Received supervised training while you were actually doing a job	
Spent time keeping up-to-date with developments in the type of work you do without taking part in a taught course (e.g. by reading books, manuals journals, or attending seminars)	
Spent time deliberately truing to improve your knowledge about anything or teach yourself a new skill without taking part in a taught course	

IF YES TO ANY OF THE ABOVE → DETAILS (e.g. what, number of occasions in last month, length of time, etc)

IF NONE OF THE ABOVE → GO TO QU 6

5. On how many occasions in the last month did you spend time studying at home outside of teaching sessions?

How long did you study for the last time you did any? How long on average do you normally study for?

6. Thinking of the last month, have you been looking for any kind of education/course?

YES → DETAILS (what, how much time, etc)

NO → GO TO VOLUNTARY WORK

VOLUNTARY WORK

Voluntary work is work that people may do for which they are not paid, except perhaps for expenses.

1. Have you done any voluntary work through a group or on behalf of an organisation at any time during the last month?

   YES → DETAILS AND ASK 2 ONWARDS

   NO → GO TO 'LEISURE ACTIVITIES'

2. How many different times did you do this work during the last month?

3. How long did you work for, the last time you did this? How long do you normally spend doing this?

LEISURE ACTIVITIES

1. I am now going to ask some questions about things that some people do in their spare time. For each activity that I mention could you please tell me whether of not you have done this in the last month, AND how often?

ACTIVITY	NUMBER OF TIMES	AMOUNT OF TIME
Been to cinema, film society or club		
Been to a sports event as a spectator		
Been to a play, musical or pantomime		
Been to the opera		
Been to a concert or performance of classical music of any kind		
Been to any other gig or live music performance (e.g. pop, rock or jazz concert, blues or folk club)		
Been to the ballet or to a modern/contemporary dance performance		
Been to a museum or art gallery		
Been to an historic house, castle or other heritage site or building		
Been to a library		
Been out to eat or drink at a café, restaurant, pub or wine bar		

Been to a shopping centre, or mall, apart from regular shopping for food and household items		
Been to a car boot sale, antiques fair or craft market or similar apart from regular shopping for food and household items		
Been to a theme park, fairground, fair or carnival		
Been to a zoo, wildlife reserve, aquarium or farm park		
Been to some other place of entertainment (e.g. dance, club, bingo, casino)		
Been on any other outdoor trips (including going to places of natural beauty, picnics, going for a drive or going to the beach)		
Other (please state)		

2. On these cards is a list of sports and physical activities. Could you please tell me whether or not you took part in any of them in the last month AND how often?

ACTIVITY	NUMBER OF TIMES	AMOUNT OF TIME
Swimming or diving		
Cycling		
Indoor or outdoor bowls		
Tenpin bowling		
Keep fit, aerobics, yoga, dance exercise		
Martial arts		
Weight training or weight lifting		
Gymnastics		
Snooker, pool or billiards		
Darts		
Rugby		
Football		
Gaelic sports		
Cricket		
Hockey		
Netball		
Tennis		
Badminton		
Squash		
Basketball		
Table tennis		
Track and field athletics		
Jogging, cross country, road running		
Angling/fishing		
Yachting or dinghy sailing		
Canoeing		
Windsurfing/board sailing		
Ice-skating		
Curling		
Golf		
Skiing		

Horse riding		
Climbing/mountaineering		
Motor sports		
Shooting		
Walking or hiking for 2 miles or more (recreationally)		
Volleyball		
Other (please state)		

3. How much time do you spend socialising? How many occasions in the last month have you seen friends, either visiting them or receiving visitors? How much time did you tend to spend socialising on each occasion on average?

4. How much time do you spend resting, i.e. taking time out and doing nothing (but not sleeping)? How much time do you spend watching television or listening to the radio? Average for last month.

HOBBIES

1. Do you have any hobbies? Show list of examples.

2. How much time do you spend on hobbies each week (on average)?

CHILD CARE

1. Are you responsible for the care of any children?

YES → ASK 2
NO → GO TO 'HOUSEWORK AND CHORES'

2. How many? How old are they?

3. How much time do you spend doing things with your children? Ask individual to include checklist in their estimate (show card).

HOUSEWORK AND CHORES

How much time do you spend doing housework and chores per week? Ask individual to include checklist in their estimate.

Food management and preparation	
Cleaning, dusting, vacuuming, washing dishes	
Food shopping	
Washing	
Gardening	
DIY and repairs	
Other (please state)	

OTHER ACTIVITIES

1. How much time do you spend sleeping per day (on average)? This includes sleep at night time and naps during the day. Ask about good and bad days.

2. Do you spend time doing any activities not already asked about? Get weekly average.

TIME USE INTERVIEW SCORE SHEET

General Codings:

0 = NO **1 = YES**
666 = NO ANSWER/MISSING **999 = NOT APPLICABLE**

EMPLOYMENT

- Is paid work in the last month present or absent?

  ☐ Present = 'YES' response to Question 1 (a), 1 (b), or Question 2

  Absent = 'NO' response to Question 1 or 2

  NB. 'YES' response to Question 3 (a) should be coded as voluntary work

- Type of work/job title (Question 4)

  ☐

- Salary band (Question 6)

  ☐ Code 1-11 or 666/999 (see interview)

- Hours per week in paid employment over the last month

  ☐ NB. This should be calculated by adding all hours paid employment (from Questions 5 and 7) and dividing by 4 to get a weekly average. This includes time spent on government training schemes.
  e.g. if someone generally gets one paid day of work per month, this is taken as 2 hours per week

- Active searching for work?

  ☐ Present = 'YES' response to Question 8

  Absent = 'NO' response to Question 8

  ☐ Number of different work searching activities (taken from Question 9)

- Has paid work ever been present? **(NB: Only code these items if no current paid work)**

  ☐ Present = 'YES' response to Question 3 (b)

  Absent = 'NO' response to Question 3 (b)

  If yes:

  ☐ Number of weeks since last worked (Response to Question 3c)

  ☐ Number of hours per week worked in last job (Response to Question 5)

  What was the last paid job? (Question 4)

  ☐

  Salary band of your last job? (Question 6)

  ☐ Code 1-11 or 666/999 (see interview)

EDUCATION

Highest level of educational qualification already achieved (Question 1b):

☐ Code 1-23 or 666/999 (see interview)

Other educational or vocational qualifications already achieved (Question 1b):

Enter codes:

- Is current education present or absent?

  ☐ Present = any 'YES' response to Questions 2, 3 or 4

  Absent = 'NO' responses to Questions 2, 3 and 4

  Hours per week in education over the last month

  ☐ NB. This should be calculated by adding all hours spent in education (from Questions 2, 3 4 and 5) and dividing by 4 to get a weekly average.

- Number of different courses taken part in over last month

  ☐ NB. Taken from Questions 2, 3, 4, 5

- Active searching for education?

  ☐ Present = 'YES' response to Question 6

  Absent = 'NO' response to Question 6

VOLUNTARY WORK

- Is voluntary work present or absent?

  ☐ Present = 'YES' response to Question 1 or Question 3 (a) from Employment section

  Absent = 'NO' response to Question 1

- Hours per week spent in voluntary work over the last month

  ☐ NB. This should be calculated by multiplying number of times (Question 2) by average length of time (Question 3) and dividing the result by 4 to get a weekly average.

LEISURE ACTIVITIES

- Are leisure activities present or absent (taken from Question 1)

  ☐

- Hours per week spent in leisure activities over the last month

  NB. This should be calculated by multiplying number of times by average length of time for each activity. Then sum all of these and divide the result by 4 to get a weekly average.

- Number of leisure activities taken part in over last month

  NB. Taken from Question 1

- Are sport/physical activities present or absent (taken from Question 2)

- Hours per week spent in sport/physical activities over the last month

  NB. This should be calculated by multiplying number of times by average length of time for each activity. Then sum all of these and divide the result by 4 to get a weekly average.

- Number of sport/physical activities taken part in over last month

  NB. Taken from Question 2

- Hours per week over last month spent:

  Socialising Resting

HOBBIES

- Are hobbies present or absent?

- Hours per week spent on hobbies over the last month

  NB. This should be calculated by multiplying number of times by average length of time for each activity. Then sum all of these and divide the result by 4 to get a weekly average.

- Number of hobbies taken part in over last month

CHILDCARE

- Childcare

  Applicable = 1 Non-applicable = 0

- Hours per week spent on childcare

NB. Taken from Question 3

HOUSEWORK AND CHORES

- Hours per week spent on housework and chores

NB. Taken from estimate of average time including items from checklist in estimate

OTHER ACTIVITIES

- Hours spent per day sleeping (Question 1)

- Hours per week spent on other activities over the last month (Question 2)

NB. This should be calculated by multiplying number of times by average length of time for each activity. Then sum all of these and divide the result by 4 to get a weekly average.

- Number of other activities taken part in over last month (Question 2)

APPENDIX F

Mann-Whitney U and regression tables for additional trauma analyses (reported in Chapter 5)

Table F1

Differences in Schizotypy, Emotional Distress and Schematic Beliefs between Individuals who have and have not been Exposed to Severe Bullying

	Exposed to trauma – Mean (*SD*)		*U*	*p*
	Yes (*N* = 75)	No (*N* = 672)		
Schizotypal Symptoms				
- Total	12.60 (11.02)	9.25 (8.94)	20807.50	.01
- Social Anxiety	6.07 (5.82)	4.29 (5.10)	20525.50	.01
- Paranoia	3.55 (4.39)	2.79 (3.53)	23121.00	.23
- Anomalous Experiences	2.99 (4.13)	2.17 (3.29)	22687.50	.14
Anxiety	8.95 (8.46)	5.61 (6.25)	19625.00	.002
Depression	13.96 (13.48)	9.76 (9.75)	21560.00	.05
BCSS				
- Negative Self	6.12 (5.08)	4.06 (4.13)	18667.00	<.001
- Positive Self	12.00 (5.48)	13.19 (4.78)	22305.50	.13
- Negative Other	6.68 (4.94)	4.68 (4.07)	18977.00	.001
- Positive Other	10.88 (4.03)	12.65 (3.78)	19134.00	.001
Interpersonal Sensitivity	97.35 (17.06)	93.53 (15.27)	20890.00	.03

Table F2

Differences in Schizotypy, Emotional Distress and Schematic Beliefs between Individuals who have and have not been Exposed to Physical Attack

	Exposed to trauma – Mean (*SD*)		U	p
	Yes (*N* = 80)	No (*N* = 667)		
Schizotypal Symptoms				
- Total	11.55 (9.19)	9.36 (9.20)	21842.50	.01
- Social Anxiety	5.41 (5.15)	4.36 (5.20)	22786.00	.03
- Paranoia	3.35 (3.76)	2.81 (3.62)	23769.50	.10
- Anomalous Experiences	2.79 (3.73)	2.18 (3.34)	24240.00	.16
Anxiety	8.35 (8.67)	5.66 (6.22)	22794.50	.04
Depression	11.43 (12.34)	10.04 (9.98)	26472.00	.96
BCSS				
- Negative Self	5.31 (4.99)	4.15 (4.17)	23547.50	.11
- Positive Self	12.29 (5.39)	13.17 (4.79)	24684.00	.33
- Negative Other	6.04 (4.42)	4.74 (4.16)	21469.50	.01
- Positive Other	11.63 (4.11)	12.58 (3.79)	229030.00	.05
Interpersonal Sensitivity	95.50 (17.06)	93.72 (15.29)	24953.50	.38

Table F3

Differences in Schizotypy, Emotional Distress and Schematic Beliefs between Individuals who have and have not been Exposed to Child Sex Abuse

	Exposed to trauma – Mean (*SD*)		*U*	*p*
	Yes (*N* = 23)	No (*N* = 724)		
Schizotypal Symptoms				
- Total	12.70 (8.44)	9.49 (9.23)	6113.00	.03
- Social Anxiety	6.48 (6.00)	4.41 (5.16)	6406.50	.06
- Paranoia	4.04 (3.38)	2.83 (3.64)	6134.00	.03
- Anomalous Experiences	2.17 (3.05)	2.25 (3.40)	8235.00	.93
Anxiety	12.09 (10.23)	5.75 (6.34)	5309.00	.003
Depression	19.04 (13.99)	9.90 (10.00)	5419.50	.005
BCSS				
- Negative Self	7.65 (5.58)	4.16 (4.19)	5027.50	.001
- Positive Self	10.52 (6.05)	13.15 (4.80)	6232.00	.05
- Negative Other	6.04 (3.81)	4.84 (4.21)	6324.50	.05
- Positive Other	10.52 (4.12)	12.54 (3.82)	6134.50	.04
Interpersonal Sensitivity	106.30 (12.60)	93.51 (15.41)	4308.50	<.001

Table F4

Differences in Schizotypy, Emotional Distress and Schematic Beliefs between Individuals who have and have not been Exposed to Sex Abuse Occurring Later in Life

	Exposed to trauma – Mean (*SD*)		*U*	*p*
	Yes (*N* = 71)	No (*N* = 676)		
Schizotypal Symptoms				
- Total	10.72 (9.99)	9.47 (9.13)	22207.00	.30
- Social Anxiety	4.90 (5.77)	4.43 (5.14)	23574.00	.80
- Paranoia	3.42 (4.20)	2.81 (3.57)	21684.00	.17
- Anomalous Experiences	2.39 (3.31)	2.23 (3.40)	22923.50	.52
Anxiety	7.35 (7.37)	5.80 (6.47)	21145.00	.11
Depression	12.30 (12.83)	9.96 (9.93)	22861.50	.55
BCSS				
- Negative Self	4.92 (4.77)	4.20 (4.22)	21980.50	.29
- Positive Self	12.55 (5.53)	13.13 (4.79)	22937.00	.62
- Negative Other	4.99 (4.09)	4.87 (4.22)	23133.00	.70
- Positive Other	11.56 (4.26)	12.57 (3.78)	21211.50	.13
Interpersonal Sensitivity	97.11 (15.43)	93.57 (15.46)	21141.00	.11

Table F5

Summary of Hierarchical Regression Analysis Investigating the Prediction of Social Anxiety Schizotypal Symptoms by Exposure to Severe Bullying, Before and After Controlling for Emotion and Schema Variables (N = 746)

Variable	*B*	*SE B*	β
Step 1			
Constant	4.25	0.20	
Severe Bullying	1.68	0.63	.10**
Step 2			
Constant	-4.19	1.37	
Severe Bullying	0.35	0.53	.02
Anxiety	0.16	0.03	.20***
Depression	-0.01	0.02	-.02
Negative Self Schema	0.12	0.06	.10†
Positive Self Schema	-0.15	0.04	-.14**
Negative Other Schema	0.04	0.04	.03
Positive Other Schema	-0.02	0.05	-.02
Interpersonal Sensitivity	0.10	0.01	.29***

Note. $R^2 = .01$, $p = .008$ for Step 1; $\Delta R^2 = .32$, $p = 0.001$ for Step 2

*$p = <.05$, **$p = <.01$, ***$p = <.001$, $^{\dagger}p = <.10$

Table F6

Summary of Hierarchical Regression Analysis Investigating the Prediction of Social Anxiety Schizotypal Symptoms by Exposure to Physical Attack, Before and After Controlling for Emotion and Schema Variables (N = 746)

Variable	*B*	*SE B*	β
Step 1			
Constant	4.30	0.20	
Physical Attack	1.12	0.61	.07†
Step 2			
Constant	-4.17	1.37	
Physical Attack	0.17	0.51	.01
Anxiety	0.16	0.03	.20***
Depression	-0.01	0.02	-.02
Negative Self Schema	0.12	0.06	.10†
Positive Self Schema	-0.15	0.04	-.14**
Negative Other Schema	0.04	0.04	.03
Positive Other Schema	-0.02	0.05	-.02
Interpersonal Sensitivity	0.10	0.01	.29***

Note. $R^2 = .01$, $p = .068$ for Step 1; $\Delta R^2 = .32$, $p < 0.001$ for Step 2

*$p = <.05$, **$p = <.01$, ***$p = <.001$, †$p = <.10$

Table F7

Summary of Hierarchical Regression Analysis Investigating the Prediction of Social Anxiety Schizotypal Symptoms by Exposure to Child Sex Abuse, Before and After Controlling for Emotion and Schema Variables (N = 746)

Variable	*B*	*SE B*	β
Step 1			
Constant	4.35	0.20	
Child Sex Abuse	2.13	1.09	.07*
Step 2			
Constant	-4.19	1.37	
Child Sex Abuse	-1.00	0.91	-.03
Anxiety	0.16	0.03	.20***
Depression	-0.01	0.02	-.02
Negative Self Schema	0.12	0.06	.10†
Positive Self Schema	-0.15	0.04	-.14**
Negative Other Schema	0.04	0.04	.03
Positive Other Schema	-0.02	0.05	-.02
Interpersonal Sensitivity	0.10	0.01	.29***

Note. $R^2 = .01$, $p = .05$ for Step 1; $\Delta R^2 = .32$, $p = <.001$ for Step 2

*$p = < .05$, **$p = < .01$, ***$p = < .001$, $^{\dagger}p = <.10$

Table F8

Summary of Hierarchical Regression Analysis Investigating the Prediction of Paranoid Schizotypal Symptoms by Exposure to Child Sex Abuse, Before and After Controlling for Emotion and Schema Variables (N = 746)

Variable	*B*	*SE B*	β
Step 1			
Constant	2.79	0.13	
Child Sex Abuse	1.25	0.76	.06†
Step 2			
Constant	-1.68	0.94	
Child Sex Abuse	-0.80	0.63	-.04
Anxiety	0.09	0.02	.16***
Depression	0.02	0.02	.07
Negative Self Schema	0.15	0.04	.18***
Positive Self Schema	0.02	0.03	.03
Negative Other Schema	0.19	0.03	.22***
Positive Other Schema	-0.08	0.03	-.09*
Interpersonal Sensitivity	0.03	0.01	.14***

Note. $R^2 = .004$, $p = .10$ for Step 1; $\Delta R^2 = .34$, $p = <.001$ for Step 2

*$p = <.05$, **$p = <.01$, ***$p = <.001$, †$p = <.10$