Brief Cognitive Assessment and Theory of Mind in First Episode Psychosis, and Their Relation to Community Functioning

Allison Whitty

D. Clin. Psy. thesis (Volume 1), 2007

University College London.

UMI Number: U592461

UMI

Dissertation Publishing

ProQuest

Overview

This thesis is divided into three parts. The Review Paper (part 1) presents a systematic review of cognitive deficits in first episode psychosis. It also examines the neurological basis for these deficits and their relation to social functioning. The Review Paper highlights the need for more research to explore the possible link between cognitive deficits and social and community functioning in first episode psychosis.

The Empirical Paper (part 2) reports on a study I conducted, which examined the relationship between cognitive and community functioning in those with a first episode of psychosis. Cognitive function was assessed by the measures that comprise the Brief Cognitive Assessment (Trail Making Test, Hopkins Verbal Learning Test and Verbal Fluency) and measures of Theory of Mind (Hints Task, Picture Sequencing Task and Theory of Mind Stories). A global cognition score calculated from the BCA and the Hints Task were positively and significantly correlated with community functioning, as assessed by the Multnomah Community Ability Scale.

Finally, the Critical Appraisal (part 3) reflects on the process of conducting this research, including recruitment issues and the assessment measures used. The strengths and weaknesses of the study are elaborated, and recommendations for future studies conclude the thesis.

Table of Contents

Appendices

Tables

Acknowledgements

Firstly, I would like to sincerely thank Graham Pickup for his expertise and support. I could not have wished for a better supervisor. I am indebted to the patients and staff from the early intervention in psychosis services who gave up their time and energy to make this research possible. I would like to thank Kirk, my family and friends for propping me up throughout. Finally, I would also like to thank my course mates for their encouragement and support.

Part 1: Review Paper

A Systematic Review of Cognitive Deficits in First Episode

Psychosis, their Neurological Basis and their Relation to Social

Functioning

6

Abstract

This review examines the research evidence for cognitive deficits in first episode psychosis (FEP), their neurological basis and their relation to social functioning. Twenty-six papers were identified by searching computer databases (e.g. PsycINFO, the Cochrane Library, etc.), hand searching relevant journals, and searching reference sections of key papers. The papers identified were then categorised into 3 subject areas: neurological, cognitive deficits, and social functioning. FEP patients showed an excess of neurological soft signs and differences in brain morphology compared to healthy controls. While there appears to be clear evidence to support a generalised cognitive deficit in FEP, there may also be a sub-group of patients who have specific impairments in some aspects of cognitive function. There appears to be little or no evidence of an association between cognitive deficits and social functioning in FEP, though this area requires further research in the future.

1.1 Introduction

The purpose of this review is to examine the research evidence for cognitive deficits in first episode psychosis (FEP), their neurological basis and relation to social functioning. Background information about psychosis is drawn together to establish a context for the review. Following a review of the evidence, recommendations for future research in FEP are discussed. For the purpose of this review, I shall use the term 'psychosis' except where studies have specifically used the term 'schizophrenia'.

1.1.1 What is psychosis?

Psychosis was first described as "dementia praecox" by Kraepelin in the fifth edition of his *Textbook of Psychiatry* (Kraepelin, 1896). This Latin term, meaning "senility of the young", reflected a variety of symptoms which Kraepelin noted first appeared in adolescence or early adulthood. These symptoms included catatonic postures, unusual perceptual experiences, attentional difficulties, inappropriate emotional responses and irrational beliefs. As the name suggests, a common underlying feature of dementia praecox was deterioration in mental functions. Kraepelin saw this process as neurodegenerative, whereby patients became mentally disabled, led unproductive lives and never recovered (Bentall, 2003). In the century or so since this illness was first described, advances in science have led to different opinions regarding the trajectory for dementia praecox, or "schizophrenia" as it known today. However, deterioration in mental functions remains a core feature of the illness (Wing, 1978).

"Psychosis" was a term used by Jaspers to differentiate a cluster of illnesses that were understood to stem from biological abnormalities (e.g. schizophrenia and manic depression) as opposed to illnesses that were believed to emanate from personality and

life experiences which were known as "neuroses" (e.g. anxiety and depression where contact with reality is maintained) (Bentall, 2003). Today the terms "schizophrenia" and "psychosis" are used interchangeably, and psychosis seems to be used more frequently than schizophrenia in the UK. The signs and symptoms associated with psychosis are varied in their manifestations and course (Frith, 1992). Some common symptoms include hearing voices in the second or third person, delusions of control or reference, paranoia, thought insertion, withdrawal and broadcast (Frith, 1992). These symptoms are classified as "positive" symptoms because they represent the presence of abnormal experiences. Other symptoms include poverty of speech, psychomotor retardation, flat affect and anhedonia (Sharma, 2000). These symptoms are classed as "negative" symptoms as they characterise the absence of behaviour or function. Positive symptoms usually occur early in psychosis and generally respond well to medication. Negative symptoms often occur in the later stage of the illness and may be predictive of poor long-term outcome (McGlashan & Fenton, 1992).

1.1.2 Prevalence of psychosis

In the year 2000, 1 in 200 people had a psychotic disorder in the UK (Office for National Statistics, 2006). Despite the low frequency of psychotic disorders in comparison to other conditions (e.g. neurotic disorders), people with psychotic disorders are more likely to be represented in services because of the nature and severity of the illness. In 2000, 83% of those with psychotic disorder were receiving pharmacological treatment, and 40% of these were receiving psychological treatment (Office for National Statistics, 2006).

1.1.3 Causes of psychosis – nature or nurture?

The exact cause of psychosis remains unknown. The importance of genetic factors in the aetiology of schizophrenia has been known since the 1970s. In a study of family risk of developing schizophrenia, the risk for siblings of those with schizophrenia was reported to be 11.5%. Second degree relatives showed a risk of approximately 4% (Kallman, 1938). This was one of the earliest studies, and the pooled results of other family studies since then suggest a slightly lower risk across all relatives of those with schizophrenia. The children of mothers with schizophrenia were reported to have the same risk as the children of fathers with schizophrenia, while the children of two parents with schizophrenia were reported to have a risk of about 40% (Slater & Cowie, 1971). The incidence of schizophrenia in monozygotic twins was reported as being much greater than in dizygotic twins. Kallmann (1946) reported overall concordance rates of 69% for monozygotic twins and 10.3% for dizygotic twins. The exact percentages vary between studies in the literature perhaps because of the heterogeneity and severity of the illness, and the different diagnostic criteria used.

In an attempt to disentangle environmental and genetic influences, some adoption studies were conducted. Rosenthal et al. (1968) compared the children of biological parents with schizophrenia who were raised by adoptive parents with a matched control group of adoptees whose parents had no history of psychiatric illness. They found that the rate of spectrum diagnoses was significantly increased in the offspring of affected parents over that of the offspring of control parents. This suggested that genetics plays a more powerful role in the genesis of schizophrenia than environment. Relapse of psychosis has been shown to be higher in patients living in

homes where critical comments, hostility and over involvement are common (Vaughn & Leff, 1976). This is known as "expressed emotion".

1.1.4 Causes of psychosis - neurological

When schizophrenia was first described in Kraepelin's time, its cause was understood to stem from neuropathology. Even though no abnormalities had been observed in the brains of patients with schizophrenia, neuropathologists continued to search diligently (Frith, 1992). As time passed, with the lack of concrete evidence of neuropathology and the advent of alternative theories, many began to lose faith in the presence of a brain abnormality associated with schizophrenia. However, this view was challenged with the discovery of antipsychotic drugs and the development of quantitative rather than qualitative studies of brain structure (Frith, 1992).

Many antipsychotic drugs have been developed since the 1950s, and although they all differ in terms of their basic chemistry, their therapeutic effectiveness appears to lie in blocking dopamine receptors (Seeman, Lee, Cahu-Wong, & Wong, 1976). The precise role of dopamine in schizophrenia remains unclear; however, there is evidence to suggest that the dopaminergic receptors in the brains of schizophrenic patients are more sensitive than in healthy controls (Owen et al., 1978). Although stimulation of the dopamine system has been found to produce symptoms of schizophrenia (Connell, 1958), there is no evidence to suggest that there is too much dopamine production in the brains of those suffering from schizophrenia (Crow et al., 1984).

The advent of Computerised Axial Tomography (CAT) in the 1970s led to increased interest in the brain structure of those with psychosis. The measurement of the cross-sectional area of the lateral ventricles showed them to be significantly enlarged in

11

a group of schizophrenic patients relative to controls (Johnstone, Crow, Frith, Husband, & Kreel, 1976). This is also common in Alzheimer's disease; however, unlike schizophrenia the ventricles become enlarged in Alzheimers disease as tissue is lost (Frith, 1992). The ventricles in schizophrenia are particularly enlarged within the temporal lobes on the left side of the brain (Crow et al., 1989), while adjacent parts of the cortex such as the hippocampus and parahippocampal gyrus have been shown to be reduced in size (Bogerts, Meartz, & Schofield-Bausch, 1985). The enlarged ventricles have not been shown to become progressively larger over time (Gattaz, Kohlmeyer, & Gasser, 1991), and some researchers have found enlarged ventricles before the onset of symptoms (O'Callaghan et al., 1988; Weinberger, 1988). This suggests that brain abnormalities may precede the onset of illness, and that schizophrenia is not a neurodegenerative disorder (Frith, 1992). Despite these findings, there is much overlap with normal subjects, which cautions against using the size of these brain structures alone as "markers" for schizophrenia (Frith, 1992). In summary, dopamine appears to have a role in psychosis, though its precise mechanism remains uncertain. Enlarged ventricles appear to be a feature of psychosis and may be present before the onset of the illness.

1.1.5 Psychological model of psychosis

It is now widely accepted that psychotic disorders are a heterogeneous group of disorders that lie on a continuum with normality (Fowler, Garety, & Kuipers, 1995). Psychological therapies, in particular Cognitive Behavioural Therapy (CBT), are becoming increasingly recognised as an effective part of treatment for psychosis. Cognitive theories of psychosis have mainly been drawn from Beck's theory of

emotional disorders (Beck, 1976). As in the case of emotional disorders, individuals with psychosis frequently present to therapy because they are in distress. CBT focuses on the underlying thoughts, beliefs and behaviours that are causing and maintaining this distress (Nelson, 2005). To illustrate a CBT model of psychosis, I shall focus on Freeman and Garety's model of delusions (2004).

A delusion may be defined as a strongly held belief not subject to reason or contradictory evidence and not explained by a person's usual cultural concepts (Freeman & Garety, 2004). In a CBT model, the origins of delusions are broadly viewed within a stress vulnerability framework (Freeman & Garety, 2004). It is hypothesised that a stressor precipitates the formation of a delusion, and this stressor causes arousal usually in the form of anxiety (Freeman, Garety, Kuipers, Fowler, & Bebbington, 2002). In individuals vulnerable to developing psychosis, this arousal may be experienced as an internal anomalous experience causing ideas of reference, depersonalisation and perceptual abnormalities. These anomalous internal experiences initiate a search for meaning, which is a critical point in the development of psychosis. The individual's core beliefs and any potentially significant information from the external environment may be drawn into the search for meaning, irrespective of whether the information is positive, negative, neutral or ambiguous (Freeman & Garety, 2004). Several cognitive biases are thought to contribute to the formation of a delusion. Studies have shown that many individuals with psychosis tend to seek less information in their decision-making and therefore 'jump to conclusions' (Garety, Hemsley, Wessely, 1991). Another cognitive bias common in delusions is 'personalisation', whereby the individual attaches personal significance to external events. Poor flexibility in considering alternative

13

explanations may also promote delusion formation (Garety et al., 1997). The interplay between cognitive processes and external events results in the formation of a delusion, the strength of conviction of which depends on the success of maintaining processes.

Delusions are maintained by a number of cognitive and behavioural processes. The individual may experience negative reinforcement when a delusion is formed that is consistent with pre-existing beliefs and explains an internal anomolous experience. Cognitive biases maintain persecutory delusions by obtaining confirmatory evidence consistent with pre-existing beliefs and ignoring disconfirmatory evidence. High levels of anxiety relating to the delusion may promote the use of safety behaviours that in turn prevent the individual from being exposed to disconfirmatory evidence (Freeman & Garety, 2004). When disconfirmatory evidence is experienced, it may be incorporated into the delusion system whereby it is believed that characteristics of the perpetrator and the situation prevented the occurrence of harm (Freeman & Garety, 2004). Threat related memory biases might aid recall of evidence thus supporting future appraisals (Freeman & Garety, 2004). The individual's behaviour towards others (e.g. hostility, withdrawal) may reinforce their delusions as others may withdraw, become hostile or stigmatise the individual. Social isolation may also maintain a delusion whereby the individual is unable to modify their thoughts based on communication with others (Freeman et al., 2002).

1.1.6 Summary

Psychosis was first documented in the literature over a hundred years ago. Although our understanding of psychosis has come a long way since then, the exact

cause(s) remain uncertain. Individuals with psychosis are highly represented in mental health services, at a large cost to the health service. Psychological therapies are becoming increasingly recognised as an important part of treatment in psychosis. Genetic predisposition and neurological abnormalities may be linked to the development of psychosis, while high expressed emotion in families may be implicated in the relapse of psychosis. Research has increasingly focused on the first episode of psychosis to help address the gaps in our understanding of this illness.

1.2.1 First episode psychosis (FEP)

There is much evidence to suggest that the early detection and treatment of psychosis results in rapid remission of the condition, better long-term outcomes for the individual and lower treatment costs (Birchwood et al., 1992; McGorry & Edwards, 1997). Before psychosis emerges, patients may experience prodromal symptoms (Aitchison, Meehan, & Murray, 1999). The prodromal phase may be described as the period from the first noticeable symptoms to the onset of actual psychotic symptoms (Beiser, Erikson, Fleming, & Iacono, 1993). These may include changed behaviours (e.g. social withdrawal, deterioration in social functioning, paranoia), changes in mood (e.g. mood swings, depression, anxiety), changes in cognition (e.g. odd or unusual ideas, difficulty concentrating) and somatic changes (e.g. loss of appetite and energy, reduced motivation) (Yung & McGorry, 1996).

Birchwood, McGorry and Jackson (1997) suggest that the early phase of psychosis is formative and predictive of long-term outcome. Prospective studies have shown that a substantial degree of social, psychological and biological deterioration occurs in the first two years after presentation, and then plateaus (Birchwood, Todd, &

Jackson, 1998). A research group in New York noted that patients with positive

psychotic symptoms who went for long periods without antipsychotic medication had a

slower and less complete recovery, and an increased risk of subsequent relapse, than

those who received prompt treatment (Loebel et al., 1992). In a study in the UK, the

delay of treatment was greater than a year in about one quarter of patients, and was

associated with increasing complications such as life-threatening behaviour and family

difficulties (Johnstone, Crow, Johnson, & Macmillan, 1986). Patients who took longer

than a year to access services showed a three-fold increase in relapse rate over the

following two years compared with those with a briefer duration of untreated psychosis.

Intervention after the plateau may be less successful than intervention earlier in the first

episode. A review of the literature by Wyatt (1991) led to the conclusion that a delay in

treating psychosis may lead to biological toxicity. Relapse, treatment resistance, and

episodes of untreated psychosis following first treatment may also contribute to this

proposed toxicity (Birchwood, McGorry, & Jackson, 1997).

1.2.2 Factors which delay treatment in FEP

Research groups in the USA (Loebel et al., 1992), Canada (Beiser et al., 1993),

Australia (McGorry, Edwards, Mihalopoulos, Harrigan, & Jackson, 1996) and Germany

(Hafner, Maurer, Loffler, & Riecher-Rossler, 1993) have reported treatment delays of an

average of one year between onset of psychotic symptoms and the initiation of

appropriate treatment. There are many factors which delay FEP patients' treatment for

psychosis. The first clinical presentation of psychosis differs greatly between patients,

which may make diagnosis difficult and delay early detection. FEP commonly occurs in

adolescence or early adulthood, and the signs and symptoms that occur may be

attributed to adolescent problems such as the stress of school or work and relationship difficulties. In many cultures there is stigma associated with mental illness, which may pose a barrier to seeking help. Some of the signs and symptoms that are displayed in the first episode may be similar to physical illnesses such as temporal lobe epilepsy, cerebral infection or trauma, and metabolic disorders (Aitchison et al., 1999). Alternatively, psychotic symptoms may be superimposed on pre-existing mental health problems such as personality disorder. Both prescribed and illicit drugs may also produce the kind of symptomatology reported in psychosis. In summary, the heterogeneous presentation of psychosis, age of onset, stigma and incorrect diagnosis by health professionals may delay treatment in FEP. Educating the community and health care professionals about psychosis may help to alleviate some of the barriers to treatment.

1.2.3 Services for FEP

Early intervention services (EIS) emerged from the need to treat young people in a user friendly way at the earliest possible stage after the onset of psychosis. Their aim is to provide a comprehensive and integrated hospital and community service with continued care throughout the critical period (2 years following entry into the programme) with the aim of reducing secondary morbidity (McGorry & Edwards, 1997). There is evidence that patients who received care from EIS had shorter durations of untreated psychosis, reduced inpatient treatment, reduced neuroleptic use, improvement in negative symptoms and better psychosocial functioning (McGorry & Edwards, 1997). In Australia the Early Psychosis Prevention and Intervention Centre (EPPIC) was shown to be cheaper and more cost effective than the pre-EPPIC system (McGorry & Edwards, 1997).

17

The first episode patient's initial experience of mental health services may predict future treatment compliance and help-seeking in the event of relapse (McGorry & Edwards, 1997). EISs need to cater to the needs of their client group, many of whom are in their teenage years, to make the service as attractive as possible to them. Some ways of catering to this client group might include adopting a more informal dress code among staff, meeting clients in places outside of the service such as cafes, keeping in touch with them via text messaging and decorating the service in a contemporary way. Those affected by FEP are often at an age where they may be concerned about how they appear to their peers. Aitchison, Meehan, and Murray (1999) recommend that treatment should maximise therapeutic benefit while minimising side-effects (e.g. tremors, tardive dyskinesia). The focus of care should be the re-integration of the patient back into their community with monitoring by services to prevent relapse (Aitchison et al., 1999). Psychological treatment should also be part of both the initial treatment package and continuing care (Drury, Birchwood, Cochrane, & Macmillan, 1996).

1.2.4 Cognition in FEP

Research suggests that cognitive difficulties have important implications for outcome in psychotic patients (Green, 1996). This is especially pertinent for those experiencing their first psychotic episode, which often occurs when post-secondary studies or occupational endeavours are a major concern (Townsend, Norman, Malla, Rychlo, & Ahmed, 2002). Advances in the development of antipsychotic medications have improved symptom reduction. However, the role of cognitive deficits in the aetiology of schizophrenia has received much less attention to date. There has been much debate in the literature regarding the nature, severity, and timing of onset of

cognitive deficits as well as the course they take. Some researchers have found evidence that cognitive deficits may pre-date the emergence of psychosis. Those at high risk of developing psychosis have been shown to have compromised cognitive performance on complex tasks involving visuospatial processing and memory (Brewer et al., 2005; Johnstone, Ebmeier, Miller, Owens, & Lawrie, 2005) and attention (Johnstone, Ebmeier, Miller, Owens, & Lawrie, 2005) before the full emergence of psychotic illness. An understanding of the neuropsychological deficits associated with FEP could have important clinical applications in the future, as these deficits may influence patients' recovery. Interventions aimed at reducing the impact of these deficits may reduce the long term social consequences of psychosis for individuals.

1.2.5 Summary

Early intervention services have arisen from the need to treat FEP at the earliest opportunity to improve treatment outcome and lower treatment costs. Cognitive function may play a pivotal role in outcome and recovery, though this area has received relatively little attention to date. A better understanding of the nature and extent of cognitive deficits in FEP is needed, as this area may have important treatment implications in the future.

1.2.6 Aims of the review

This aims of this review are to examine the research evidence for cognitive deficits in FEP, the neurological basis of these possible deficits and their relation to social functioning.

19

2. Method

Relevant studies of patients with first-episode psychosis were identified using a three-stage search strategy. Firstly, multiple computer searches of the databases CINAHL, PsycINFO, Medline, Web of Science, Francis, Trip, the Cochrane library and Science direct were conducted between July and December 2006 utilising the full date ranges of publication available for the databases. The following keywords were used to search across titles "cognitive with deficit* or dysfunction* or impairment*" and "first episode with psychosis or schizophrenia". Secondly, relevant journals (e.g. Schizophrenia Research, Psychopathology) were hand searched from the past 5 years (2001-2006). Thirdly, the reference sections of key papers were examined in order to identify further relevant papers.

3. Results

One hundred and thirteen papers were identified using this search strategy (98 from databases, 15 from hand-search). Of these, 87 were excluded because they were either not published in English or were concerned with pharmacotherapy, early intervention service development, expressed emotion in relatives, bipolar disorder, childbirth induced psychosis, insight in psychosis, crisis intervention, alcohol and substance abuse, duration of untreated psychosis and outcomes, non compliance with medication, service users perspectives and/or the prodromal phase of schizophrenia. Twenty-six were included in the review (16 from the database and 10 from the hand search). The included papers were then categorised according to their subject area. These were neurological (6), cognitive deficits (15), and social functioning (5). Each set of papers will be reviewed in turn in the sections below.

20

3.1 Neurological

The neurological papers were mainly concerned with the presence of

neurological soft signs (NSS) and brain morphology.

3.1.1 Neurological soft signs

The presence of NSS in FEP is widely reported; however, there is some

difference of opinion regarding what form these signs take. Dazzan and Murray (2002)

conducted a systematic review of 9 studies that examined NSS at the time of first

presentation in FEP or schizophrenia and compared them with healthy controls or those

at high-risk of developing psychosis. The authors found that first episode patients

showed an excess of NSS especially in the areas of motor coordination and sequencing,

sensory integration and reflexes. A limitation noted in the review was the relatively

small number of studies that have examined NSS in FEP. Also, the variety of

instruments used to assess NSS made it difficult to compare results (Dazzan & Murray,

2002).

Since NSS appear to be present at the beginning of the psychotic illness, some

authors have examined the stability of these signs over time. Bachmann, Bottmer,

Weimer, and Schröder (2003) conducted a follow-up study of 39 first episode

schizophrenia patients at 14.2 months after treatment for schizophrenia or

schizophreniform disorder. At follow-up, NSS as assessed by the Heidelberg scale

(Schröder et al., 1992) had decreased significantly. The authors concluded that NSS are

a state related variable.

Some researchers have examined whether NSS are associated with cognitive

impairments. Schuepbach, Sanders, and Keshavan (2002) assessed 59 neuroleptic naïve

first episode patients and 51 matched healthy controls using a modified neurological

evaluation scale (NES) (Buchanan & Heinrichs, 1989), clinical ratings and a

neuropsychological battery. Those with FEP showed more NSS and decreased

performance in neurocognition. The cognitive perceptual domain of NES predicted

specific neuropsychological functions measuring attention, language skills and executive

functioning. The authors concluded that parts of the NES (e.g. cognitive perceptual

domain) can reliably predict prefrontally mediated cognitive dysfunction in first episode

schizophrenia. A potential criticism of this study is that the NES does not have a

"cognitive perceptual" subscale, and it was unclear from the article how the authors had

calculated this. However, their finding that patients with FEP showed an excess of NSS

was consistent with Dazzan and Murray's (2002) findings as discussed above.

It appears that first episode patients show an excess of NSS in comparison to

healthy controls and those at high-risk of developing psychosis (Dazzan & Murray,

2002; Schuepbach et al., 2002). NSS in FEP decrease over time (Bachmann et al., 2003)

and may be related to prefrontally mediated cognitive dysfunction (Schuepbach et al.,

2002).

3.1.2 Brain morphology

Some researchers have investigated whether there are differences in the

underlying brain morphology of first episode patients compared with normal subjects.

Steen, Mull, McClure, Hamer, and Lieberman (2006) conducted a systematic review and

meta-analysis of Magnetic Resonance Imaging (MRI) studies comparing brain volume

in FEP patients and healthy controls. They included 66 papers in their analyses and

found that whole brain and hippocampal volume were reduced and ventricular volume

was increased in first episode patients relative to healthy controls. A potential limitation of the studies reviewed was their small sample size and a lack of replication where particular significant effects were found. In addition to this, few environmental factors (e.g. medication, alcoholism and social deprivation) were controlled for in the studies reviewed, and it is possible that these factors may have contributed to the morphological changes observed.

Similar findings were reported in a study by Vita, De Peri, Silenzi, and Dieci (2006). They conducted a meta-analysis of quantitative MRI studies in first episode schizophrenia. They included 21 studies and found significant overall effect sizes for lateral and third ventricular volume increase, and for volume reduction of whole brain and hippocampus relative to healthy controls. The authors concluded that brain abnormalities are present at the early stages of psychosis.

To see whether morphological differences were stable over time, Lieberman et al. (2001) conducted a longitudinal study in first episode schizophrenia. They compared first episode schizophrenia (n=107) and healthy controls (n=20) on MRI, clinical assessments of psychopathology, and treatment outcome for up to 6 years. The authors found ventricular enlargement and anterior hippocampal volume reductions in first episode schizophrenia relative to healthy controls. In addition to this, patients who were classified as having poor outcome schizophrenia (failure to respond to treatment and reoccurrence of persistent positive and negative symptoms) showed total ventricular enlargement over time compared with good outcome patients. This finding contrasts with the assumption mentioned earlier in this review that the ventricles in patients with schizophrenia do not enlarge over time (Gattaz et al., 1991). A possible limitation of this study was the small number of control participants used. Also, the average age of

the control group was significantly higher than the first episode schizophrenia group. As

the brain changes structurally over time (Droge & Schipper, 2007), the control group

may not have provided an accurate comparison for the first episode schizophrenia group

There appears to be consistent findings in the studies reviewed regarding the

brain morphology of first episode patients. Whole brain and hippocampal volume were

reduced and ventricular volume was increased in FEP (Lieberman et al, 2001; Steen et

al., 2006; Vita et al., 2006) relative to healthy controls. Those who did not respond well

to treatment and had persistent symptoms showed ventricular enlargement over time

(Lieberman et al., 2001). This suggests that the brains of those who do not respond well

to treatment may be subject to neurodegeneration over time. However, this

interpretation is tentative as it is based on the results of only one study.

3.2 Cognitive deficits

As FEP patients show an excess of NSS and differences in brain morphology

compared to healthy controls it is plausible that there may be some resulting cognitive

dysfunction. This will be explored in the next section of the review. Due to the large

number of cognitive papers, they were divided into the following categories: generalised

deficits, specific deficits, subjective experiences, course, symptoms, and treatment.

3.2.1 Generalised deficits

There is much literature regarding the general profile of cognitive deficits in

FEP. Addington, Brooks, and Addington (2003) looked at FEP compared with healthy

controls by assessing a large sample (n= 312) of first episode patients who had no more

than 3 months of previous treatment. First episode participants scored lower than

controls on all tests administered. They demonstrated particular difficulty in verbal fluency, verbal memory, and working memory. They also scored poorly on measures of attention, information processing, and motor skills. These deficits have some similarity to the deficits in executive function, memory, attention and motor skills found in schizophrenia generally (Green, 1998). There were no associations between cognition and positive symptoms and a range of associations between negative symptoms and cognition. These findings are commonly found in schizophrenia generally (Green, 1998).

Mohamed, Paulsen, O'Leary, Arndt, and Andreasen, (1999) found a similar pattern of deficits in their study which looked at 94 recently admitted first episode patients and 305 healthy controls. They used a comprehensive battery of tests that assessed the domains of psychomotor speed, executive function, memory, and problem solving. Those patients with first episode schizophrenia showed impairments on most measures relative to controls, and the authors concluded that first episode patients have a generalised deficit in cognitive function.

In summary, there appear to be consistent findings from the literature to support evidence for a generalised cognitive deficit in FEP. First episode patients were impaired relative to controls on most areas of cognition including verbal fluency, verbal memory, working memory, attention, processing skills, and motor skills (Addington et al., 2003; Mohamed et al., 1999). Negative symptoms were associated with cognition (Addington et al., 2003). These findings reflect the literature on psychosis more generally (Green, 1998).

3.2.2 Specific deficits

Since there is much research evidence to support the presence of cognitive deficits in FEP, some researchers have queried whether there may be more specific rather than generalised deficits. Barrett, Mulholland, Cooper, and Rushe (2006) compared first episode schizophrenia patients and healthy controls on a range of neuropsychological assessments. They divided the patient group into a poor organisation and strategy (OS) group and a good OS group. Poor OS patients had higher levels of depressive symptoms. They were also more impaired on tests of semantic fluency, verbal memory, attention, working memory and executive function compared to good OS patients and controls. The authors concluded that a high percentage of patients demonstrated a clear fronto-parietal deficit, which may reflect a disruption of normal brain maturational processes in adolescence. However, the mean age of their sample (30.8 years, $\pm$ 11.7) was not representative of the adolescent age group, which may not offer sufficient support for their conclusion. Also, it is possible that the depression experienced by the poor OS patients may have interfered with their performance on the neuropsychological assessments. Nevertheless, their results suggested that there were two sub-groups within the sample of patients, one of which was significantly more impaired than the other in neuropsychological function and had higher levels of depressive symptoms.

Fagerlund, Pagsberg, and Hemmingsen (2006) examined cognitive deficits and levels of IQ in adolescent onset schizophrenia and other psychotic disorders. They assessed 39 first episode adolescents (18 diagnosed with first episode schizophrenia, 21 with non-organic non-affective psychoses), and a matched healthy control group. A range of neuropsychological tasks was used which assessed IQ, attention, executive function, reaction time and memory. A similar profile and level of impairment were

found on measures of attention, executive function, reaction time, and memory across the first episode schizophrenia and non-organic non-affective psychoses groups in comparison to controls. However, the first episode schizophrenia group had more global IQ deficits than those with non-organic non-affective psychoses. Declines in levels of IQ from pre-morbid functioning have been reported generally in studies of schizophrenia (Weickert et al., 2000), and the authors proposed that the illness might have a negative impact on IQ because none of the participants were reported to have learning difficulties prior to illness onset. A measure of pre-morbid IQ would have clarified this issue.

Some studies have looked at olfactory deficits as a predictor of the severity of symptoms, because olfactory deficits are thought to be a marker of orbito-frontal function. Brewer et al. (1998) compared healthy controls (n=24), patients at high risk for psychosis (n=65), never medicated FEP patients (n=49) and medicated FEP (n=25) patients. The high risk group and both patient groups demonstrated significant reductions in current IQ, smell identification as measured by the University of Pennsylvania Smell Identification Test (UPSIT) (Doty, Shaman, & Dann, 1984), attention, memory and executive function compared to controls. Over the 18 month recruitment phase 32% of the high risk patients became psychotic. The authors concluded that reductions in IQ and deficits in smell identification, attention, memory and executive function are present at the outset of psychosis and are not due to medication effects.

Good, Whitehorn, Rui, Milliken, and Kopala (2006) used the UPSIT to assess 58 medication naïve FEP patients at baseline and at 1 year. Patients with non-remission of negative and cognitive/disorganized symptoms had significantly lower baseline UPSIT scores compared with patients in remission. The authors did not suggest a possible

27

mechanism for this and concluded that UPSIT scores could be used to identify patients at risk for persistent negative and cognitive/disorganised symptoms. It is plausible that the orbito-frontal area of the brain, which is responsible for smell, may also be associated with negative and cognitive/disorganized symptoms in FEP.

In summary, some authors have reported more specific deficits in IQ among those patients at high risk of developing psychosis (Brewer et al., 1998) and those with FEP compared with patients with non-organic non-affective psychosis (Fagerlund et al., 2006). Some authors have identified sub-groups of FEP patients who are more impaired in attention, working memory, semantic fluency, verbal memory, and executive function and have higher levels of depressive symptoms (Barrett et al., 2006). Smell identification, which is understood to reflect orbito-frontal function, may be impaired from the outset of the illness and may be used to predict those at risk of more persistent symptoms (Brewer at al., 1998; Good et al., 2006). However, as few studies have been conducted in this area any conclusions drawn from the literature must be tentative.

3.2.3 Subjective experiences of cognitive dysfunction

There appears to be much objective evidence regarding the presence of cognitive deficits in FEP, and some authors have questioned whether first episode patients are aware of these. Moritz et al. (2001) used the Frankfurt Complaint Questionnaire (FCQ) (Sülwood, 1986) to assess subjective cognitive dysfunction in 20 first episode patients, 36 patients with chronic schizophrenia and 20 healthy controls. Ratings were comparable for each schizophrenia group and were significantly higher than for healthy controls. The authors concluded that since both groups had equal awareness of their deficits, cognitive functions do not deteriorate further after the first episode of psychosis.

One criticism of this study is that the FCQ has been shown to have poor diagnostic

validity and reliability compared to the Eppendorf Schizophrenia Inventory (ESI) (Mass,

2000), which may have been a more appropriate measure to use (Mass, 2005).

Moritz et al. (2000) investigated the relationship between subjective cognitive

disturbances and symptomatic outcome. Fifty-three FEP patients were assessed at

baseline, and 31 were followed up 1 year later. The FCQ and the Brief Psychiatric

Rating Scale (BPRS) (Ventura, Green, Shaner, & Liberman, 1993) were used, the latter

to assess symptoms. Awareness of memory dysfunction was the strongest predictor of

symptomatic worsening. However, the authors did not suggest a mechanism to account

for this relationship. The authors concluded that memory dysfunction may be a

potential risk factor for symptom worsening and suggested that first episode patients

may benefit from cognitive rehabilitation to remedy this deficit. Some potential

weaknesses of this study were that objective neuropsychological tests were not used to

explore the self-reported memory dysfunction, and the authors did not suggest a

mechanism whereby memory and symptoms were linked.

3.2.4 Course of cognitive deficits

Several studies have looked at the stability of cognitive deficits over the course

of psychosis. Addington and Addington (2000) looked at initial presentation and

follow-up at 1 and 2 years in first episode patients. Ninety subjects were followed up at

1 year and 47 subjects at 2 years. Poor neurocognitive performance was associated with

negative symptoms rather than positive symptoms at both follow-ups. There was a trend

towards general improvement in neurocognition and a significant improvement on some

memory tasks in both periods; however, performance remained in the impaired range.

The authors did not report whether or not the participants were taking medication, which may have impacted on the results.

Addington, Saeedi, and Addington (2005) later conducted a 3 year longitudinal study of 247 first episode patients compared with non-psychiatric controls. They assessed cognition at baseline, 1 year (n=154) and 2 year follow-up (n=124). They found that positive symptoms improved significantly from baseline to 1 year. There was significant improvement over time in several neurocognitive domains including executive function, psychomotor speed, response inhibition and verbal memory. One potential weaknesses of this study was that all participants showed improvement over the course of the study, perhaps due to learning effects because there were no alternate forms of the tests used. Again, the authors did not report whether or not the participants were medicated, which may have impacted on the results.

It is difficult to draw any firm conclusions from the research on the course of cognitive deficits in FEP because there have been few studies in this area, and those studies that have been conducted exhibit weaknesses in design. Both studies reviewed suggested some improvement in cognitive function (Addington & Addington, 2000; Addington et al., 2005); however, this improvement remains in the impaired range (Addington & Addington, 2000). This fits with Moritz et al.'s (2001) finding that cognitive deficits do not deteriorate further after the first episode. In one study poor neurocognitive performance was associated with negative symptoms rather than positive symptoms (Addington & Addington, 2000). This is consistent with Addington et al.'s (2003) finding earlier in the review that negative symptoms were associated with cognition. The contribution of symptoms to cognitive function will be examined further in the next section.

<u>3.2.5 Symptoms</u>

Some researchers have specifically examined the relation between psychotic symptoms and cognitive function in first episode patients. Prikryl et al. (2003) looked at the association between severity of psychotic symptoms as measured by the Positive and Negative Syndrome Scale (PANSS) (Kay, Fiszbein, & Opler, 1987) and performance on the Wisconsin Card Sorting Test (WCST). The PANSS was administered before and 28 days after treatment with anti-psychotic medication. There were significant reductions in PANSS ratings after treatment, but PANSS scores were not correlated with WCST scores. The authors concluded that symptoms may not be related to cognitive function in FEP.

Addington and Addington (1998) also looked at the relation between psychotic symptoms and cognitive function. They compared first episode patients who were recently diagnosed with schizophrenia or schizophreniform disorder with a sample of patients who had been ill for many years with a diagnosis of schizophrenia. The first episode group had more positive symptoms, while the patients who had been ill for longer had more negative symptoms. Those who had been ill for longer scored more poorly on the WCST and tests of memory than first episode patients. The authors concluded that neurocognitive function was more related to negative than positive symptoms, which is consistent with Addington and Addington's (2000) finding. Addington and Addington (1998) also found that memory and executive function may deteriorate after the first episode. This is in contrast to findings reported earlier in the review that cognitive function may not deteriorate further after the first episode (Addington & Addington, 2000; Addington et al., 2005; Mortiz et al., 2001).

The evidence regarding the relationship between symptoms and cognitive deficits is inconclusive. Prikryl et al. (2003) found no association, while Addington and Addington found that cognitive function was more related to negative than positive symptoms in two separate studies (1998; 2000). From the three studies reviewed it appears that cognitive function may be related to negative symptoms. However, further research in this area is needed in order to draw more definitive conclusions.

3.2.6 Treatment effects

Some studies have looked at the effectiveness of pharmacological treatment and cognitive dysfunction in FEP. Harvey, Rabinowitz, Eerdekens and Davidson (2005) compared the effectiveness of Haloperidol and Risperidone in 533 patients experiencing their first episode of schizophrenia or a related psychosis. Those treated with Risperidone showed wide-ranging improvements in cognitive function. Overall improvement was greater with Risperidone than Haloperidol.

Keefe (2004) conducted a more extensive study comparing the cognitive effects of low doses of atypical and typical antipsychotic medication. In the first part of the study, 167 first episode patients were randomized to 2 years of double-blind treatment with Olanzapine or Haloperidol and were given neuropsychological assessments at baseline, 12 weeks, 6 months, 12 months and 2 years after treatment. Both drugs improved cognitive function; however, Olanzapine had a greater effect. In the second part of the study, low doses of Haloperidol were compared with Risperidone. Patients in each group demonstrated significant improvement; however, Risperidone patients demonstrated greater improvement. The authors concluded that atypical antipsychotics

are superior at improving cognition, which is consistent with the findings of Harvey et al. (2005).

Schuepbach, Keshavan, Kmiec, and Sweeney (2002) found no advantage in using Risperidone over Haloperidol in their study of 34 first episode patients. Twenty-three patients were treated with Risperidone and 16 with Haloperidol. The scale for Assessment of Negative Symptoms (SANS) (Andreasen, 1983) and the Scale for Assessment of Positive Symptoms (SAPS) (Andreasen, 1984) were used to assess symptoms. Although anti-psychotic medication markedly reduced clinical symptomatology, there was no clinical advantage in using Risperidone over Haloperidol. In contrast to Keefe's (2004) study a smaller sample was used and there was a shorter follow-up period after medication. Therefore, Keefe's (2004) study may be more informative.

It seems that Risperidone and Olanzapine may improve cognitive deficits in FEP more than Haloperidol (Harvey et al., 2005; Keefe, 2004).

3.2.7 Summary

There appears to be clear evidence to support a generalised cognitive deficit in FEP, which is also reported in the psychosis literature more generally. There may be a sub-group of FEP participants who are more impaired in some aspects of cognitive function and IQ, and show higher levels of depression. Depression may affect performance on neuropsychological tasks, and this relationship should be explored further in future studies. Subjective awareness of memory dysfunction may predict symptomatic worsening. Awareness of cognitive dysfunction does not appear to differ between first episode and multi-episode groups. This area of research would benefit

from employing objective neuropsychological tests alongside subjective measures of cognitive dysfunction. It seems that cognitive dysfunction may improve over the course of the illness; however, functioning may remain in the impaired range and may be related to negative symptoms. In terms of pharmacological treatment, atypical antipsychotics (e.g. Risperidone and Olanzapine) may be more effective than Haloperidol in treating cognitive deficits in FEP.

The final part of the review will examine the relationship between cognitive and social functioning in FEP. The cognitive deficits found in FEP may have a significant impact on many of these skills and the quality of life of these individuals.

3.3 Social functioning

The term "social functioning" refers to skills and competencies in a number of different areas including community and occupational functioning, employment, and activities of independent living and self-care. There are few studies on social functioning in FEP, which was reflected in the search results. The papers reviewed were divided into the following areas: cognition and social functioning; first and multi-episode psychosis; and social anxiety.

3.3.1 Cognition and social functioning

Some studies have looked at the influence of cognitive deficits on social and community functioning in FEP, with mixed results. Addington and Addington (2004) assessed cognition and social outcome in 182 first episode patients and 127 of these at a 2 year follow-up. The authors did not specify the outcome measures used to assess these

variables. They found a few statistically but not clinically significant improvements at 2 years. After controlling for symptoms, regression analyses showed that cognitive impairment accounted for only 4-6% of the variance in social functioning. The authors concluded that although related, poor social functioning may be independent of cognitive function in FEP.

In a separate study, Addington and Addington (2005) conducted a prospective cohort study of 194 people with FEP. They assessed pre-morbid functioning using the Pre-morbid Assessment Scale (PAS) (Cannon-Spoor, Potkin, & Wyatt, 1982), symptoms using the PANSS, and social functioning using the Quality of Life Scale (QOL) (Burckhardt & Anderson, 2003). Data was collected at baseline, 1 and 2 years. The authors identified 4 patterns of pre-morbid functioning: deteriorating, stable-deteriorating, stable-good, and stable-intermediate. They found that at both 1 and 2 years, participants with a deteriorating or stable-deteriorating pattern of pre-morbid function had more severe negative symptoms and more impaired social functioning than those with stable-good and stable-intermediate patterns of pre-morbid function. The authors concluded that poor pre-morbid functioning prior to diagnosis may predict poor social and symptomatic outcome in FEP.

Verdoux, Liraud, Assens, Abalan, and Van-Os (2002) did a follow-up study of cognitive and social functioning in FEP to examine whether baseline memory and executive function deficits predicted poor social and clinical outcome over a 2 year period following first admission. Cognitive function was assessed in 35 patients admitted with FEP. The cognitive tests assessed the domains of executive function, response inhibition, psychomotor speed, attention, and verbal and visual memory. Patients were assessed at 6 monthly intervals over the 2 year follow-up. Social outcome

was assessed by occupational and residential status, whereas clinical outcome was assessed by re-hospitalisation data and symptomatic status. The authors found no significant association between cognitive performance and social outcome (either occupational or residential), but significant associations were found between poor memory scores and worse clinical outcome. A potential limitation of this study was the lack of standardised social functioning and symptomatic measures used.

The studies reviewed show little (Addington & Addington, 2004) or no evidence (Verdoux et al., 2002) of an association between cognitive deficits and social functioning in FEP. Pre-morbid function prior to diagnosis may predict social and symptomatic outcome in FEP (Addington & Addington, 2005). A weakness of the studies reviewed is a lack of standardised measures used to assess social functioning, which may have reduced their ability to detect significant differences (i.e. Type II error).

3.3.2 First and multi-episode (ME) psychosis

Some authors have compared first episode and multi-episode patients with psychosis to see if there are differences in social functioning between the groups. Grant, Addington, Addington, and Konnert (2000) assessed social functioning in 40 patients experiencing first episode schizophrenia, 40 individuals who had experienced ME schizophrenia, and 40 non-psychiatric controls. The participants were assessed using the Social Functioning Scale (SFS) (Birchwood, Smith, Cochrane, Wetton, and Copestake, 1990), QOL, the assessment of interpersonal problem solving skills (AIPSS) (Donahue et al., 1990) and the PAS. The control group performed much better on all measures than both patient groups. There were no differences between the two schizophrenia groups on the PAS, SFS, and the AIPSS. On the QOL, first episode participants scored

more poorly than the multi-episode group. This result did not fit with their hypothesis that first episode patients would have improved social functioning compared to multi-episode patients. The authors concluded that social functioning deficits appear to be present at the beginning of the illness and do not deteriorate throughout the course of the illness. Although there appears to be little or no relationship between social functioning and cognition in FEP, this finding is consistent with research discussed earlier in the review, which showed that cognitive deficits do not appear to worsen over time (Addington & Addington, 2000; Addington et al., 2005; Moritz et al., 2001).

3.3.3 Social anxiety

Voges and Addington (2005) looked at the association between social anxiety and social functioning in FEP. They assessed 60 FEP patients. Social phobia was assessed using a structured clinical interview for DSM-IV criteria (SCID-I) (Spitzer, Williams, Gibbon, & First, 1992). They also used the Social Phobia and Anxiety Inventory (SPAI) (Turner, Bendel, & Dancu, 1996) and the SFS. They found that 32% of the sample met SCID-I criteria for social phobia, and approximately 60% were experiencing elevated levels of social anxiety according to the SPAI. Higher social anxiety scores as measured by the SPAI were related to poor social functioning as measured by the SFS. The authors concluded that high levels of social anxiety were significant predictors of social functioning in FEP. A potential weakness of this study was the lack of a non-psychiatric control group for comparison to elucidate the extent of social phobia and social anxiety in the general population.

3.3.4 Summary of social functioning studies

The paucity of studies on social functioning in FEP, lack of standardised measures to assess social functioning, and absence of control groups makes it difficult to draw any firm conclusions from the research in this area. However, from the studies reviewed it seems there is little or no association between cognitive deficits and social functioning in FEP. This contrasts to findings in schizophrenia generally where cognitive deficits are associated with social functioning (Green, 1996; Green, Kern, Braff, & Mintz, 2000). Pre-morbid function prior to diagnosis and social anxiety may predict social outcome in FEP. Social functioning does not appear to worsen over the course of the illness and difficulties in this area may be present from the outset, but clinical experience suggests that some patient's social functioning deteriorates over the course of psychotic illness.

4. Discussion

This review examined the research evidence for neurological and cognitive deficits in FEP and their relation to social functioning. The studies included in the review were broadly categorised into neurological, cognitive deficits and social functioning subject areas. The following section will discuss the main findings of the review and compare these findings to the wider literature.

4.1 Neurological

The neurological papers reviewed suggested that individuals with FEP show an excess of NSS in comparison to healthy controls (Dazzan & Murray, 2002; Scheupbach et al., 2002). This finding has been reported in the schizophrenia literature more generally (Bombin, Arrango, & Buchanan, 2005; Rossi et al., 1990), and there is little

evidence to suggest that these NSS are related to neuroleptic medication (Bartko, Zador, Horvath, & Herezeg, 1988; Rossi et al., 1990). One study included in this review suggested that NSS decrease over time with treatment (Buchmann et al., 2002). There are mixed findings in the wider literature regarding the stability of NSS over time. However, there is some evidence to suggest that those who developed psychosis and NSS had abnormalities in childhood motor responses, which may reflect early disturbances in brain function (Walker & Lewine, 1990). It was hypothesised that these abnormalities remain stable throughout life, emerging as NSS during the onset of psychosis and fluctuating over the course of the illness when there are variations in state such as symptom exacerbations or exposure to illicit drugs or alcohol (Bombin et al., 2005). More longitudinal research is needed in this area tracking the pattern of NSS over the course of psychosis.

The studies reviewed reported differences in the brain morphology of first episode patients compared with healthy controls. First episode patients showed reduced whole brain and hippocampal volume, ventricular enlargement, increased lateral and third ventricle volume and reduced anterior hippocampal volume relative to healthy controls (Lieberman et al., 2001; Steen et al., 2006; Vita et al., 2006). The findings regarding ventricular enlargement, third ventricle volume and reduced whole brain volume have been reported in the schizophrenia literature generally (Chua & McKenna, 1995; Narayan et al., 1986; Shelton et al., 1988). One study reviewed reported that first episode schizophrenia patients with poor outcome showed ventricular enlargement over time (Lieberman et al., 2001). Again, ventricular enlargement has been reported in the schizophrenia literature more generally, though there are mixed findings regarding its relation to outcome (DeLisi, Sakuma, Maurizo, Relja, & Hoff, 2004).

4.2 Cognitive deficits

The next section of the review looked at the evidence for cognitive deficits in FEP. First episode participants were consistently reported to perform more poorly on neuropsychological tasks than healthy controls, especially in the areas of verbal fluency, verbal memory, working memory, attention, information processing and motor skills (Addington et al., 2003; Mohamed et. al, 1999). This finding reflects the cognitive domains known to be impaired in schizophrenia generally (Gold & Harvey, 1993; Green et al., 2000) and supports the idea that cognitive deficits are a core feature of the illness (Green et al., 2004). One study found specific deficits in IQ in first episode patients and those at risk of developing psychosis compared to healthy controls and patients with non- organic non-affective psychosis (Fagerlund et al., 2006). In the wider literature the average IQ reported for those with psychosis is around 90, and some researchers have termed this "a generalised performance deficit" (Frith et al., 1991). Future studies would benefit from exploring premorbid levels of IQ in an effort to establish whether the reported differences in IQ between psychosis patients and healthy controls are due to the effects of the illness or a generalised IQ deficit that predates the illness.

Some studies reviewed reported that smell identification was impaired from the outset of psychotic illness and may be used to predict those at risk of developing psychosis and persistent negative and cognitive/disorganised symptoms (Brewer et al., 1998; Good et al., 2006). This area requires further research because if these findings are supported, those at risk of developing psychosis and these symptoms could receive interventions to reduce their progression and impact. Regarding treatment, Risperidone and Olanzapine appear to have superior effects in treating cognitive deficits to Haloperidol.

One study looking at the subjective experiences of cognitive deficits in FEP found equal awareness of impairments in first and multi-episode patients (Moritz et al., 2001). This area of research would benefit from incorporating objective measures of cognitive function alongside subjective measures that are reliable and valid. Awareness of memory dysfunction was found to be a predictor of symptomatic worsening in one study of first episode patients (Moritz et al., 2000). The relationship between cognitive functioning and psychotic symptoms is widely reported in the literature with negative symptoms being especially associated with cognitive impairment (Buchanan et al., 1994; Hammer, Katsanis, & Iacono, 1995).

4.3 Social functioning

From the studies reviewed, it seems there is little or no relationship between cognition and social functioning in FEP. This contrasts with the findings from the wider schizophrenia literature where neurocognitive functioning has been shown to be closely associated with social functioning (Green, 1996; Johnstone, MacMillan, Frith, Benn, & Crow, 1990). One study compared first and multi-episode FEP patients' social functioning and concluded that social functioning does not deteriorate throughout the course of the illness (Grant et al., 2000). This is consistent with Addington and Addington's (2000) finding that social functioning in patients with schizophrenia remained stable over a 2 ½ year follow-up period. However, further research is needed in this area using longer follow-up periods and standardised measures of social functioning. The contribution of social anxiety (Voges & Addington, 2005) and premorbid social functioning (Addington & Addington, 2005) to social functioning

should be examined as the studies reviewed suggested there may be links between these variables.

4.4 Methodological limitations

There were a number of potential weaknesses in the papers reviewed. Some of the systematic reviews and meta-analyses used a small number of papers, which did not allow for an extensive review of the subject area concerned. Also, some of the research studies used a small number of experimental and/or control participants, which may have reduced their statistical power. In one study, there was a significant age difference between the control and experimental groups, which may have led to inaccurate comparisons between the groups. Some of the social functioning studies did not use objective neuropsychological tests or validated measures of social functioning. A few studies used questionnaires that have poor diagnostic validity and reliability. The follow-up periods of some of the studies were rather short at times. Some studies did not report on potential confounding factors such as drug and alcohol use and medication.

There were also some weaknesses in this review and the methodology used. The search criteria may not have been specific enough to target papers on specific areas of cognitive function, which may have resulted in some relevant papers being excluded from the review. Because of the high ratio of abstracts to full-length papers and the space constraints associated with publication, methodological details were often lacking, which made it difficult to assess the quality of the studies.

4.5 Recommendations for future studies

Future studies would benefit from exploring the relationship between IQ and performance on neuropsychological assessments. A sub-group of the participants assessed in Fagerlund et al.'s (2006) study showed global IQ deficits compared to healthy controls and a psychiatric control group. It was not clear whether these participants' pre-morbid IQs were in the low range or if they were impaired as a consequence of the illness. Hence, it is unclear whether the impaired performance of first episode patients on a range of neuropsychological tasks was due to pre-morbid global IQ deficits or illness.

Due to the relatively few studies on social functioning in FEP, further research in this area is needed, considering that social functioning may be important for long-term social and occupational outcome and personal well-being. Studies in this area should employ standardised measures which assesses the domains thought to be important to social functioning (e.g. physical and psychological well being, work/education, leisure, finances, living situation, social relations, etc.).

4.6 Conclusions

In spite of the methodological limitations discussed above, this review has established that individuals with FEP show neurological abnormalities and a range of cognitive deficits. The data suggests that cognitive deficits may not be related to social functioning in FEP, which contrasts with findings in schizophrenia generally; however, this area in particular requires further research.

References

Addington, J., & Addington, D. (1998). Neurocognitive functioning in first episode

schizophrenia. *Schizophrenia Research, 29,* 50-51.

Addington, J., & Addington, D. (2000). Neurocognition in first episode schizophrenia: A

2 year follow-up. *Schizophrenia Research, 41,* 271-272.

Addington, J., & Addington, D. (2004). Impact of cognitive impairments on 2-year

outcome in first-episode psychosis. *Schizophrenia Research, 70,* 101.

Addington, J., Brooks, B. L., & Addington, D. (2003). Cognitive functioning in first

episode psychosis: initial presentation. *Schizophrenia Research, 62,* 59-64.

Addington, J., Saeedi, H., & Addington, D. (2005). The course of cognitive functioning

in first episode psychosis: Changes over time and impact on outcome.

Schizophrenia Research, 78, 35-43.

Aitchison, K. J., Meehan, K., & Murray, R. M. (1999). *First episode psychosis.* London:

Martin Dunitz Ltd.

Andreasen, N. C. (1983). *The Scale for the Assessment of Negative Symptoms (SAPS).*

Department of Psychiatry. USA: The University of Iowa College of Medicine.

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

Department of Psychiatry. The University of Iowa College of Medicine, Iowa

City.

Bachmann, S., Bottmer, C., Weimer, D., & Schröder, J. (2003). First-episode

schizophrenia: A follow-up study on neurological soft signs. *Schizophrenia*

Research, 60, 10.

Barrett, S. L., Mulholland, C. C., Cooper, S. J., & Rushe, T. M. (2006). Visuospatial

construction skills in first episode schizophrenia: Relationship to

symptomatology and cognitive deficits. *Schizophrenia research, 81,* 227.

Bartko, G., Zador, G., Horvath, S., Herczeg, I. (1988). Neurological soft signs in

chronic schizophrenic patients: clinical correlates. *Biological Psychiatry, 24,*

458–460.

Beck A. T. (1976). *Cognitive therapy and the emotional disorders.* New York:

International Universities Press.

Beiser, M., Erikson, D., Fleming, J. A., & Iacono, W. G. (1993). Establishing the onset

of psychotic illness. *American Journal of Psychiatry, 150,* 1349-54.

Bentall, R. P. (2003). *Madness explained.* England: The Penguin Press.

Birchwood, M., Cochrane, R., MacMillan, F., Copestake, S., Kucharska, J., & Carriss,

M. (1992). The influence of ethnicity and family structure on relapse in first-

episode schizophrenia. *British Journal of Psychiatry, 161,* 783-90.

Birchwood, M., McGorry, P., & Jackson, H. (1997). Early intervention in schizophrenia.

British Journal of Psychiatry, 170, 2-5.

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., Todd, P., & Jackson, C. (1998). Early intervention in psychosis: the

critical period hypothesis. *International Journal of Clinical*

Psychopharmacology, 13, S31-S40.

Bogerts, B., Meertz, E., & Schonfield-Bausch, R. (1985). Basal ganglia and limbic

system pathology in schizophrenia: a morphometric study of brain volume and

shrinkage. *Archives of General Psychiatry, 42,* 784-791.

Bombin, I., Arrango, C., & Buchanan, R. W. (2005). Significance and Meaning of

Neurological Signs in Schizophrenia: Two Decades Later, *Schizophrenia*

Bulletin, 31, 962-77.

Brewer W. J., Francey, S. M., Wood, S. J., Jackson, H. J., Pantelis, C., Phillips, L. J.,

Yung, A. R., Anderson, V. A., & McGorry, P. D. (2005). Memory

impairments identified in people at ultra-high risk for psychosis who later

develop first-episode psychosis. *American Journal of Psychiatry, 162,* 71-8.

Brewer, W., Francey, S., Yung, A., Velakoulis, D., Anderson, V., McGorry, P., Singh,

B., Copolov, D., & Pantelis, C. (1998). Cognitive and olfactory deficits: Course

from high risk to first-episode psychosis. *Schizophrenia Research, 29,* 54-55.

Buchanan, R. W., & Heinrichs, D. W. (1989). The Neurological Evaluation Scale

(NES): a structured instrument for the assessment of neurological signs in

schizophrenia. *Psychiatry Research, 27,* 335-50.

Buchanan, R. W., Strauss, M. E., Kirkpatrick, B., Holstein, C., Breier, A. and W. T.

Carpenter, W. T. (1994). Neuropsychological impairments in deficit vs.

nondeficit forms of schizophrenia. *Archives of General Psychiatry, 51,* 804–

811.

Burckhardt, C. S., & Anderson, K. L. (2003). The Quality of Life Scale (QOLS):

Reliability, Validity, and Utilization. *Health and Quality of Life Outcomes, 1,*

60.

Cannon-Spoor, H. E., Potkin, S. G., & Wyatt, R. J. (1982). Measurement of premorbid

adjustment in chronic schizophrenia. *Schizophrenia Bulletin, 8,* 470-484.

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.

Connell, P. (1958). *Amphetamine psychosis.* London: Chapman and Hall.

Crow, T. J., Ball, J., Bloom, S. R., Brown, R., Bruton, C. J., Colter, N., Frith, C. D.,

Johnstone, E. C., Owens, D. G. C., & Roberts, G. W. (1989). Schizophrenia as an

anomaly of development of cerebral asymmetry. *Archives of General Psychiatry,

46,* 1145-1150.

Crow, T. J., Cross, A. J., Johnson, J. A., Johnstone, E. C., Joseph, M. H., Owen, F.,

Owens, D. G. C., & Poulter, M. (1984). Catecholamines and schizophrenia: an

assessment of the evidence. In E. Usdin, A. Carlsson, A. Dahlstrom & J. Engel

(Eds.), *Catecholamines. Part C: Neuropharmacology and central nervous

system - therapeutic aspects.* (pp. 11-20) New York: Liss.

Dazzan, P., & Murray, R. M. (2002). Neurological soft signs in first-episode psychosis:

a systematic review. *British Journal of Psychiatry Supplement, 43,* S50-S57.

DeLisi, L. E., Sakuma, M., Maurizo, A. M., Relja, M., & Hoff, A. L. (2004). Cerebral

ventricular change over the first 10 years after the onset of schizophrenia.

Psychiatry Research: Neuroimaging, 130, 57-70.

Donahoe, C. P., Carter, M. J., Bloem, W. D., Hirsch, G. L., Laasi, N., & Wallace, C. J. (1990). Assessment of interpersonal problem-solving skills, *Psychiatry, 53,* 329–339.

Doty, R. L., Shaman, P., & Dann, M. (1984). Development of the University of Pennsylvania Smell Identification Test: a standardized microencapsulated test of olfactory function. *Physiology and Behaviour, 32,* 489-502.

Droge, W., & Schipper, H. M. (2007). Oxidative stress and aberrant signaling in aging and cognitive decline. *Aging Cell, 6,* 361-70.

Drury, V., Birchwood, M., Cochrane, R., & Macmillan, F. (1996). Cognitive therapy and recovery from acute psychosis: a controlled trial. I. Impact on psychotic symptoms. *British Journal of Psychiatry, 169,* 593-601.

Fagerlund, B., Pagsberg, A. K., & Hemmingsen, R. P. (2006). Cognitive deficits and levels of IQ in adolescent onset schizophrenia and other psychotic disorders. *Schizophrenia Research, 85,* 30-39.

Fowler, D., Garety, P., Kuipers, E. (1995). *Cognitive behaviour therapy for psychosis: Theory and practice.* Chichester: Wiley.

Frith, C. D. (1992). *The cognitive neuropsychology of schizophrenia.* UK: Lawrence Erlbaum Associates Publishers.

Frith, C. D., Leary, J., Cahill, C., & Johnstone, E. C. (1991). Performance on psychological tests: Demographic and clinical correlates of the results of these tests. *British Journal of Psychiatry, 159,* 26 – 29.

Freeman, D., & Garety, P. A. (2004). *Paranoia: The Psychology of Persecutory delusions.* Sussex, UK: Psychology Press.

Freeman, D., Garety, P. A., Kuipers, E., Fowler, D., & Bebbington, P. E. (2002). A

cognitive model of persecutory delusions. *British Journal of Clinical*

Psychology, 41, 331-347.

Garety, P. A., Fowler, D., Kuipers, E., Freeman, D., Dunn, G., Bebbington, P. E.,

Hadley, C., & Jones, S. (1997). The London-East Anglia randomised controlled

trial of cognitive behaviour therapy for psychosis II: Predictors of outcome.

British Journal of Psychiatry, 171, 420-426.

Garety, P. A, Hemsley, D. R., & Wessely, S. (1991). Reasoning in deluded

schizophrenic and paranoid patients: Biases in performance on a probabilistic

inference task. *Journal of Nervous and Mental Disorder, 179,* 194-201.

Gataz, W. F., Kohlmeyer, K., & Gasser, T. (1991). Computer tomographic studies in

schizophrenia. In H. Hafner, & W.F. Gattaz (eds.), *Search for the causes of*

schizophrenia (vol. II). Berlin: Springer.

Gold, J. M. & Harvey, P. D. (1993). Cognitive deficits in schizophrenia. *The Psychiatric*

Clinics of North America, 16, 295-312.

Good, K. P., Whitehorn, D., Rui, Q., Milliken, H., & Kopala, L. C. (2006). Olfactory

identification deficits in first-episode psychosis may predict patients at risk for

persistent negative and disorganized or cognitive symptoms. *American Journal*

of Psychiatry, 163, 932-933.

Grant, C., Addington, J., Addington, D., & Konnert, C. (2000). Social functioning in

first and multi-episode schizophrenia. *Schizophrenia Research, 41,* 165.

Green, M. F. (1996). What are the functional consequences of neuro-cognitive deficits in schizophrenia? *American Journal of Psychiatry, 153,* 321-330.

Green, M. F. (1998). *Schizophrenia from a neurocognitive perspective.* Allyn & Bacon, Boston.

Green, M. F., Kern, R. S., Braff, D. L., Mintz, J. (2000). Neurocognitive deficits and functional outcome in schizophrenia: Are we measuring the "right stuff"? Schizophrenia Bulletin 26 (1), 119-136.

Green, M. F., Nuechterlein, K. H., Gold, J. M., Barch, D. M., Cohen, J., Essock, S., Fenton, W. S., Frese, F., Goldberg, T. E., Heaton, R. K., Keefe, R. S. E., Kern, R. S., Kraemer, H., Stover, E., Weinberger, D. R., Zalcman, S., & Marder, S. R. (2004). Approaching a Consensus Cognitive Battery for Clinical Trials in Schizophrenia: The NIMH-MATRICS Conference to select cognitive domains and test criteria. *Biological Psychiatry, 56,* 301-307.

Hafner, H., Maurer, K., Loffler, W., & Riecher-Rossler, A. (1993). The influence of age and sex on the onset and early course of schizophrenia. *British Journal of Psychiatry, 162,* 80-6.

Hammer, M. A., Katsanis, J., & Iacono, W. G. (1995). The relationship between negative symptoms and neuropsychological performance. *Biological Psychiatry, 37,* 828–830.

Hare, E. H., & Moran, P. A. P. (1979). Raised parental age in psychiatric patients: evidence for the constitutional hypothesis. *British Journal of Psychiatry, 134,* 169-77.

Harvey, P. D., Rabinowitz, J., Eerdekens, M., & Davidson, M. (2005). Treatment of

cognitive impairment in early psychosis: a comparison of Risperidone and

Haloperidol in a large long-term trial. *American Journal of Psychiatry, 162,*

1888-1895.

Johnstone, E. C., Crow, T. J., Frith, C. D., Husband, J., & Kreel, L. (1976). Cerebral

ventricle size and cognitive impairment in chronic schizophrenia. *Lancet, ii,* 924-

926.

Johnstone, E. C., Crow, T. J., Johnson, A. L., & Macmillan, J. F. (1986). The Northwick

Park Study of first episodes of schizophrenia. I. Presentation of the illness and

problems relating to admission. *British Journal of Psychiatry, 148,* 115-20.

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

Kallmann, F. J. (1938). *The genetics of schizophrenia.* New York: Augustin.

Kallmann, F. J. (1946). The genetic theory of schizophrenia. *American Journal of*

Psychiatry, 103, 309-22.

Kay, S. R., Fiszbein, A., & Opler, L. A. (1987). The Positive and Negative Syndrome

Scale for schizophrenia. *Schizophrenia Bulletin, 13,* 261-276.

Keefe, R. S. E. (2004). Treatment of cognitive deficits in first episode schizophrenia. *International Journal of Neuropsychopharmacology, 7*, S87.

Kraeplin, E. (1896). Psychiatrie, 5th ed. Translated in part 1986 in: Cutting, J. C. & Shepherd, M. (eds.). *Schizohprenia: the origin and development of its study in Europe*. Cambridge: Cambridge University Press.

Lieberman, J., Chakos, M., Wu, H., Alvir, J., Hoffman, E., Robinson, D., & Bilder, R. (2001). Longitudinal study of brain morphology in first episode schizophrenia. *Biological Psychiatry, 49*, 487-499.

Loebel, A. D., Lieberman, J. A., Alvir, J. M., Mayerhoff, D. I., Geisler, S. H., & Szymanski, S. R. (1992). Duration of untreated psychosis and outcome in first-episode schizophrenia. *American Journal of Psychiatry, 149*, 1183-88.

Mass, R. (2000). Characteristic subjective experiences of schizophrenia, *Schizophrenia Bulletin, 26*, 921–931.

Mass, R. (2005). Eppendorf Schizophrenia Inventory (ESI) vs. Frankfurt Complaint Questionnaire (FCQ). Direct comparison in a clinical trial. *Nervenarzt, 76*, 1109-16.

McGlasham, T. H., & Fenton, W. S. (1992). The positive and negative distinction in schizophrenia - review of natural history validators. *Archives of General Psychiatry, 49*, 63-72.

McGorry, P. D. & Edwards, J. (1997). *Early psychosis training pack*. Macclesfield, UK: Gardiner-Caldwell Communications Ltd.

McGorry, P. D., Edwards, J., Mihalopoulos, C., Harrigan, S. M., & Jackson, H. J.

(1996). EPPIC: an evolving system of early detection and optimal management.

Schizophrenia Bulletin, 22, 305-26.

Mohamed, S., Paulsen, J. S., O'Leary, D., Arndt, S., & Andreasen, N. (1999).

Generalized cognitive deficits in schizophrenia: A study of first-episode patients.

US: American Medical Association.

Moritz, S., Krausz, M., Gottwalz, E., Lambert, M., Perro, C., Ganzer, S., & Naber, D.

(2000). Cognitive dysfunction at baseline predicts symptomatic 1-year outcome

in first-episode schizophrenics. *Psychopathology, 33,* 48-51.

Moritz, S., Lambert, M., Andresen, B., Bothern, A., Naber, D., & Krausz, M. (2001).

Subjective cognitive dysfunction in first-episode and chronic schizophrenic

patients. *Comprehensive Psychiatry, 42,* 213-6.

Nasrallah H. A., Andreasen N. C., Coffman J. A., Olson S. C., Dunn V. D., Ehrhardt J.

C., & Chapman, S. M. (1986). A controlled magnetic resonance imaging study

of corpus callosum thickness in schizophrenia. *Biological Psychiatry, 21,* 274-

82.

Nelson, H. E. (2005). *Cognitive Behavioural Therapy with Delusions and*

Hallucinations: A Practice Manual. 2nd edition. Cheltenham, UK: Nelson

Thornes.

O'Callaghan, E., Larkin, C., Redmond, O., Stack, J., Ennis, J. T., & Waddington, J.

(1988). Early onset schizophrenia after teenage head injury. *British Journal of*

Psychiatry, 153, 394- 396.

Office for National Statistics (2006). Surveys of Psychiatric Morbidity among Adults in

Great Britain.

Owen, F., Cross, A. J., Crow, T. J., Longden, A., Poulter, M., & Riley, G. J. (1978).

Increased dopamine-receptor sensitivity in schizophrenia. *Lancet, ii,* 223-226.

Prikryl, R., Kasparek, T., Ceskova, E., Kucerova, H., Ondrusova, M., Drybcak, P., &

Lorenc, M. (2003). The cognitive dysfunction in the first episode of

schizophrenia. *Schizophrenia Research, 60,* 178-179.

Rosenthal, D., Wender, P. H., Kety, S. S., Schulsinger, F., Welner, J., & Østergaard, L.

(1968). Schizophrenics' offspring reared in adoptive homes. In *The*

Transmission of Schizophrenia, ed. D. Rosenthal, & S. S. Kety, pp. 377-92.

Oxford: Pergamon Press.

Rossi, A., De Cataldo, S., Di Michele, V., Manna, V., Ceccoli, S., Stratta, P., &

Casacchia, M. (1990). Neurological soft signs in schizophrenia. *British Journal*

of Psychiatry, 157, 735–739.

Schröder, J., Niethammer, R., Geider, F. J., Reitz, C., Binkert, M., Jauß, M., & Sauer, H.

(1992). Neurological soft signs in schizophrenia. *Schizophrenia Research, 6,* 25–

30.

Schuepbach, D., Keshavan, M. S., Kmiec, J. A., & Sweeney, J.A. (2002). Negative

symptom resolution and improvements in specific cognitive deficits after acute

treatment in first-episode schizophrenia. *Schizophrenia Research, 53,* 249-261.

Schuepbach, D., Sanders, R. D., & Keshavan, M. S. (2002). Cognitive impairments

correlate with specific neurological soft signs in first-episode schizophrenia.

*Biological Psychiatry, 51,*26S.

Seeman, P., Lee, T., Cahu-Wong, M., & Wong, K. (1976). Antipsychotic drug doses

and neuroleptic/dopamine receptors. *Nature (London), 261,* 717-719.

Sharma, T., & Harvey, P. (2000). *Cognition in schizophrenia.* London: Oxford

University Press.

Shelton, R. C., Karson, C. N., Doran, A. R., Picker, D., Bigelow, L. W., Weinberger, D.

R. (1988). Cerebral structural pathology in schizophrenia: evidence for a

selective prefrontal cortex deficit. *American Journal of Psychology, 145,* 154–

163.

Slater, E., & Cowie, V. (1971). *Genetics of mental disorders.* London: Oxford

University Press.

Spitzer R. L., Williams J. B. W., Gibbon M., & First, M. B. (1992). The Structured

Clinical Interview for DSM-III-R (SCID -I): history, rationale, and description.

Archives of General Psychiatry, 49, 624–629.

Steen, R. G., Mull, C., McClure, R., Hamer, R. M., & Lieberman, J. A. (2006). Brain

volume in first-episode schizophrenia: systematic review and meta-analysis of

magnetic resonance imaging studies. *British Journal of Psychiatry, 188,* 510-

518.

Sülwood, L. (1986). *Frankfurter Beschwerde-Frageboden (FBF).* Springer, Heidelberg.

Townsend, L. A., Malla, A. K., & Norman, R. M. G. (2001). Cognitive functioning in

stabilised first-episode patients. *Psychiatry Research, 104,* 119-131.

Turner, S. M., Beidel, D. C., & Dancu, C. V. (1996). *Social phobia and anxiety inventory: manual.* Toronto, Ontario: Multi-Health Systems Inc.

Vaugn, C. E., & Leff, J. P. (1976). The influence of family and social factors on the course of psychiatric illness: a comparison of schizophrenic and depressed patients. *British Journal of Psychiatry, 129,* 125-37.

Ventura, M. A., Green, M. F., Shaner, A., & Liberman, R. P. (1993). Training and quality assurance with the brief psychiatric rating scale: "The drift buster". *International Journal of Methods in Psychiatric Research, 3,* 221-244.

Verdoux, H., Liraud, F., Assens, F., Abalan, F., & Van-Os, J. (2002). Social and clinical consequences of cognitive deficits in early psychosis: a two-year follow-up study of first-admitted patients. *Schizophrenia Research, 56,* 149-159.

Vita, A., De Peri, L., Silenzi, C., & Dieci, M. (2006). Brain morphology in first-episode schizophrenia: a meta-analysis of quantitative magnetic resonance imaging studies. *Schizophrenia Research, 82,* 75-88.

Voges, M., & Addington, J. (2005). The association between social anxiety and social functioning in first episode psychosis. *Schizophrenia Research, 76,* 287-292.

Walker, E., & Lewine, R. J. (1990). Prediction of adult-onset schizophrenia from childhood home movies of the patients. *American Journal of Psychiatry, 147,* 1052-1056.

Weickert, T. W., Goldberg, T. E., Gold, J. M., Bigelow, L. B., Egan, M. F., & Weinberger, D. R. (2000). Cognitive impairments in patients with schizophrenia

displaying preserved compromised intellect. *Archives of General Psychiatry, 57,* 907-913.

Weinberger, D. R. (1988). Premorbid neuropathology in schizophrenia. *Lancet, ii,* 959-960.

Wing, J. K. (1978). *Schizophrenia: Towards a new synthesis.* London: Academic Press.

Wyatt, R. J. (1991). Neuroleptics and the natural course of schizophrenia. Schizophrenia Bulletin, *17,* 325–351.

Yung, A. R., & McGorry, P. D. (1996). The prodromal phase of first-episode psychosis: past and current conceptualizations. *Schizophrenia Bullletin, 22,* 353-70.

Part 2: Empirical Paper

Brief Cognitive Assessment and Theory of Mind in First Episode

Psychosis and their Relation to Community Function

Abstract

This study explored the relationship between cognitive and community function in patients with first episode psychosis. Cognitive function was assessed using the 'Brief Cognitive Assessment' (BCA) and Theory of Mind (ToM) tasks. The BCA assesses verbal memory, attention and executive functioning. Twenty-four patients from two early intervention in psychosis services were recruited to complete the tests that comprise the BCA (Trails Making Test, the Hopkins Verbal Learning Test and Verbal Fluency). ToM was assessed by means of the Hints Task, Picture Sequencing Stories and ToM Stories. Community function was assessed using the Multnomah Community Ability Scale, which was rated by the participants' care-coordinators. A measure of symptoms (using the BPRS) and a brief measure of current IQ were also completed. On the BCA only Trails A was significantly impaired relative to norms. Performance on the BCA and the Hints Task were associated with community functioning, and these findings provide some support for the notion that the BCA and Hints Task may be useful predicators of community functioning.

1. Introduction

Many studies have demonstrated neuropsychological deficits in psychosis and a continuation of these deficits over the course of the illness (e.g. Gold & Harvey, 1993; Green et al., 2004). There is some evidence that these deficits are related to community function (Green, 1996). Research in first episode psychosis (FEP) may give a clearer understanding of the pattern and range of deficits at the beginning of the illness, where confounding factors such as medication and hospitalisation are less significant than in more chronic patients (McGorry & Edwards, 1997). Intervening in FEP may reduce the long-term social and clinical effects of this illness (McGorry & Edwards, 1997).

1.1 Cognitive Assessment

Several studies have found cognitive deficits in the areas of attention, executive function, memory and processing speed in first episode populations (Sweeney, Haas, Kelip, & Long, 1991; Hutton et al., 1998; Riley, et al., 2000), which suggests that these deficits are present at the beginning of psychotic illness. Discrepancies in scores between the National Adult Reading Test (NART; Nelson, 1982) and Wechsler Abbreviated Scale of Intelligence (3rd edition) (WAIS III; Wechsler, 1997) suggest declines in IQ following onset of FEP (Townsend, Malla, & Norman, 2001). Research suggests that cognitive difficulties have important implications for social and community functioning in psychosis (Green, 1996), though this has been less well explored in first episode populations. This is especially pertinent for those experiencing their first psychotic episode at an age where post-secondary studies or occupational endeavours are a major concern (Townsend, Norman, Malla, Rychlo, & Ahmed, 2002).

Despite the established relationship between cognition and functional outcome in psychosis, cognitive testing is uncommon in psychiatric outpatient settings (Velligan et al., 2004). This may be because such tests require a high degree of expertise and time commitment to be carried out, scored and written up. In order to make cognitive testing more feasible in clinical practice, several researchers have focused their attention on the development of brief cognitive assessment batteries for use with individuals with schizophrenia. One such battery, the Brief Cognitive Assessment (BCA; Velligan et al., 2004), utilises standardised tests that examine a variety of cognitive domains in a limited fashion. Normative data are available for all of the tests in this battery. The BCA has been tested for reliability and validity in an outpatient sample of schizophrenia patients in the USA (Velligan et al., 2004). A 'global cognitive function score' (GCFS) can be calculated by averaging the z-scores achieved for each of the three subtests that make up the BCA. The GCFS is taken as an index of the general level of cognitive functioning. Velligan et al. (2004) found that the GFCS was correlated with social and community functioning in their patient sample. The BCA has yet to be used with FEP patients. The cognitive test data from the BCA may be used to monitor change over time, to evaluate the effect of different anti-psychotics on cognitive function and to evaluate whether or not cognitive rehabilitation is warranted (Velligan et al., 2004).

1.2 Theory of Mind (ToM)

ToM may be defined as the capacity to infer one's own and other persons' mental states (thoughts, intentions and beliefs) and to use this capacity to predict and understand behaviours (Premack & Woodruff, 1978). Many studies have confirmed that individuals with schizophrenia have ToM deficits (see Brüne, 2005, and Harrington,

Siegert, & McClure, 2005, for reviews). There is much debate over whether ToM

involves a separate 'module' of functioning or is an aspect of executive functioning. In

a study by Brüne (2005), he found independent effects of ToM and executive

functioning in patients with schizophrenia and a closer association between ToM and

social functioning than between executive ability and social functioning. Based on a

review of studies, Abu-Akel (2003) proposed that ToM ability depends on working

memory, executive function and other general cognitive abilities. These contrasting

findings in the literature may lead to confusion regarding the identification of those at

risk of ToM deficits.

There has been some difference of opinion regarding whether ToM is a trait or

state marker in schizophrenia, and if a state marker, what psychotic symptoms are

associated with it. The research evidence to date is weighted towards impaired ToM

being a state marker, and many studies have tried to elucidate the symptoms associated

with it. These findings may have important implications as ToM deficits may be

treatable via the symptoms associated with them. Frith and Corcoran (1996) compared

ToM in patients with schizophrenia and controls and found that those with paranoid

delusions and negative features of schizophrenia were impaired on questions concerning

mental states; whereas patients with symptoms of passivity (i.e. delusions of control)

and those in remission did not differ from controls. Pickup and Frith (2001) found

similar results whereby patients with behavioural signs were impaired relative to

controls on ToM, and remitted patients performed as well as controls. However, in

contrast to Frith and Corcoran's findings they found only weak evidence that a subgroup

with paranoid symptoms had ToM impairments. Sarfati and Hardy-Baylé (1999)

showed that those with thought and speech disorganisation showed a deficit in ToM

relative to manic and control participants. From these studies it can be concluded that

psychotic symptoms including behavioural signs and speech disorganization are

associated with impaired ToM in schizophrenia, and those who are asymptomatic do not

have ToM impairments. The association between paranoid symptoms and impaired

ToM in psychosis remains unclear.

ToM has not been studied extensively in first episode populations. A recent

study by Inoue et al. (2006) found that those in remission from first episode

schizophrenia showed impaired ToM relative to controls. In contrast to the widely held

opinion that impaired ToM is a state marker and may be associated with specific

symptoms, the authors concluded that their study may provide evidence that impaired

ToM is a trait marker in schizophrenia. It is clear from the paucity of literature on ToM

in FEP that more research is needed in this area.

There is some evidence to suggest that ToM ability may be implicated in

community functioning in schizophrenia and may have more predictive value in relation

to social and interpersonal skills than other cognitive domains such as memory, attention

and executive functioning (Brüne, 2005; Pollice et al., 2002). More research is needed

to examine the relationship between cognitive deficits, symptomatology and ToM, and

their relation to community function in people with a psychotic illness.

1.3 Social and community functioning

An understanding of the neuropsychological deficits associated with FEP could

have important clinical applications in the future, as these deficits may be associated

with patients' ability to function in the community. The most consistent findings in the

literature are based on studies of chronic schizophrenia populations where community

functioning was correlated with verbal memory, vigilance and executive functioning (Green, 1996; Green, Kern, Braff & Mintz, 2000). There has been only limited evidence of associations between symptoms and community functioning (Green, 1996). Most studies have examined patients with long durations of schizophrenia where factors such as hospitalisation, medication effects and age may have confounded results on social and community functioning measures. Because of these factors, more recent research has focused on those with a first episode of psychosis.

There have been mixed results regarding the contribution of cognitive deficits to social and community outcome in FEP. This may be because of the variety of assessment instruments used and the different durations of follow-up. Milev, Ho, Arndt, and Andreasen (2005) conducted a 7-year follow-up of first episode schizophrenia patients. They found that cognition (verbal memory, processing speed, and attention) and negative symptoms at baseline were related to subsequent outcome. However, negative symptoms explained more of the variance in outcome than cognition. Other studies did not incorporate a measure of symptomatology into their design. Fujii and Wylie (2003) found that verbal memory explained nearly half of the variance in community outcome at approximately 15 years follow-up. Jaeger and Douglas (1992) found a correlation between perseverative errors on an executive function task and social adjustment at 18 month follow-up. In summary, as in the case of chronic schizophrenia populations, community function may be related to verbal memory and executive functioning in those with FEP. Symptoms may also contribute to community function, which perhaps has been overlooked in the research as evidenced by the small number of studies that have incorporated a measure of symptomatology into their design.

1.4 Conclusion

Despite the existence of widespread cognitive deficits and their association with community function in schizophrenia, cognitive assessment is not routinely carried out in clinical practice. Advances in the development of antipsychotic medications have improved symptom reduction, while cognitive interventions have received much less attention. A holistic approach to treatment of the cognitive deficits as well as the symptoms of psychosis might improve patient care significantly and reduce the long term social consequences of psychosis. Cognitive rehabilitation has been increasingly cited as an essential component of comprehensive treatment (Storzbach & Corrigan, 1996; Kern & Green, 1998). This is potentially very important with first episode populations where cognitive rehabilitation could have a significant impact on the person's quality of life and community function.

1.5 Hypotheses

This study will explore some of the issues described above in more detail. It was predicted that: 1) Patients with FEP will be significantly impaired in their performance on the BCA relative to normative data. This is based on Velligan et al.'s (2004) study where patients with schizophrenia were significantly impaired relative to norms on all subtests of the BCA; 2) BCA performance will correlate positively with community functioning, as measured by the Multnomah Community Ability Scale (MCAS) (Barker, Barron, McFarland, & Bigelow, 1994). Again, this is based on Velligan et al.'s (2004) finding that the BCA was positively and moderately correlated with measures of functional outcome; 3) In a regression model, the BCA will predict more of the variance in community functioning than symptom ratings. This is consistent with Green (1996)

who found limited evidence of associations between symptoms and community

functioning in a review of studies; 4) The BCA will be more closely related to level of

community function than IQ, as measured by the Wechsler Abbreviated Scale of

Intelligence (WASI) (Psychological Corporation, 1999). This is supported by the idea

that people with schizophrenia are specifically impaired in attention, memory, and

executive function beyond generalised cognitive impairment (i.e. IQ) (Green, 1996;

Green et al., 2000); 5) Based on the results of the studies carried out by Pollice et al.

(2002) and Brüne (2005) it is predicted that scores on the ToM tasks will be a significant

independent predictor of community functioning.

2. Method

2.1 Subjects

A power calculation (Cohen, 1992) to detect a large effect size with an alpha

level of .05 using regression analyses with 4 predictors recommended a sample size of

40. However, due to recruitment constraints 24 participants were recruited. The

participants had a DSM-IV (American Psychiatric Association, 1994) diagnosis or

schizophrenia or related disorder and were recruited from 2 early intervention services

(EISs) in London. All participants were fluent in English (for a full description of the

inclusion criteria see Appendix 1). The researcher (AW) met with each care coordinator

in the services and identified suitable candidates from their caseload. The care

coordinators were given a handout outlining the process of referring suitable candidates

to the study (Appendix 1). The care coordinators gave the identified patients an

information leaflet about the study (Appendix 2) and asked for consent for the researcher

to contact them to answer questions and to arrange a time to meet if interested.

The EIS's referral criteria were as follows: patients must be aged 16-30; a

resident of the local catchment area; experiencing psychotic symptoms; and not in

previous contact with secondary mental health services for longer than 1 year. The

inclusion criteria for this study required patients to be in a period of clinical stability.

The exclusion criteria were a poor understanding of English or a history of head injury,

neurological disability or leucotomy. Demographic variables for the sample are shown

in Table 1.

Table 1: Sample demographics

Mean age (years)	22.80
Mean education (years)	13.21
Gender	*n*
Males	17
Females	7
Ethnicity	*n*
White British	4
White Other	1
Black British Caribbean	6
Black British African	3
Asian British	10
Spoke English as a first language (*n*)	12
Employment	*n*
Employed	5

Employment (contd.)	*n*
Unemployed	11
Student	8
Residential Status	*n*
Living with family	16
Living alone	6
Living with friends	2
Diagnosis	*n*
First psychotic episode	3
Schizophrenia	5
Acute Psychotic Episode	10
Paranoid Schizophrenia	4
Drug Induced Psychosis	2
Mean duration of illness (months)	19.88
Mean age of onset (years)	20.54
Mean number of relapses	0.42
Medication	*n*
Atypical antipsychotics	20
Typical antipsychotics	0
Depot medication	1
Mean Risperidone Equivalent Dose	2.74
Mood stabilizers	1
Antidepressants	3
Anxiolytics	2

2.2 Design and Procedure

The study was approved by East London & the City Research Ethics Committee (Appendix 3). The assessment took place in a quiet room at the EIS base. Informed consent (Appendix 4) was obtained from all participants prior to their inclusion in the study. Participants also had the option of consenting to have a summary of their results included in their file at the early intervention service (see Appendix 5 for a report template). Only one participant did not consent to this. Participants completed a 60-90 minute interview with the researcher. Demographic information (see Appendix 6 of questionnaire used) was collected before the participants completed the study measures. In all cases the measures were completed in one session. Participants were paid £15 for taking part. Within 2 weeks of each participant's assessment the researcher met with their care coordinator to provide them with a report regarding the participant's performance (if the participant consented to this) and to complete the measure of social and community functioning.

2.3 Measures

The following measures were completed in the order below.

2.3.1 Brief cognitive assessment (BCA; Velligan et al., 2004)

The BCA consists of three standard tests widely used in clinical neuropsychology, that tap the cognitive domains known to be impaired in schizophrenia, namely executive function, memory, attention and processing speed. In Velligan et al.'s (2004) study, the BCA showed good inter-item consistency (Cronbach's $\alpha = 0.77$; n = 305), and the test-retest reliability was consistent with published data.

2.3.1.1 The Trail Making Test (TMT; Reitan & Wolfson, 1985)

The TMT is a measure of visuomotor sequencing and comes in 2 parts. Trails A

measures psychomotor speed while Trails B measures psychomotor speed and the ability

to inhibit inappropriate responses. Trails A consists of a series of numbered circles (1–

25), scattered at random on a worksheet. The participant was required to draw a line

between the circles in numerical order as quickly as possible. Trails B has both numbers

and letters arrayed in a random order. The participant was required to draw lines

between numbers and letters in an alternating sequence (i.e. 1-A-2-B-3-C etc.), again as

quickly as possible.

In this task the score is based on time alone. The researcher pointed out errors if

they occurred, and the participant was required to correct the error before continuing

with the test. A cut-off time of 300 seconds was imposed to allow comparison with

Velligan et al.'s (2004) study.

2.3.1.2 Verbal Fluency (Benton & Hamsher, 1978)

Verbal Fluency is a test of executive function. In the first part of the task the

participant was asked to generate as many words as possible beginning with certain

letters of the alphabet (F, A and S). In the second part of the task the participant was

asked to name as many different types of animals as possible. The score for each part

was the total number of words generated in one minute.

2.3.1.3 The Hopkins Verbal Learning Test (HVLT) (Brandt, 1991)

70

The HVLT is a list learning exercise that assesses verbal learning over 3 trials. It consists of a list of twelve nouns that are drawn from three different semantic categories with four nouns coming from each category. The list was read aloud to the participant, and their free recall of the list was recorded. The procedure was repeated twice making a total of three trials. After a delay of 20-25 minutes, the participant was asked if they could recall any words from the list. The score was the total number of words generated from the 3 learning trials.

2.3.2 Theory of Mind (ToM) Tasks

There were three ToM tasks used in this study.

2.3.2.1 ToM Stories (Frith & Corcoran, 1996)

Four ToM stories were read aloud to each participant. Two of the stories were 'first order' in which a story character has a false belief about a particular situation. The other two stories were 'second order' in which a story character has a false belief about the belief of another character. While the stories were read aloud, corresponding cartoon pictures were shown to each participant. For each story, the participant had to answer two questions: The first question assessed understanding of the character's false belief and required intact ToM. The second question assessed memory for the story. Scores were only considered if the participant correctly answered the memory question (Frith & Corcoran, 1996). The ToM Stories score reflected the percentage of ToM questions that were answered correctly when their corresponding memory question was also answered correctly.

2.3.2.2 Picture Sequencing Task (Langdon et al., 1997)

This task consists of stories depicted in black and white drawings. Each story is presented on four cards. There are two practice sequences and three experimental sequences: social script, mechanical and false belief. Social script stories depict people acting out everyday social routines and test knowledge of logical routines using social script knowledge (involving no appreciation of mental states). Mechanical stories depict cause and effect sequences and test ability to infer causal relations. False belief stories depict the story character acting on the basis of false beliefs; these stories are the test of Theory of Mind. There were four stories in each experimental sequence, and the story presentation was randomized (Langdon et al., 1997). The participant was presented with each set of story cards placed face down and was required to turn the cards over and to order them in a logical sequence. The order of card placement and the time taken to complete each story was recorded. The scores were the average time taken to complete each sequence and the average score for each sequence.

2.3.2.3 Hints Task (Corcoran, Mercer, & Frith, 1995)

This task consists of ten short vignettes presenting an interaction between two characters. Each vignette was read aloud to the participant, and was repeated if the participant asked. At the end of each vignette, one of the characters gave an obvious hint. The participant was asked to infer what the character really meant by what he/she said. A correct response was given a score of 2. If an inference was not made or if an inappropriate conclusion was drawn, the participant was given additional information in the form of a more obvious hint. If a correct response was given at this stage, a score of

one was given. If the participant failed to infer what the character meant a score of zero was given. The recorded score was the total score for the ten vignettes.

2.3.3 The Wechsler Abbreviated Scale of Intelligence (WASI) (Psychological Corporation, 1999)

The WASI provides a reliable and brief test of IQ comparable to the WAIS-III (Wechsler, 1997). The two-subtest form of the WASI was used, which can be administered in about 15 minutes. This includes vocabulary and matrix reasoning and provides an estimate of full scale IQ. The two-subtest form of the WASI has shown good reliability (0.96) and test-retest reliability (0.88) (Psychological Corporation, 1999).

2.3.4 The Brief Psychiatric Rating Scale (BPRS; Ventura, Green, Shaner, & Lieberman, 1993)

The BPRS is a clinician-rated tool designed to assess the nature and extent of psychopathology in patients with psychotic illness over the two weeks prior to interview. Participants are rated on 24 different symptom constructs according to a seven-point scale ranging from 'not present' to 'extremely severe'. A total score is obtained from these ratings with lower scores indicating better functioning. In the analyses, the factor structure used by Velligan et al. (2005) for the negative symptom/retardation factor (blunted affect, emotional withdrawal, psychomotor retardation) and the psychosis factor (unusual thought content, hallucinations, conceptual disorganisation, bizarre behaviour) was used. The intraclass correlation coefficient of 0.95 for the BPRS items is excellent (Community Mental Health Evaluation Initiative (CMHEI) Working Paper, 2001). A

dual-rating technique was incorporated into the research design with the aim of

maximising the reliability of ratings. This involved the principal researcher and a

clinical psychologist (GP) rating a subset of the interviews independently and comparing

ratings. However, no participants consented to being audio taped for dual rating, so it

was not possible to dual rate the BPRS.

2.3.5 Global Assessment of Functioning Scale (GAF; American Psychiatric Association, 1994)

This measure is a tool for Axis V assessment in DSM-IV (APA, 1994). It was

completed by the researcher to assess overall functioning at the time of assessment. The

GAF scale is a 100-point scale that measures patients' overall level of psychological,

social, and occupational functioning on a hypothetical continuum. The upper end of the

scale indicates superior functioning, while the lower end of the scale indicates severe

impairment of functioning. The inter-rater reliability of the GAF is good (ICC/k=0.60–

0.75) (Fleiss, 1981).

2.3.6 Multnomah Community Ability Scale (MCAS) (Barker, Barron, McFarland, & Bigelow, 1994)

The MCAS was completed by the participant's care coordinator. It consisted of

17 items used to assess disability associated with chronic mental illness over the

previous month in persons living in the community. The MCAS provided a global

measure of functioning across four domains: interference with functioning, adjustment

to living, social competence and behavioural problems. Each item was rated on a five

point scale with higher scores indicating better functioning. The researcher was present during ratings to clarify what the items meant and to answer any questions posed by the care-coordinators. The reliability of the MCAS is good with an intraclass correlation value of 0.97 (Community Mental Health Evaluation Initiative (CMHEI) Working Paper, 2001).

3. Results

3.1 Scores on the measures

The mean (SD) administration time for the BCA was 14.58 (2.28) minutes. The mean (SD) time taken to complete the whole experimental battery was 68.96 (10.63) minutes. Following the procedure used in Velligan et al.'s (2004) study, raw scores for each of the subtests of the BCA were converted into z-scores by subtracting the obtained score on each test from the mean score for the appropriate normative sample and dividing this by the standard deviation for that normative sample. The z-scores therefore reflect the degree of deviation of the patient group from the published normative data. A global cognition function score (GCFS) was then calculated by averaging the five individual z-scores. Normative data for TMT were derived from Fromm-Auch and Yeudall (1983) for 18-19 year olds and from Kennedy (1981) for 20-30 year olds. Normative data for Verbal Fluency were derived from Yeudall, Fromm, Reddon and Stefanyk (1986) and Selnes et al. (1991) for letter and category fluency respectively, and HVLT norms were obtained from Brandt and Benedict (2001). Norms were stratified by age for each subtest except category fluency where norms were stratified by years of education. The mean z-scores and standard deviations for the individual subtests and the GCFS are shown in Table 2. Before calculating the GCFS, one outlier on Trails B was

recoded to one score higher than the next highest score following the procedure of

Howell (1992).

Table 2. Mean z-scores and standard deviations for the BCA subtests and GFCS

Measure	Mean z- Score	Standard Deviation (SD)
Trails A	-2.09*	1.78
Trails B	-1.54	1.97
Verbal Fluency Letters (FAS)	-1.07	1.02
Verbal Fluency Categories (Animals)	-1.31	1.01
Hopkins Verbal Learning Test (HVLT)	-1.71	1.31
BCA – Global Cognition Function Score (GCFS)	-1.52	0.85

*> 1.96, $p < 0.05$ (Tabachnick & Fidell, 2001)

The patient group was impaired relative to norms on all of the subtests of the

BCA. They were most impaired on Trails A with a score of approximately 2 SDs below

the mean of the normative sample. Consistent with Velligan et al.'s (2004) finding the

patients were less impaired on Trails B than A. In addition, the patients in this sample

showed a similar level of impairment on Trails A and B to Velligan et al.'s (2004) more

chronic schizophrenia sample that had mean z-scores of -2.10 and -1.62 respectively.

The patients were least impaired relative to norms on Verbal Fluency Letters (FAS).

Following Velligan et al.'s (2004) study, the reliability of the BCA in the present

sample was measured by calculating its internal consistency. Using the z-scores

generated from each subtest, the inter-item consistency was modest (Cronbach's α = 0.58; n = 24). This compares to α = 0.77 in Velligan et al.'s (2004) study, although they had greater statistical power with a sample size of 305 participants.

3.2 Summary of scores on other measures

Descriptive statistics for the other measures used in the study are summarised in Table 3.

Table 3: Descriptive statistics for scores on the other measures

Measures	n	Range	Mean	Standard Deviation
WASI FSIQ	23	56 - 119	89.30	16.64
ToM Hints	24	8 - 20	15.67	3.24
ToM Stories total % correct	24	0 - 100	77.08	30.02
first order % correct	24	0 - 100	81.25	28.79
second order % correct	24	0 - 100	77.08	36.05
Picture Sequencing Task - time false belief /secs	24	16.52 - 44.70	28.89	9.54
social script /secs	24	10.89 - 40.24	21.31	6.71
mechanical /secs	24	14.80 - 50.8	27.66	9.52
Picture Sequencing Task – score				
false belief	24	0.50 - 6	3.91	1.41
social script	24	3.25 - 6	5.09	0.93
mechanical	24	0.75 - 6	4.68	1.23
Total MCAS score	24	47 - 80	67.46	8.22
Total BPRS score	24	26 - 57	35.71	8.17
Total GAF score	24	60 - 100	86.88	11.96

One data point was missing on the WASI because one participant did not

understand the Matrix Reasoning subtest and did not score any items correctly. The

patients were more impaired on the second order than the first order tasks, though this

78

difference was not significant (t (23) = 0.53, p = .60). On the picture sequencing task, the patients took longest to complete and scored relatively lowest on the false belief subtest. There were significant differences between the false belief score and the mechanical score (t (23) = -3.33, p = .003) and between the false belief score and the social script score (t (23) = 4.28, p = .001). There was no difference between the social script score and the mechanical score (t (23) = 1.58, p = .13). Regarding the time taken to complete each sequence, there were significant differences between the social script and mechanical sequences (t (23) = -5.71, p = .001) and between the social script and false belief sequences (t (23) = -6.99, p = .001). There was no difference between the false belief and mechanical times taken to complete the sequences (t (23) = 1.45, p = 0.16). Scores on the MCAS and GAF indicated generally good social and community functioning. The participants scored approximately 87 on the GAF, which is within the range of "good functioning in all areas". The maximum score on this measure is 100. There are no normative data on the MCAS for comparison. However, the mean score for a chronic schizophrenia group of patients (n=240) on the MCAS was 55.4 (SD 9.7), which suggested that the first episode sample of participants was functioning at a much higher level with a mean score of 67.46 (SD 8.22) (Barker et al., 1994), which indicated little psychopathology on the days of testing. The maximum score achievable on the BPRS is 85.

The IQ data did not deviate significantly from normality as shown by the Kolmogorov-Smirnov test (D = 0.09, p = 0.20), and the median and mode IQs were both 92. The mean IQ of 89.3 was lower than the average score of 95.2 (n= 92) quoted in Townsend et al.'s (2001) study of FEP.

Some distributions of variables appeared slightly skewed. However, when the significance test for skewness was performed (Tabachnick & Fidell, 2001) it was revealed that all distributions did not significantly differ from normality as summarized in Table 4.

Table 4: Significance test for skewed variables

Skewed variables	Skewness/ Standard error	z score	p
WASI FSIQ	-0.11 / 0.48	0.23	<1.96 n.s.
ToM Hints	-0.79 / 0.47	1.68	<1.96 n.s
Picture Sequencing Task - time false belief / secs	0.28 / 0.47	0.6	<1.96 n.s
social script /secs	0.81 / 0.47	1.72	<1.96 n.s
mechanical /secs	0.45 / .47	0.96	<1.96 n.s
Picture Sequencing Task - score false belief	-0.53 / 0.47	1.13	<1.96 n.s
social script score	-0.41 / 0.47	0.87	<1.96 n.s
mechanical score	-0.39 / 0.47	0.83	<1.96 n.s
Total MCAS score	-0.62 / 0.47	1.32	<1.96 n.s
Total BPRS score	0.56 / 0.47	1.19	<1.96 n.s
Total GAF	-0.79 / 0.47	1.68	<1.96 n.s
GCFS	-0.73 / 0.47	1.55	<1.96 n.s

3.3 Relation between GCFS and community functioning

In the following analysis, multivariate statistics were not used because of the relatively low sample size in the study. A Pearson's product-moment correlation between global cognition score on the BCA (GCFS) and community function (MCAS total score) revealed r (24) = 0.36; p = 0.04, 1-tailed, indicating a moderate statistically significant positive relationship between patients' GCFS and their community function. This is similar to Velligan et al.'s (2004) finding of a positive relationship between GCFS and MCAS (total score) (r (24) = 0.42; p < 0.02, 1-tailed).

3.4 Other correlates of GCFS

A Bonferroni correction was performed to reduce Type I error in these analyses; as 6 statistical tests were used, a p value of .008 (.05/6) was used to indicate statistical significance. To investigate the association between GCFS and IQ, a Pearson's product-moment correlation was calculated. This was highly statistically significant (r (24) = 0.59; p = .002, 1-tailed), showing as would be expected that these measures were strongly and positively correlated. GCFS was positively and significantly correlated with GAF score (r (24) = 0.62; p = .001, 1-tailed). The association between GCFS and the psychosis factor of the BPRS was not statistically significant (r (24) = -0.18; p = .20, 1-tailed), which is consistent with other studies that show little relation between the positive symptoms of psychosis and cognitive function (e.g. Gold & Harvey, 1993). The association between GCFS and the retardation factor of the BPRS was not significant (r (24) = -0.34, p = .054, 1-tailed), and neither was the association between GCFS and medication (in Risperidone equivalent dosage) (r (23) = -0.36; p = .04, 1-tailed).

Finally, the association between GCFS and duration of illness was not significant (r (24)

$= 0.10; p = .32$, 1-tailed).

3.5 Relation between ToM scores and MCAS

A Bonferroni correction was performed to reduce Type I error; as 7 statistical

tests were used in the analyses of ToM, a p value of 0.007 (0.05/7) was used to indicate

statistical significance. Only Hints total was positively and significantly associated with

MCAS total (r (24) = 0.493; p = 0.007, 1-tailed). The relationship between ToM

Stories total % correct and MCAS total score was not significant (r (24) = 0.37; p = .04,

1-tailed), and neither was the relation between MCAS total score and first order %

correct (r (24) = 0.42, p = .02, 1-tailed). Second order % correct was not significantly

associated with MCAS total (r (24) = 0.26, p = .11). None of the Picture Sequencing

Task average scores were related to MCAS total score: false belief sequence and MCAS

(r (24) = 0.38; p = .03, 1-tailed); mechanical sequence and MCAS (r (24) = 0.39; p =

.03, 1-tailed); social script sequence and MCAS (r (24) = 0.20; p = .17, 1-tailed).

3.6 Other correlates of community functioning

Pearson's product-moment correlations were calculated to investigate the

relations between community functioning (MCAS total score) and other variables. A

Bonferroni correction was performed to reduce Type I error; as 5 statistical tests were

used, a p value of 0.01 (0.05/5) was used to indicate statistical significance. There were

significant associations between IQ and MCAS (r (24) = 0.50; p = .007, 1-tailed), and

between the retardation factor of the BPRS and MCAS (r (24) = -0.51; p = .005, 1-

tailed). There were no associations between MCAS total score and the psychosis factor

of the BPRS (r (24) = -0.22; p = .16, 1-tailed) or duration of illness (r (24) = 0.12; p = .30, 1-tailed) or medication dosage (in Risperidone equivalents) (r (24) = -0.27, p = .11, 1-tailed).

3.7 Regression analysis

Analysis of the distribution of residuals revealed they were normal (D = 0.146, p = .20). A multiple regression was performed in order to explore the varying contributions of IQ, GCFS, retardation factor of BPRS and Hints score (ToM) in predicting community function (MCAS total score). The dependent variable in this regression was MCAS total score, and the independent variables were GCFS, IQ, retardation factor of BPRS and Hints. Overall, this model was significant (F (4, 18) = 3.23, p = 0.036), with adjusted R-square calculated to be 0.289, meaning that 28.9% of the variance in community function was explained by the independent variables GCFS, IQ, retardation factor of BPRS and Hints scores. In terms of independent effects, neither IQ ($Beta$ = 0.101, p = .76) nor GCFS ($Beta$ = 0.11, p = .67) nor retardation factor of BPRS ($Beta$ = -.304, p = .153) nor Hints ($Beta$ = 0.30, p = .33) were significant predictors of MCAS total score.

4. Discussion

This study examined the use of a new measure for brief cognitive assessment, the BCA, in a FEP population. As only one published study to date (Velligan et al., 2004) has used this measure in a chronic schizophrenia population, the present study was the first to assess its use in an FEP population. In contrast to Velligan et al.'s (2004) study, this study incorporated a number of additional measures, which examined Theory of

Mind, current IQ and symptomatology. The relationship of these variables to BCA

score and community functioning (MCAS) was explored. Theory of mind has received

very little attention in the FEP literature, and this study is among the first to explore this

construct using a variety of ToM measures.

4.1 Discussion of the results in relation to the hypotheses

The first hypothesis that the FEP participants would be significantly impaired

relative to normative data was only partially supported by the findings. Although the

FEP patients were impaired relative to normative data on all subtests of the BCA, the

only statistically significant difference was on Trails A. In Velligan et al.'s (2004) study

their participants were significantly impaired on all subtests of the BCA except Trails B

($z = 1.62$, $SD = 1.13$). The difference between our findings may be explained by the

more chronic presentation of Velligan et al.'s (2004) participants whose cognitive

functioning appeared to be more compromised than that of the participants in the present

study. However, there are also similarities between ours and Velligan et al.'s (2004)

findings that are note worthy. Firstly, in both studies the participants were less impaired

on Trails B than A and showed a similar level of impairment on each of these tasks

despite the larger sample size used in Velligan et al.'s (2004) study (n=340) and their

more chronic presentation. This finding was counterintuitive as Trails B appears to be a

more demanding task measuring psychomotor speed and the ability to inhibit

inappropriate responses, while Trails A only assesses psychomotor speed. This result

may reflect a practice effect, as Trails A was administered before Trails B, which may

have given the participants an advantage on the psychomotor aspect of Trails B thus

allowing them to concentrate better on the response inhibition part of the task. A second

similarity between our findings relates to Verbal Fluency tasks where both groups of

participants were more impaired on the Categories than the Letters subtests. A

differential impairment on Verbal Fluency Categories has been reported in other studies

of schizophrenia and may reflect a compromised semantic system in those with

schizophrenia (Bokat & Goldberg, 2002; Elvevåg, Weinstock, Akil, Kleinman, &

Goldberg, 2001).

Consistent with the second hypothesis, the Global Cognition Function Score

(GCFS) on the BCA correlated positively with community functioning as measured by

the MCAS. This is supported by Velligan et al.'s (2004) finding of an association

between the BCA and the MCAS. These results suggest that the BCA may be a useful

predictive measure of community functioning.

The third hypothesis was only partially supported by the findings. Our results

showed a lack of association between the positive symptoms (as measured by the BPRS)

of psychosis and community functioning (as measured by MCAS). This is consistent

with other studies, which showed weak evidence for an association between positive

symptoms and community function (Green 1996; 2000). This suggested that in the

present sample, GCFS was a better predictor of community functioning than the positive

symptoms of psychosis, which again is consistent with other studies (e.g. Green, 1996;

Green et al., 2000; Velligan, Bow-Thomas, Mahurin, Miller, Halgunseth, 2000). There

was a significant association between the negative symptoms of psychosis (as measured

by the BPRS) and MCAS total. There are mixed findings in the literature regarding an

association between negative symptoms and community functioning, and in Green's

review (1996) negative symptoms were found to be inconsistently related to community

outcome. In the regression analysis, neither the negative symptoms of psychosis nor

GCFS were independent predictors of community function. The sample size used in the present study may not have been large enough to detect a significant effect where there was one (Type II error). Future studies would benefit from exploring this link further using larger sample sizes than the one used in the current study.

The fourth hypothesis that BCA would be more closely related to level of community functioning than IQ was not supported, as the correlation between IQ and MCAS was greater than between BCA (GCFS) and MCAS. This suggested that IQ in addition to the BCA (GCFS) may be a useful predicator of social and community functioning. Neither IQ nor GCFS were significant independent predictors of community functioning in the regression analysis. Again, the small sample size used may not have generated enough statistical power to detect a difference where there was one.

The fifth hypothesis was not supported by the findings. Although the Hints Task was positively and significantly related to MCAS total score, it was not a significant independent predictor of community function in the regression analysis. This result may have been due to the small sample size used. The relationship between ToM and community functioning has been confirmed in other studies, and ToM has been shown to be more related to community function than neurocognition (Brüne, 2005; Pinkham & Penn, 2006; Pollice et al., 2002). In the present study, IQ was more correlated with community function than the BCA (GCFS). Future studies would benefit from exploring the predictive power of ToM and community functioning using a larger sample size.

In addition to the above findings, GCFS was not associated with medication or duration of illness. These were important findings because they suggested that

medication and duration of illness were not acting as confounding factors in the present study, as they were not significantly associated with cognitive impairment. This finding should be interpreted with caution due to the small sample size (n=21), which may have resulted in insufficient statistical power to detect a relationship between medication and cognitive impairment when in fact there was one (Type II error). Nevertheless, these results reflect other findings in the literature regarding a lack of association between cognitive function and duration of illness (Prikryl et al., 2003).

4.2 Strengths and limitations of the study

The strengths and limitations of this study must be considered when interpreting the results. In terms of strengths, this study was the first to administer the BCA in an FEP population. Despite the small sample size, the BCA showed modest internal consistency, further supporting its validity and use as a brief assessment of cognition in schizophrenia. Consistent with Velligan et al.'s (2004) study, the measures that comprise the BCA took approximately 15 minutes to administer, which adds further weight to its usefulness in a clinical setting as it gives an overview of the main cognitive domains known to be impaired in psychosis, in a brief space of time.

In contrast to Velligan et al.'s (2004) study, the current study incorporated a number of additional measures into its design including the WASI, ToM measures and the BPRS. These additional measures were used to explore the independent contributions of IQ, ToM and symptoms to participants' levels of community and cognitive function. Psychosis is a complex area, which was reflected in the design and analysis of this study. Although this study reflected these complexities, it remained hypothesis driven so as not to become overwhelmed by them.

There are also some limitations to this study that are worth noting. Firstly, the sample size was small and had fewer participants than the power calculation recommended. The difficulty recruiting participants was due to factors related to FEP generally (e.g. poor engagement, relapse and hospitalization) and issues within the services I recruited from (high turnover of staff, little time for staff to help with recruitment). The small sample size increased the risk of Type II error, that is, lack of power to detect an effect when in reality one exists. Unfortunately, difficulty recruiting participants reflects the inherent obstacles to research in this area that future researchers could try to address by approaching more services thus gaining a larger pool of potential participants to draw from.

Secondly, although all of the participants were fluent in English, half of the sample did not speak English as a first language, which may have impacted on their understanding of and scores on the tests. This corresponds to the cultural diversity of the participants, who were recruited from an area of London that has a high concentration of minority ethnic groups. The high number of participants from minority ethnic groups may also reflect the reported increased incidence of psychotic illness among these groups (Fearon et al., 2006). As is often the case in neuropsychological testing, the norms used for comparison with the BCA measures were based on English speaking populations who were predominantly Caucasian. These cultural and linguistic differences between the participants and norms may have led to inaccurate comparisons between the two groups, which may have confounded the results. There is an urgent need for norms that reflect cultural diversity from which to make comparisons with those with psychotic illness.

The MCAS was completed by the participants' care-coordinator. While the care-coordinators generally provided an objective and well informed perspective on the participants' abilities, it may have been beneficial to incorporate information from other sources. Brüne (2005) noted that nursing staff generally underrated patients when they completed a measure of social functioning (Social Behaviour Scale; Wykes & Sturt, 1986) perhaps because they were used to 'oddities' or 'habits' of the patients they were rating. In consideration of this, future studies might benefit from incorporating information from the clients themselves, clinical reports and the Responsible Medical Officer when rating the MCAS. Also, because many different care-coordinators completed the measure across two different services, inter-rater reliability was an important issue to ensure that the different care coordinators were completing the MCAS based on the same criteria. In consideration of this issue the researcher was present each time the questionnaire was completed to answer any questions about the MCAS items. Brüne (2005) recommended that staff should be trained in the use of measures such as the MCAS, which future studies might consider.

Unfortunately, none of the participants consented to being audio taped for dual rating on the BPRS. This may have been due to symptoms of psychosis such as paranoia or a lack of motivation to be taped, as it did not affect their participation in the study or the financial incentive they received. Due to time constraints, my supervisor was unable to attend the assessments to observe and rate the BPRS. Perhaps if a larger number of participants had been recruited there would have been a greater chance of some of them consenting to be audio taped.

4.3 Recommendations for future studies

In addition to the recommendations outlined above, future studies might consider which aspects of ToM relate to community functioning, thus moving from exploratory studies to hypothesis driven ones. Pinkham and Penn (2006) looked at social cognition by breaking this construct down into three domains: emotion perception, social knowledge and ToM. They found that social cognition contributed significantly more variance to interpersonal skill than neurocognition and concluded that social cognition may not be best characterised by one specific deficit but by the impairments that span the range of social cognitive skills. Community functioning might also be broken down into its constituent parts. This might be achieved through incorporating functional assessments of living skills (e.g. Functional Needs Assessment; Domborwski, Kane, Tuttle, & Kinceid, 1990) into assessment batteries so that direct assessment of these skills could be observed in addition to collecting information via questionnaires. Velligan et al. (2004) used the Functional Needs Assessment as well as the MCAS to assess community functioning, and both measures were correlated with the BCA.

It would also be interesting for future studies to gather information from the participant regarding their self perception of cognitive deficits to see if this bears any relationship to the measures used to assess these domains. As part of the study, I fed back individual participants' results upon their request, and several participants offered further information about these deficits shedding light on how they manifested in day to day function and were experienced by the participant. This information was then used in targeted interventions. Stip, Caron, Renaud, Pampoulova and Lecomte (2003) used the Subjective Scale to Investigate Cognition in Schizophrenia (SSTICS) to assess memory (working, explicit long-term memory), attention (divided, distractibility, alertness, sustained), language, and praxia in those with schizophrenia. The SSTICS was shown to

have good internal consistency and stability over time and was correlated with objective cognitive assessments. Future studies might benefit from incorporating this measure in addition to objective neuropsychological tests.

4.4 Implications of the findings

Our results showed that patients with FEP are impaired relative to normative data on the cognitive domains of memory, attention and executive function, though only performance on Trails A was significantly different from norms. The larger sample size and more chronic population examined in Velligan et al.'s (2004) study make direct comparisons with this study difficult. However, the participants in Velligan et al.'s (2004) study were significantly more impaired on the BCA, which suggests that cognition may become more impaired as psychotic illness progresses. This impairment is something that needs to be addressed, and its implications can no longer be overlooked. Interventions aimed at reducing the impact of the cognitive deficits may reduce the long term social consequences of psychosis for individuals.

Cognitive rehabilitation has been increasingly cited as an essential component of comprehensive treatment (Kern & Green, 1998; Storzbach & Corrigan, 1996). Cognitive rehabilitation approaches seek to enhance cognitive processes or to circumvent cognitive impairments in schizophrenia in an effort to improve functional outcome (Velligan, Kern, & Gold, 2006). This is potentially very important with first episode populations where cognitive rehabilitation could have a significant impact on the person's quality of life and community functioning. The BCA may be used to assess neurocognitive abilities and to place patients on different rehabilitation tracks based on the results of their assessment. For example, if a patient has deficits in verbal memory,

more repetition of verbal instructions may be needed, which could be incorporated into their rehabilitation program (Velligan et al., 2006).

The BCA may also be used by mental health professionals to assess changes in cognitive function over the course of treatment. The BCA was straightforward to administer and score and would be relatively simple to train a wide range of mental health professionals to use. It was also brief, which prevented fatigue among the participants, and they appeared to enjoy the tasks and seemed motivated to perform their best. These are important aspects of any cognitive assessment that is to be widely and routinely used in this population, both in terms of persuading staff to administer it and encouraging patients to complete it.

4.5 Conclusion

In summary, the purpose of this study was to examine the relationship between cognitive and community functioning in FEP. The BCA (GCFS) score and ToM Hints task were related to community functioning. Although neither BCA nor ToM were significant independent predictors of community function in a regression analysis, future studies should explore this relationship further using larger sample sizes. This study has provided additional evidence of subtle cognitive deficits in FEP (Sweeney, Haas, Kelip, & Long, 1991; Hutton et al., 1998; Riley, et al., 2000) and highlights the need for a standardised way of assessing these. The BCA is a simple, brief and reliable assessment of the domains consistently reported to be impaired in psychosis and may have a future role in psychosis services to provide a baseline measure of cognitive function and to monitor change over time. This is particularly pertinent in FEP where quick assessment and intervention may have a significant impact on future course of illness.

References

Abu-Akel, A. (2003). A neurobiological mapping of theory of mind. *Brain Research Reviews, 43,* 29-40.

American Psychiatric Association (1994). *Diagnostic and Statistical Manual of Mental Disorder (4th ed.).* American Psychiatric Press. Washington, DC.

Barker, S., Barron, N., McFarland, B. H. & Bigelow, D. A. (1994). A community ability scale for chronically mentally ill consumers: Part I. Reliability and validity. *Community Mental Health Journal, 30,* 363-383.

Benton, A. L., & Hamsher, K. (1978). *Multilingual Aphasia Examination (manual, revised).* Iowa City, IA: University of Iowa.

Bokat, C. E., & Goldberg, T. E. (2003). Letter and category fluency in schizophrenic patients: a meta-analysis. *Schizophrenia Research, 64,* 73-78.

Brandt, J. (1991). The Hopkins Verbal Learning Test: development of a new verbal memory test with six equivalent forms. *Clinical Laboratory Neuropsychology, 5,* 125-142.

Brandt, J., & Benedict, R. H. B. (2001). *Hopkins Verbal Learning Test – Revised. Professional Manual.* Florida: Psychological Assessment Resources.

Brüne, M. (2005). Emotion recognition, 'theory of mind' and social behaviour in schizophrenia. *Psychiatry Research, 113,* 135-147.

Community Mental Health Evaluation Initiative (CMHEI) Working Paper. (2001). The Interrater Reliability of the BPRS and the MCAS: A Multi-Site Experience Using the "Train the Trainer" Approach. Retrieved 18th April, 2007, from http://www.ontario.cmha.ca/cmhei/docs/cmhei_working_paper_02-01.pdf.

Corcoran, R., Mercer, G., & Frith, C. D. (1995). Schizophrenia, symptomatology and

social inference: investigating theory of mind in people with schizophrenia.

Schizophrenia Research, 17, 5-13.

Dombrowski, S. B., Kane, M., Tuttle, N. B., Kincaid, W. (1990). *Functional needs*

assessment program for chronic psychiatric patients. Communication Skill

Builders. Tucson, A.Z.

Elvevåg, B., Weinstock, D. M., Akil, M., Kleinman, J. E., & Goldberg, T. E. (2001). A

comparison of verbal fluency tasks in schizophrenic patients and normal controls

Schizophrenia Research, 51, 119-126.

Fearon, P., Kirkbride, J. B.,Morgan, C., Dazzan, P., Morgan, K., Lloyd, T., Hutchison,

G., Tarrant, J., Fung, W. L. A., Holloway, J., Mallett, R., Harrison, G., Leff, J.,

Jones, P. B., & Murray, R. M. (2006). Incidence of schizophrenia and other

psychoses in ethnic minority groups: results from the MRC AESOP Study.

Psychological Medicine, 36, 1541-50

Fleiss, J. (1981). *Statistical Methods for Rates and Proportions (2nd ed).* New York,

Wiley.

Frith, C. D., & Corcoran, R. (1996). Exploring 'theory of mind' in people with

schizophrenia. *Psychological Medicine, 26,* 521-530.

Fromm-Auch, D., & Yeudall, L. T. (1983). Normative data for the Halstead-Reitan

neuropsychological tests. *Journal of Clinical Neuropsychology,* 5, 221-238.

Fujii, D. E., & Wylie, A. M. (2003). Neurocognition and community outcome in

schizophrenia: long-term predictive validity. *Schizophrenia Research, 59,* 219-

223.

Gold, J. M., & Harvey, P. D., (1993). Cognitive deficits in schizophrenia. *Psychiatric Clinics of North America 16,* 2, 295-312.

Green, M. F. (1996). What are the functional consequences of neuro-cognitive deficits in schizophrenia? *American Journal of Psychiatry, 153,* 321-330.

Green, M. F., Kern, B. S., Braff, D. L., Mintz, J. (2000). Neurocognitive deficits and functional outcome in schizophrenia: are we measuring the "right stuff"? *Schizophrenia Bulletin, 26,* 119-136.

Green, M. F., Nuechterlein, K. H., Gold, J. M., Barch, D. M., Cohen, J., Essock, S., Fenton, W. S., Frese, F., Goldberg, T. E., Heaton, R. K., Keefe, R. S. E., Kern, R. S., Kraemer, H., Stover, E., Weinberger, D. R., Zalcman, S., & Marder, S. R. (2004). Approaching a Consensus Cognitive Battery for Clinical Trials in Schizophrenia: The NIMH-MATRICS Conference to select cognitive domains and test criteria. *Biological Psychiatry 56,* 301-307.

Harrington, L., Siegert, R. J., & McClure, J. (2005). Theory of mind in schizophrenia: A critical review. *Cognitive Neuropsychiatry 10,* 249-286.

Howell, D. C. (1992). *Statistical Methods for Psychology, 3rd edition.* California: Duxberry Press.

Hutton, S. B., Puri, L. J, Duncan, T. W., Robbins, T. W., Barnes, T. R. E., Joyce, E. M. (1998). Executive function in first-episode schizophrenia. *Psychological Medicine, 28,* 463-473.

Inoue, Y., Yamada, K., Hirano, M., Shinohara, M., Tamaoki, T., Iguchi, H., Tonooka, Y., Kanba, S. (2006). Impairment of theory of mind in patients in remission

following first episode of schizophrenia. *European Archives of Psychiatry and Clinical Neuroscience, 256,* 326-8.

Jaeger, J., & Douglas, E. (1992). Neuropsychiatric rehabilitation for persistent mental illness. *Psychiatric Quarterly, 63,* 71-94.

Kennedy, K. J. (1981). Age effects on the Trail Making Test performance. *Perceptual and Motor Skills, 52,* 671-675.

Kern, R. S., & Green, M. F. (1998). Cognitive Remediation in Schizophrenia. In *Handbook of Social Functioning in Schizophrenia* (ed. K. T. Mueser and N. Tarrier), pp. 342-354. Allyn & Bacon: Boston.

Langdon, R., Michie, P., Ward, P., McConaghy, N., Catts, S., & Coltheart, M. (1997). Defective self and/or other mentalising in schizophrenia: a cognitive neuropsychological approach. *Cognitive Neuropsychiatry, 2,* 167-193.

McGorry, P. D., & Edwards, J. (1997). *Early psychosis training pack.* Macclesfield, UK: Gardiner-Caldwell Communications Ltd.

Milev, P., Ho, B. C., Arndt, S., & Andreasen, N. C. (2005). Predictive Values of neurocognition and negative symptoms on functional outcome in schizophrenia: a longitudinal first-episode study with 7-year follow-up. *American Journal of Psychiatry, 162,* 495-506.

Nelson, E. (1982). *National Adult Reading Test (NART): Test Manual,* NFER Nelson, Windsor, UK.

Pickup, G. J., & Frith, C. D. (2001). Theory of mind impairments in schizophrenia: symptomatology, severity and specificity. *Psychological Medicine, 31,* 207-220.

Pinkham, A. E., & Penn, D. L. (2006). Neurocognitive and social cognitive predictors

of interpersonal skill in schizophrenia. *Psychiatry Research, 143,* 167-78.

Pollice, R., Roncone, R., Falloon, I., Mazza, M., De Risio, A., Necozione, S., Morosini,

P., & Casacchia, M. (2002). Is theory of mind in schizophrenia more strongly

associated with clinical and social functioning than neurocognitive deficits?

Psychopathology, 35, 280-288.

Premack, D., & Woodruff, G. (1978). Does the chimpanzee have a 'theory of mind'?

Behavioural and Brain Sciences, 4, 515-526.

Prikryl, R., Kasparek, T., Ceskova, E., Kucerova, H., Ondrusova, M., Drybcak, P., &

Lorenc, M. (2003). The cognitive dysfunction in the first episode of

schizophrenia. *Schizophrenia Research, 60,* 178-179.

Psychological Corporation. (1999). *Wechsler Abbreviated Scale of Intelligence (WASI)*

manual. San Antonia, TX: Author.

Reitan, R. M., & Wolfson, D. (1985). The Halstead-Reitan Neuropsychological Test

Battery. Neuropsychology Press, Tucson, AZ.

Riley, E. M., McGovern, D., Mockler, D., Doku, V. C. K., O Ceallaigh, S., Fannon, D.

G., Tennakoon, L., Santamaria, M., Soni, W., Morris, R.G., & Sharma, T.

(2000). Neuropsychological functioning in first-episode psychosis - evidence for

specific deficits. *Schizophrenia Research, 43,* 47-55.

Sarfati, Y., & Hardy- Baylé, M. C. (1999). How do people with schizophrenia explain

the behaviour of others? A study of theory of mind and its relationship to thought

and speech disorganisation in schizophrenia. *Psychological Medicine, 29,* 613-620.

Selnes, O. A., Jacobson, L., Machado, A. M., Becker, J. T., Wesch, J., Miller, E. N., Visscher, B., & McArthur, J. C. (1991). Normative data for a brief neuropsychological screening battery. *Perceptual and Motor Skills, 73,* 539-550.

Stip, E., Caron, J., Renaud, S., Pampoulova, T., & Lecomte, Y. (2003). Exploring cognitive complaints in schizophrenia: SSTICS—Subjective Scale to Investigate Cognition in Schizophrenia. *Comprehensive. Psychiatry, 44,* 331–340.

Storzbach, D. M., & Corrigan, P. W. (1996). Cognitive Rehabilitation for Schizophrenia. In *Cognitive Rehabilitation for Neuropsychiatric Disorders* (ed. P. W. Corrigan and S. C. Yudofsky), pp. 299-328. American Psychiatric Press: Washington, DC.

Sweeney, J. A., Haas, G. L., Kelip, J. G., & Long, M. (1991). Evaluation of the stability of neuropsychological functioning after acute episodes of schizophrenia: a one year follow-up study. *Psychiatric Research, 38,* 63-76.

Tabachnick, B. G., & Fidell, L. S. (2001). *Using Multivariate statistics, 4th edition.* Boston: Allyn & Bacon.

Townsend, L. A., Malla, A. K., & Norman, R. M. G. (2001). Cognitive functioning in stabilized first-episode psychosis patients. *Psychiatry Research, 104,* 119-131.

Townsend, L. A., Norman, R. M. G., Malla, A. K., Rychlo, A. D., Ahmed, R. R. (2002). Changes in cognitive functioning following comprehensive treatment for first episode patients with schizophrenia spectrum disorders. *Psychiatry Research, 113,* 69-81.

Velligan, D. I., Bow-Thomas, C. C., Mahurin, R. K., Miller, A. L., & Halgunseth, L. C. (2000). Do specific neurocognitive deficits predict specific domains of community function in schizophrenia? *The Journal of nervous and mental disease, 188,* 518-24.

Velligan, D. I., DiCocco, M., Bow-Thomas, C. C., Cadle, C., Glahn, D. C., Miller, A. L., Biggs, M. M., Shores-Wilson, K., McKenzie, C. A., & Crismon, M.L. (2004). A brief cognitive assessment for use with schizophrenia patients in community clinics. *Schizophrenia Research, 71,* 273-283.

Velligan, D. I., Kern, R. S., Gold, J. M. (2006). Cognitive Rehabilitation for Schizophrenia and the Putative Role of Motivation and Expectancies. Schizophrenia Bulletin 2006, 32, 474-485.

Velligan, D., Prihoda, T., Dennehy, E., Biggs, M., Shores-Wilson, K., Crismon, M. L., Rush, A. J., Miller, A., Suppes, T., Trivedi, M., Kashner, T. M., Witte, B., Toprac, M., Carmody, T., Chiles, J., & Shon, S. (2005). Brief Psychiatric Rating Scale Expanded Version: How do new items affect factor structure? *Psychiatry Research, 135,* 217-228.

Ventura, M. A., Green, M. F., Shaner, A., & Liberman, R. P. (1993). Training and quality assurance with the brief psychiatric rating scale: "The drift buster". *International Journal of Methods in Psychiatric Research, 3,* 221-244.

Wechsler, D. (1997). *Wechsler Adult Intelligence Scale, 3rd edition.* San Antonio, Texas: Psychological Corporation.

Wykes, T., & Sturt, E. (1986). The measurement of social behaviour in psychiatric

patients: an assessment of the reliability and validity of the SBS schedule, *British*

Journal of Psychiatry, 148, 1-11.

Yeudall, L. T., Fromm, D., Reddon, J. R., & Stefanyk, W. O. (1986). Normative data

stratified by age and sex for 12 neuropsychological tests. *Journal of Clinical*

Psychology, 42, 918–946.

Part 3: Critical Appraisal

The following part of my thesis will reflect on the process of conducting this research. I will focus in particular on the experience of working in the early intervention service (EIS), recruitment, the writing of reports regarding the participants' results, and the selection of assessment measures.

1. Experience of working with the EIS teams

Participants were recruited from two EISs in the hope that enough would be recruited. One other EIS was approached but declined to take part in the research project. Although both teams were early intervention in psychosis teams, they were very different in terms of their ethos. One team was led by a clinical psychologist and had more psychologists working in it than the other team, which was led by a psychiatrist and mainly composed of nurses and social workers. Because of their training and experience, the team with greater psychology input was more familiar with the process of research and had a greater understanding of the project. The other team seemed a little suspicious of the project initially, and they needed reassurance that the research would not harm their clients and was not an evaluation of their work or the service. Both services were very busy, and at times I felt as though my research was at the bottom of their agenda. Although this was disappointing, it was inevitable given the work pressures on the staff in the EISs.

2. Recruitment

One weakness of this study was the numbers of participants recruited, which were 16 less than the power calculation recommended. There were a number of factors that impinged on recruitment, which were as follows:

I collected data from two EISs in order to maximise recruitment. There were three other research projects taking place between the two teams who consented to take part, which impacted on my ability to recruit participants. Although these projects focused on different topics, the care coordinators (CCs) were reluctant to approach clients who had already taken part in another study, fearing it might hamper engagement with the EIS. Also, despite the financial incentives offered, many of the clients were reluctant to take part in multiple research projects over a short period of time. The number of concurrent research projects also led to confusion from the CCs about the focus of different projects, and anxiety about the increased demands on their time in terms of recruitment. The difficulties with recruitment and the high number of concurrent research projects taking place in the EISs may reflect the increasing research interest in EISs and first episode psychosis (FEP). It may also reflect the increasing numbers of trainee clinical psychologists working in the National Health Service (NHS) who are required to conduct research as part of their training.

The recruitment protocol was decided upon after meetings with the service managers and Responsible Medical Officers (RMO) from the two services who took part. Initially the recruitment protocol was; 1) the researcher (AW) met with each CC to identify suitable participants from their caseload; 2) the RMO approved/disapproved that the client(s) identified from the CCs' caseloads were suitable to take part; 3) the CC gave an approved client an information leaflet about the study, and asked their consent for the researcher to contact them; 4) if the client gave consent, the researcher contacted

them to answer any questions they had about the study and arranged a time to meet if they were interested in taking part.

There were a number of problems with stage one of this protocol that impacted on recruitment. When the CCs were emailed or asked in person to arrange a time to meet about the research, some CCs did not reply to the emails or said they were too busy to meet at that time. This often delayed recruitment and was perhaps indicative of the priority of the research project in their busy schedules.

Of the CCs who agreed to meet, many of them deemed lots of the clients on their caseload unsuitable to take part despite the inclusion criteria being quite broad (Appendix 1). Each CC had a caseload of approximately 15 clients, and the highest number of clients identified from the CCs' caseloads was approximately 1-3. It was difficult to identify why so many participants were thought unsuitable. Often the CCs gave reasons such as "they won't engage with me so they probably won't engage in the study", even though I was offering a financial incentive to take part. This presented a dilemma for me between challenging the reasons why most clients were deemed unsuitable to take part or remaining quiet in an effort to keep the CCs on board with the study. After discussion with my supervisor, I chose not to challenge the CCs about why so many of their clients were unsuitable to take part, as I was new to the team and did not want to antagonise its members. In hindsight, perhaps it would have benefited recruitment to be more challenging.

In stage two of the recruitment protocol the RMO generally approved most clients identified as suitable to take part. However, this often delayed stage three of the protocol, as it often took the RMO a long time to sanction whether or not the clients were suitable. The RMO's involvement at this stage in the protocol also perhaps

undermined the CCs' decisions about who was suitable to take part. However, this was an unavoidable dynamic in the service, which was quite hierarchical.

Regarding stage three of the recruitment protocol, it took varying lengths of time for the CCs to approach the clients identified about the study. This was often due to forgetting to give an information leaflet to the participant when they met, engagement problems on the clients' part and concerns from the team about the clients' mental state. The CCs were very busy, and it often seemed as though giving information leaflets to the clients they identified placed an extra burden on them. Some CCs expressed anxiety about the level of knowledge they were expected to have about the study. To minimise their anxiety and to limit the information given by CCs to clients, the CCs were instructed to say, "There's a research project running in the service and we thought you might be interested in taking part". The CCs were then asked to give the client the information leaflet and not to answer any questions about the study. If the client had some queries, they were instructed to direct the client to the information leaflet or offer that the researcher would contact them to answer their questions. Despite these instructions, some CCs relayed misinformation about the study to the clients. For example, when I contacted one client she reported that she did not want to take part because she did not want to talk about the events that precipitated her onset of psychosis. When I informed her that no part of the study required that information she seemed surprised and said that her CC had told her she would probably have to talk about these experiences. Also, as there were three other research projects being conducted between the two EISs, the CCs sometimes understandably confused the various projects.

In the final stage of the recruitment protocol, few clients declined to take part when I contacted them to arrange a time to meet. However, some frequently cancelled

their appointments or did not attend. Those who did not want to be contacted about the study often did not give reasons for this.

In the first few months of the project, there was a very low referral rate to the study. As a consequence, I met with the RMO to discuss ways to increase recruitment, while reducing dependence on the CCs. The RMO suggested that he would identify suitable clients to take part and that I should then ask the CCs if it would be alright to send the clients identified from their caseload an information leaflet about the study. Although the RMO identified a high number of clients to approach, gaining the CCs consent to contact their clients was still a barrier. Often their reasons were unclear, but many were concerned that the client was not properly engaged in the service and that asking them to take part in the study might further hamper their engagement. Nevertheless, this new protocol increased the numbers recruited and reduced the onus on the CCs to identify suitable clients and give them information leaflets. After sending information leaflets about the study, some clients were difficult to contact due to lack of a telephone service, and some involvement from the CCs was needed to ask if they were interested in taking part.

Several extraneous factors within the teams also impinged on recruitment. In one team, four CCs resigned during the recruitment stage. This meant many clients had to be followed up by new staff, which resulted in delays and often subsequent difficulties with engagement. Also, since the new staff knew little about their clients it was difficult for them to identify suitable candidates for the research. Two CCs had significant life events that resulted in extended absences, which also delayed recruitment and impacted on client engagement. Both EISs moved location whilst I was recruiting, which also

impacted on client engagement, room bookings and the priority of my research in staff members' minds.

In addition, one of my field project supervisors left the EIS before I began recruitment, forcing me to seek consent from the new team leader before I could begin. Although the team leader was open to the idea of my research, he suggested many new ideas, which would have changed the nature and design of the research. Because the project had already received ethical approval, I was limited in the changes I could make and was only able to incorporate a few of his ideas. However, this helped to get him on board with the project.

If I were to do a similar study again, I would initially ask the RMO for list of potentially suitable clients from the services' overall caseload rather than asking the CCs to identify clients from their individual caseloads. In the present study, the latter model of recruitment was decided upon by the RMOs and service managers at the beginning of the study, but was revised when it did not yield high numbers of clients to approach. This method of negotiation and revision was a strength of the way I approached and conducted the study (i.e. not acting as an 'expert' and commanding what to do), which may have helped recruitment as it promoted good relationships between myself and the staff.

3. Reports

Once clients took part in the study, I fed back to their CC about whether they had attended our meeting and whether they had given consent for a report to be provided to the team regarding their results (see Appendix 3 for a report template). If the client consented to a report, I completed this and met with the CC within two weeks of the

client's participation in the study to deliver it. I also used this opportunity to ask the CC

to complete the Multnomah Community ability Scale (MCAS; Barker, Barron,

McFarland, & Bigelow, 1994). It was important to meet with the CC within this one

month period because the MCAS ratings are based on the client's presentation over the

previous month. All of the CCs agreed to complete this questionnaire, perhaps because

they were getting something back from the study in the form of a report.

Only one client did not consent to having a report of his results included in his

file. Of the remaining 23 participants who consented, eight participants requested

feedback regarding their results. This feedback was given in person or over the phone if

the participant did not want to arrange a meeting. This was a difficult part of the

research process for me as it often involved communicating sometimes poor results to

the clients, many of whom had little insight regarding their difficulties. However, some

clients reported that the results fitted with their experience, for example, of having

memory problems or difficulty placing themselves in another person's shoes (Theory of

Mind). In some cases, the results provided an objective measure of what the client

already knew but had not articulated.

Although time-consuming to write, the reports proved to be an important

component of the clients' care and were valued by the EIS staff. Some staff felt

overwhelmed by the clients on their caseloads, and the reports often provided some key

information that changed the way the staff related to the clients and their understanding

of clients' difficulties. For example, one client was very socially adept but displayed

immature behaviour, which frustrated the staff especially when on group outings. Based

on his results from the assessment, his score on the Wechsler Abbreviated Scale of

Intelligence (WASI) (Psychological Corporation, 1999) was within the learning

disability range. The client's CC was initially shocked by this, perhaps because his

social skills may have masked this deficit in IQ. This finding led to greater

understanding about why he acted immaturely and did not obey staff directions when out

in the community. This particular client asked for feedback regarding his results. I was

initially concerned about giving him feedback because he appeared to have high self-

esteem and he was regarded by his family as "the clever one". However, he

acknowledged that he often had difficulty understanding things that others appeared to

have no difficulty understanding, but was afraid to say so. He appeared somewhat

relieved that there was an explanation for this. His CC discussed this further in meetings

with him and thought of ways he could ask for extra support when he did not understand

things.

4. Reflection on the choice of assessment measures used

We chose the assessment measures to reflect the domains thought to be impaired

in FEP, namely executive function, verbal memory, attention and psychomotor speed.

Theory of mind and IQ measures were also incorporated into the research design, as

these areas have not received much attention in the FEP literature. In the next section,

the process of using each of the measures is discussed.

4.1 Brief Cognitive Assessment (BCA) (Velligan et al., 2004)

The Trail Making Test (TMT) (Reitan & Wolfson, 1985) was the first test

administered, and many participants took longer than expected to complete the Trails A

subtest perhaps because they were nervous or settling in to the assessment. In general, it

seemed as though participants found the TMT easy, which may have reduced their

anxiety about the rest of the assessment. Two participants reported that seeing the numbers printed on the page reminded them of school and studying mathematics, which they believed they were not good at. This suggested that the participants' experiences at school may have influenced their performance during the assessment and may have deterred some clients from taking part. The potential influence of prior academic experiences on participants' willingness and performance during neuropsychological assessment should be explored in future research.

Although all participants were fluent in English, 50% of the sample of participants did not speak English as a first language. This may have influenced their performance on the Verbal Fluency task (Benton & Hamsher, 1978). After completing this task several participants reported that they found it difficult to think of the words they were trying to say in English.

Some participants who had active symptoms of psychosis recalled words from the Hopkins Verbal Learning Test (HVLT) (Brandt, 1991) that were not in the trials or related to any of the words semantically or phonetically. One participant recalled the same set of words across the trials even though the words he was recalling were not in the trials. These intrusions may have reflected thought disorder. Information on intrusions is not usually recorded during administration of the HVLT or reported in published studies. This information may provide a useful indicator of thought disorder in clients with psychosis.

4.2 Theory of Mind tasks

The Hints task (Corcoran, Mercer, & Frith, 1995) was quite Westernised in its phraseology, and several participants asked me to translate some words and phrases such

as "Ajax", "flat broke" and "right up my street". The names of the characters in the

Hints Task and Theory of Mind Stories (Frith & Corcoran, 1996) were also quite

westernised, and many of the participants who were not from the UK may not have

related to the stories as well as those who were from the UK. Future researchers

planning on using these tasks should consider these issues. Most participants seemed to

enjoy the Picture Sequencing Task (Langdon et al., 1997). The non-verbal aspect of the

task provided a good measure of theory of mind for those participants who did not speak

English as a first language.

4.3 Other tasks

The shorter version of the WASI was used to give an estimate of full scale IQ

only. This incorporated a performance task (Matrix Reasoning) and a verbal task

(Vocabulary). As with the other verbal tasks, the Vocabulary subtest appeared more

difficult for those who did not have English as a first language. In neuropsychological

assessment, there is a lack of norms for ethnic minorities and those who do not speak

English as a first language. This area requires further research in the future so that these

participants are compared to culturally appropriate norms.

Cultural factors also played a role in rating the Brief Psychiatric Rating Scale

(BPRS) (Ventura, Green, Shaner, & Lieberman, 1993), particularly the 'grandiosity'

item. When interviewed regarding this item, many participants reported that they

believed they had special powers and abilities and a special purpose in life. A high

proportion of the sample was Muslim, and some reported that these beliefs were related

to their religion. It was often difficult to explore this further with those participants,

perhaps due to the private nature of those beliefs and because they had reservations addressing these matters with a Western Caucasian female.

The General Assessment of Functioning scale (GAF) (American Psychiatric Association, 1994) provided a very brief and easy measure of functioning based on the participants' presentation during the assessment. This measure was correlated with the global cognitive function score (GCFS) from the BCA. On reflection, it would have been beneficial to obtain a GAF rating from the CCs and compare it to the score taken on the day of the assessment.

The Multnomah Community Ability Scale (MCAS) (Barker, Barron, McFarland, & Bigelow, 1994) provided a good estimate of community function, which was correlated with GCFS and the Hints task. The high turnover of staff in the EIS meant that some CCs who were new to the services were less familiar with their clients than other CCs. This may have meant that some staff were not in a position to give as full a picture of their clients as other staff. Unfortunately, this limitation was unavoidable but may have been compensated for if collateral information regarding the MCAS items had been obtained from other sources of information (e.g. recent reports, RMO).

Conclusion

In summary, there were a number of factors that impacted on my ability to recruit enough participants for this research study. The CCs were crucial in facilitating the recruitment of participants. Unfortunately, research in the NHS often seems to be a low priority in comparison with seeing clients and dealing with day to day administrative tasks. The pressures that staff are under to meet NHS targets often mean that there is little time or energy to engage in research related activities. These factors

impacted on this research project in terms of the numbers recruited. Several extraneous factors including staff turnover and CCs' life events also impacted on recruitment. These factors were unpredictable but reflect the reality of conducting research in the NHS.

Writing the reports consumed a great deal of my time and often detracted from recruitment. However, they also bolstered recruitment by giving something meaningful back to the service thus engaging the CCs in the research project. Providing reports on the clients' results enabled me to return something of value from my research to the services and their clients, which improved my relations with them and therefore eased the process of recruitment somewhat. The reports often played an important role in the clients' care plans and the CCs' view of their difficulties. This illustrates how, if the BCA were to be used routinely by psychologists in teams, it might be a relatively straightforward and acceptable way of psychologists impacting on care plans for many clients.

Lastly, one of the key issues that arose from reflecting on the assessment measures used was the need for culturally appropriate norms. The participants' experience of the research process and responses to the measures may have been influenced somewhat by cultural differences between the researcher and participant. These issues are commonly reflected on in qualitative research, but less so in quantitative research where they are equally relevant.

References

American Psychiatric Association (1994). *Diagnostic and Statistical Manual of Mental Disorder (4th ed.)*. American Psychiatric Press. Washington, DC.

Barker, S., Barron, N., McFarland, B. H. & Bigelow, D. A. (1994). A community ability scale for chronically mentally ill consumers: Part I. Reliability and validity. *Community Mental Health Journal, 30*, 363-383.

Benton, A. L., & Hamsher, K. (1978). *Multilingual Aphasia Examination (manual, revised)*. Iowa City, IA: University of Iowa.

Brandt, J., (1991). The Hopkins Verbal Learning Test: development of a new verbal memory test with six equivalent forms. *Clinical Laboratory Neuropsychology, 5*, 125-142.

Corcoran, R., Mercer, G., & Frith, C. D. (1995). Schizophrenia, symptomatology and social inference: investigating theory of mind in people with schizophrenia. *Schizophrenia Research, 17*, 5-13.

Frith, C. D., & Corcoran, R. (1996). Exploring 'theory of mind' in people with schizophrenia. *Psychological Medicine, 26*, 521-530.

Langdon, R., Michie, P., Ward, P., McConaghy, N., Catts, S., & Coltheart, M. (1997). Defective self and/or other mentalising in schizophrenia: a cognitive neuropsychological approach. *Cognitive Neuropsychiatry, 2*, 167-193.

Psychological Corporation. (1999). *Wechsler Abbreviated Scale of Intelligence (WASI) manual*. San Antonia, TX: Author.

Reitan, R. M., & Wolfson, D. (1985). *The Halstead-Reitan Neuropsychological Test Battery*. Neuropsychology Press, Tucson, AZ.

Velligan, D. I., DiCocco, M., Bow-Thomas, C. C., Cadle, C., Glahn, D. C., Miller, A. L.,

Biggs, M. M., Shores-Wilson, K., McKenzie, C. A., Crismon, M. L. (2004). A

brief cognitive assessment for use with schizophrenia patients in community

clinics. *Schizophrenia Research, 71,* 273-283.

Ventura, M. A., Green, M. F., Shaner, A., & Liberman, R. P. (1993). Training and

quality assurance with the brief psychiatric rating scale: "The drift buster".

International Journal of Methods in Psychiatric Research, 3, 221-244.

Appendix 1: Inclusion Criteria and Instructions for Referring

Clients to the Study

Brief Cognitive Assessment in First Episode Psychosis
Allison Whitty

Study Inclusion Criteria
1. Client in either Tower Hamlets or Hackney early intervention services.
2. Able to sit with an interviewer for a period of time and answer questions.
3. Aged 16-35.
4. Fluent English speakers.
5. Either gender.
6. No history of head injury or neurological disability.

If you have any queries about the suitability of a patient please contact me on the above number.

If you have a client on your caseload who fits the above criteria:

1. Please give them the accompanying patient information sheet about the study, and ask them if it would be OK for me to contact them. (Passing on their contact details to me does not mean they are committing themselves to take part in the study. I will contact them to answer any questions they may have. If they would like to take part I will arrange a time to meet with them. If they do not wish to take part I will not question why).

2. If the person is happy for me to contact them, please leave their contact details (name, number & address) on the accompanying sheet and place in my tray. Alternatively you may contact me on the above number or email address to pass on this information.

3. When I have spoken to the person, I will contact you to let you know whether or not they have decided to take part in the study. If they do take part, after I have assessed them I will contact you to go through a brief questionnaire which asks about their social and community functioning. If they consent for a summary of their results to be included in their file I will provide you with a brief report of this information.

Thank you.

Appendix 2: Client Information Leaflet
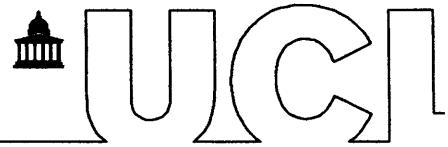
CLIENT INFORMATION SHEET

Study title: *Brief Cognitive Assessment in First Episode Psychosis*

You are 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 some time to read the following information carefully. Talk to others about the study if you wish.

What is the purpose of the study?

Research has suggested that some individuals with psychosis have difficulties with memory, attention and thinking. However, most of this research has looked at individuals who have had psychosis for a long time. The purpose of this study is to see if individuals with a more recent onset of psychosis have difficulties with memory, attention and thinking. This could mean that in the future, interventions could be developed for people suffering from psychosis, to help them better manage their memory, attention and thinking.

Why have I been chosen?

You have been chosen because you are a patient who is under the care of an early intervention service.

Do I have to take part?

No, you do not have to take part in this study. 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 you will be asked to sign a consent form. You are still free to withdraw from the study at any time and without giving a reason. A decision to withdraw at any time, or a decision not to take part in this study, will not affect the standard of health care you receive.

What will happen to me if I take part?

If you decide to take part, you will be asked to attend the early intervention service for one appointment. This appointment is expected to last for one hour and thirty minutes, however the time taken to complete the study may vary with each participant. During your visit, you will have an opportunity to ask any questions you may have about the study. You will then be asked to complete some tasks that examine memory, attention and thinking. You may find some of these tasks easy, while others may be more difficult. You will also be asked a few details about yourself, such as your age and educational background. You may be asked to have your responses on one of the study questionnaires audio recorded. This is so that the accuracy of my ratings can be checked by another researcher who is involved in the study. This is entirely optional, and if you decide not to be recorded you may still participate in the study. Any information you provide will be treated in confidence. You will receive £15 for your participation.

What are the possible disadvantages and risks of taking part?
There are no major disadvantages or risks. Most people feel a little anxious when performing a task they are unfamiliar with. However, no one gets all the tasks completely right, and all that is required is your best effort.

What are the possible benefits of taking part?
You will have the opportunity to consent to a brief summary of your results being included in your file at the early intervention service. This may be helpful for those involved in your care. However, if you would not like your scores to be put in your file you do not have to consent to this.

What if there is a problem?
It is unlikely that any problems will arise, but if you are unhappy participating in the study you are free to withdraw at any time. If you wish to complain about any aspect of the way you have been approached or treated during the course of this study, the normal National Health Service complaints mechanisms may be available to you. If you are harmed and this is due to someone's negligence then you may have grounds for legal action for compensation against East London and the City Mental Health Trust, but you may have to pay your legal costs. The Patient Advice and Liaison Service (PALS) at the East London and The City Mental Health NHS Trust is available to help if you need advice or have concerns. They may be contacted on Freephone: .

Will my taking part in the study be kept confidential?
Yes. Any information you provide in the study will be kept strictly confidential, and will not be passed on to a third party. You will have an opportunity to consent to a brief summary of your results being included in your file at the early intervention service. All documents related to your participation will be stored in a locked cupboard, and only those people involved in the study will be able to view them. If you consent to having your responses on one of the study questionnaires audio recorded, the tape will be listened to by another researcher and then destroyed within 1 month of your participation. The rest of the data will be stored for up to 3 years and then destroyed. If you participate in the study you will be given a unique patient identifier code and your name will not appear on any of the documents.

What will happen to the results of the research study?
The results of the study will form part of a dissertation for post graduate qualification. At a later date the results may be published in a scientific journal. You will not be identified in any reports resulting from this study, and you will have the option of being notified of any publication that emerges from the study.

Who has reviewed the study?
This study has been approved by the East London and City Research Ethics Committee.

Thank you for taking the time to read this information sheet.

Allison Whitty, Trainee Clinical Psychologist.

Appendix 3: Letter of Ethical Approval

21 August 2006

Miss Allison Whitty
Trainee Clinical Psychologist
Sub-Department of Clinical Health Psychology
University College London
Gower Street
London
WC1E 6BT

Dear Miss Whitty

Full title of study: **The Role of the 'Brief Cognitive Assessment' in the Screening of Cognitive Deficits in First Episode Psychosis.**

REC reference number:

Thank you for your letter of 26th July 2006 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 Chair.

Confirmation of 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.

Ethical review of research sites

The Committee has designated this study as exempt from site-specific assessment (SSA. There is no requirement for [other] Local Research Ethics Committees to be informed or for site-specific assessment to be carried out at each site.

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.

Approved documents

The final list of documents reviewed and approved by the Committee is as follows:

Document	Version	Date
Application	2	26th July 2006
Investigator CV	1	03 March 2006
Protocol	2	08 March 2006

Covering Letter	1	19 May 2006
Letter from Sponsor	1	10 February 2006
Peer Review	1	19 May 2006
Compensation Arrangements	1	01 August 2005
Questionnaire	2	
Letter of invitation to participant	2	05 February 2006
Participant Information Sheet	3	26 July 2006
Participant Consent Form: Consent form	2	05 February 2006
Response to Request for Further Information	1	
Authorisation Letter for 2nd yr trainee	1	20 July 2006
Honarary Contract re Letter of Indemnity	1	15 June 2006
Letter sanctioning study and confirming site	1	07 August 2006
Curriculum Vitae	1	03 March 2006
Checklist	1	19 May 2006
Funding Letter	1	05 January 2006
Indemnity	1	05 May 2006
Test Booklet Form 1	1	19 May 2006
Trailmaking Part A	1	19 May 2006
Guidelines for Administering the BCA	1	19 May 2006
Picture Sequencing Task	1	19 May 2006
Theory of Mind Stories	1	19 May 2006
Multnomah Community Ability Scale	1	19 May 2006
Brief Psychiatric Rating Scale	1	19 May 2006

Research governance approval

You should arrange for the R&D department at all relevant NHS care organisations to be notified that the research will be taking place, and provide a copy of the REC application, the protocol and this letter.

All researchers and research collaborators who will be participating in the research must obtain final research governance approval before commencing any research procedures. Where a substantive contract is not held with the care organisation, it may be necessary for an honorary contract to be issued before approval for the research can be given.

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.

Please quote this number on all correspondence

With the Committee's best wishes for the success of this project

Yours sincerely

Chair

SF1 list of approved sites

Email:

Copy to: R&D Department
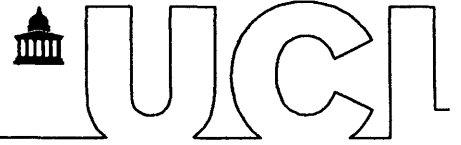   University College London
                    Gower Street
                    London
                    WC1E 6BT

SF1 list of approved sites

Appendix 4: Consent Forms

CONSENT FORM

Title of project: *Brief Cognitive Assessment in First Episode Psychosis*

Name of Researcher: Allison Whitty

1. I confirm that I have read and understood the information sheet dated ....
for the above study. I have had the opportunity to consider the information, ask
questions and have these answered satisfactorily.

YES / NO (Please circle)   (Please initial)

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.

YES / NO (Please circle)   (Please initial)

3. When I have completed the study, I wish to have a brief summary of my results
included in my file.

YES / NO (Please circle)   (Please initial)

4. If the results of the study are published, I wish to be informed where I may access
the publication. I understand that I need to provide a postal address where I may be
contacted.

YES / NO (Please circle)   (Please initial)

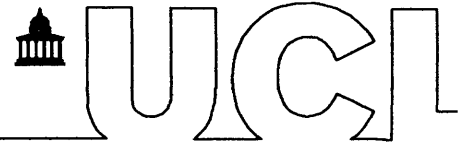If YES, address:...

5. I agree to take part in the above study.

Participant's name:_____   Researcher's name: _____

Date: _____   Date:_____

Signature: _____   Signature: _____

CONSENT FORM FOR AUDIO RECORDING

Title of project: *Brief Cognitive Assessment in First Episode Psychosis*

Name of Researcher: Allison Whitty

As part of this research we would like to audio record some of the participants' responses on one of the questionnaires only (the brief psychiatric rating scale). This is so that another researcher (a clinical psychologist from University College London) can listen to the tape and rate the questionnaire separately from the main researcher (Allison Whitty). We will then compare our ratings and reach an agreement where there are any differences. This will improve the accuracy of our ratings.

As with all the study materials your name or any identifying information will not appear on the cassette tape. The tape will be stored at UCL until listened to by the clinical psychologist and then destroyed within a month of your participation in the study. Only Allison Whitty and the clinical psychologist will have access to the tape and no copies will be made.

If you decide not to give consent for audio recording, the standard of care you receive at the early intervention service will not be affected and you may still take part in the study. If you do provide consent for tape recording you may withdraw this consent at anytime and the tape will not be used.

1. I confirm that I have read and understood the above information. I have had the opportunity to consider the information, ask questions and have these answered satisfactorily.
YES / NO (Please circle)   (Please initial)

2. I understand that being tape recorded 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.
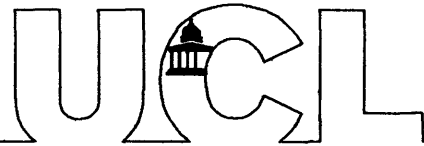YES / NO (Please circle)   (Please initial)

3. I AGREE/DO NOT AGREE (please circle) for my responses on the Brief Psychiatric Rating Scale to be audio recorded:

Participant's name:_____Researcher's name:_____

Date: _____ Date: _____

Signature: _____ Signature: _____

Appendix 5: Report Template

**Confidential Report – The results of this report should only be interpreted or
discussed with the patient by a clinical psychologist.**

RE: Patient's name and d.o.b.
Address:
Date of report:

Mr./Ms.... consented to participate in a research project entitled "Brief Cognitive
Assessment in First Episode Psychosis". The research project was approved by East
London and The City Research Ethics Committee, and was carried out by Allison
Whitty as part of her Doctoral training in Clinical Psychology. Mr./Ms.... was tested
on dd/mm/yyyy and consented for a summary of their results to be included in their
file at Tower Hamlets Early Intervention Service (THEIS)/Hackney Early
Intervention Service (EQUIP). It should be noted that as with all neuropsychological
tests, these results may have been influenced by factors such as motivation,
medication and mood and so may not give a complete picture of Ms./Mr......'s
ability.

Behaviour during the session
Mr./Ms.... arrived on time for the assessment. He/she co-operated well with the
assessment process and completed all measures administered.

Brief Cognitive Assessment Battery (BCA; Velligan et al. 2004)
The BCA consists of three standardised tests commonly used in clinical
neuropsychology: Trails A and B (Reitan, 1958), Verbal Fluency (Benton &
Hamsher, 1976) and the Hopkins Verbal Learning Test (Brandt, 1991).

i) Trails A and B
Trails A measures psychomotor speed while Trails B measures psychomotor speed
and divided attention. Trails A consists of a series of numbered circles (1–25),
scattered at random on a worksheet. The participant is required to draw a line
between the circles in numerical order as quickly as possible. Trails B has both
numbers and letters arrayed in a random order. The participant is required to draw
lines between numbers and letters in an alternating sequence (i.e. 1-A, 2-B, 3-C etc.).
The score is based on time alone. Mr./Ms.... completed Trails A in ? seconds and
Trails B in ? seconds. The time taken to complete Trails A/Trails B/both tests were
lower/higher/ within the normal range when compared with normative data (Selnes et
al., 1991).

ii) Hopkins Verbal Learning Test (HVLT)

The HVLT is a list learning exercise that assesses verbal memory. It consists of a list of twelve nouns, which are drawn from three different semantic categories, with four nouns coming from each category. The list is read aloud to the participant and their free recall of the list is recorded. The procedure is repeated twice making a total of three trials. After a delay of 20-25 minutes the participant is asked if they can recall any words from the list. Mr./Ms... recalled ? words in total from the first three trials. This was lower/higher/ within the normal range when compared with normative data (Brandt, 1991). Mr./Ms.... recalled ? words from the delayed recall trial. This score is lower/higher/ within the normal range when compared with normative data (Brandt, 1991).

iii) Verbal fluency

Verbal Fluency is a test of executive function. In the first part of the task the participant is asked to generate as many words as possible beginning with a letter of the alphabet (F, A or S). In the second part the participant is asked to generate as many words as possible from a category of animals. The score is the total number of words generated in each category in one minute. Mr./Ms.... generated ? words for letter fluency, and ? words for category fluency. Compared to normative data this score is higher/lower/within the normal range (Selnes et al., 1991).

Theory of Mind (ToM) tasks

ToM may be defined as the capacity to infer one's own and other people's mental states (thoughts, intentions and beliefs) and to use this capacity to predict and understand behaviours. There were three ToM tasks used in this study: Hints Task (Corcoran et al., 1995), Picture Sequencing Task, (Langdon et al., 1997) and ToM Stories (Frith & Corcoran, 1996). As yet there are no normative data available for comparison so the results are compared with those of control subjects from published studies.

i) Hints Task

This task consists of ten short vignettes. Each vignette is read aloud to the participant. The participant is asked to infer what the story character really meant by what he/she said. If an inference is not made or if an inappropriate conclusion is drawn, the participant is given additional information in the form of a more obvious hint. Mr./Ms.... scored ? out of 20. This score was higher/lower/equal to scores by control subjects (Corcoran et al., 1995).

ii) Picture Sequencing Task

This task consists of stories depicted in black and white drawings. Each story is presented on four cards. There are 2 practice sequences and 3 experimental sequences: social script, mechanical and false belief. Social script stories depict people acting out everyday social routines and test logical routines using social script knowledge (involving no appreciation of mental states). Mechanical stories depict cause and effect sequences and test ability to infer causal relations. False belief stories depict the story character acting on the basis of false beliefs; these stories are the test of Theory of Mind. There are 4 stories in each sequence. The participant is presented with each set of story cards placed face down and is required to turn the cards over and to order them in a logical sequence. The order of card placement and

the time taken to complete each scenario is recorded. Mr./Ms.... scored an average of ? in the social script sequence, ? in the mechanical sequence and ? in the false belief sequence. The maximum score achievable per sequence is 6. Relative to controls, these scores were in the higher/lower/equal to range. Mr./Ms. completed the social script task in ? seconds, the mechanical task in ? seconds and the false belief task in ? seconds. Relative to controls, these timings were in the higher/lower/equal to range (Langdon et al., 1997).

iii) ToM Stories

Four ToM story tasks were read aloud to the participant. Two of the stories were 'first order' in which a story character has a false belief about a particular situation. The other two stories were 'second order' in which a story character has a false belief about the belief of another character. While the stories were read aloud, corresponding cartoon pictures were shown. The participant had to answer two questions for each story. The first question assessed ToM, and the second was a control question that tested the participant's understanding and memory of the story. Scores were only considered when the second question (memory) was answered correctly. The participant was given one point for each of the first and second order questions. Mr./Ms. answered ? of the memory questions correctly/incorrectly so their scores on this task were/were not considered. On the first order belief questions Mr./Ms. score ? out of 2. On the second order belief questions Mr./Ms. scored ? out of 2 (Frith & Corcoran, 1996).

Weschler Abbreviated Scale of Intelligence (WASI; Psychological Corporation, 1999)

The WASI provides a reliable and brief test of intelligence quotient (IQ) comparable to the Weschler Adult Intelligence Scale 3rd edition (WAIS-III). The two-subtest form of the WASI, including Vocabulary and Matrix Reasoning, provides an estimate of full scale IQ only. The Vocabulary subtest is a test of verbal comprehension. The participant is read aloud a list of words of increasing difficulty and is asked to report the meaning of each word. Matrix Reasoning is a nonverbal perceptual reasoning task in which the participant is asked to complete a missing portion of abstract patterns. The combined score on the Vocabulary and Matrix Reasoning subtests gives Mr./Ms.... a full scale IQ of ? (confidence interval ?-?).

British Psychiatric Rating Scale (BPRS; Ventura et al., 1993)

The BPRS is a clinician-rated tool designed to assess the nature and extent of psychopathology in patients with psychotic illness over the two weeks prior to interview. Participants are interviewed in a semi-structured way and are rated on 24 different symptom constructs according to a seven-point scale ranging from 'not present' to 'extremely severe'. A total score is obtained from these ratings. Mr./Ms. scored ? on the BPRS. Items which received a score between moderate (a score of 3) and extremely severe (a score of 7) were......

Summary

Scores on the BCA suggest.... Scores on ToM suggest........... Scores on the BPRS suggest...............

Yours sincerely,

Allison Whitty
Trainee Clinical Psychologist, UCL
Supervised by ....

References

Benton, A. L., & Hamsher, K. (1978). Multilingual Aphasia Examination (manual, revised). Iowa City, IA: University of Iowa.

Brandt, J. (1991). The Hopkins Verbal Learning Test: development of a new verbal memory test with six equivalent forms. *Clinical Laboratory Neuropsychology*, 5, 125-142.

Corcoran, R., Mercer, G., & Frith, C. D. (1995). Schizophrenia, symptomatology and social inference: investigating theory of mind in people with schizophrenia. *Schizophrenia Research*, 17, 5-13.

Frith, C. D., & Corcoran, R. (1996). Exploring 'theory of mind' in people with schizophrenia. *Psychological Medicine*, 26, 521-530.

Langdon, R., Michie, P., Ward, P., McConaghy, N., Catts, S., & Coltheart, M. (1997). Defective self and/or other mentalising in schizophrenia: a cognitive neuropsychological approach. *Cognitive Neuropsychiatry, 2,* 167-193.

Psychological Corporation. (1999). Wechsler Abbreviated Scale of Intelligence (WASI) manual. San Antonia, TX: Author.

Reitan, R. M. (1958). Validity of the Trail Making test as an indicator of organic brain damage. *Perceptual and Motor Skills*, 8, 271-276.

Selnes, O. A., Jacobson, L., Machado, A. M., Becker, J. T., Wesch, J., Miller, E. N., Visscher, B., & McArthur, J. C. (1991). Normative data for a brief neuropsychological screening battery. *Perceptual and Motor Skills, 73,* 539-550.

Velligan, D. I., DiCocco, M., Bow-Thomas, C.C., Cadle, C., Glahn, D. C., Miller, A.L., Biggs, M. M., Shores-Wilson, K., McKenzie, C. A., & Crismon, M. L. (2004). A brief cognitive assessment for use with schizophrenia patients in community clinics. *Schizophrenia Research*, 71, 273-283.

Ventura, M. A., Green, M. F., Shaner, A., & Liberman, R. P. (1993). Training and quality assurance with the brief psychiatric rating scale: "The drift buster". *International Journal of Methods in Psychiatric Research, 3,* 221-244.

Appendix 6: Demographic Information Questionnaire

Demographic Information

1. Gender..

2. DOB...

3. Age..

4. Ethnicity...

5. Language spoken at home.............................

6. Years of education.....................................

7. Employed or unemployed.............................

8. Residential status.....................................

9. Diagnosis..

10. Age of onset..

11. Duration of illness...................................

12. Number of relapses...................................

13. Type of neuroleptic medication....................
    and dosage............................

14. Any other psychiatric medication.................
    and dosage............................

15. Whether or not used illicit drugs in past month ....
    and type of drug(s) used...

16. Drank alcohol in past 24 hours...

17. Caffeine in past 12 hours..