Social cognition, neurocognition and symptomatology in first episode psychosis

by

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ABSTRACT

Neurocognitive impairment and social dysfunction have long been recognised as core components of schizophrenia. Research has linked deficits in neurocognition (e.g., attention, memory, and executive functions) to poor social functioning and outcomes, however a large amount of variance has been left unaccounted for by such impairments. In an effort to identify other contributing factors to poor social functioning in schizophrenia, research interest has more recently shifted to social cognition given that such mental processes are thought to govern social behaviour. The empirical literature in this field has grown considerably over the past two decades, with social cognitive impairment now also considered as a key characteristic in schizophrenia. Furthermore, social cognitive deficits have been shown to adversely affect social functioning in individuals with the illness. Most research has been conducted using established schizophrenia samples and therefore less known about social cognition in the earlier phase of illness. There has been some evidence of deficits in social cognition in people who have experienced a first-episode of psychosis (FEP), although methodological issues in this area of research have limited interpretation of findings and more research is needed to elucidate the relationship between social cognition and social functioning.

This thesis aimed to ascertain whether multiple areas of social cognition are impaired in FEP, and to explore the relationship between social cognition and other core features of psychosis, including neurocognition, symptomatology and social functioning. The findings were anticipated to inform rehabilitative treatment approaches in early psychosis, particularly those related to addressing social dysfunction. The first four chapters of this thesis provide a background to schizophrenia and review the literature with an emphasis on social cognition. Chapter Five stipulates the rationale for the
current research and specifies the aims and hypotheses of four separate research studies, which are then presented sequentially in Chapter Six through to Chapter Nine. In the final chapter, a general discussion is presented that includes a summary of the research aims and findings, and highlights the methodological issues and limitations of the research. Recommendations for future research are also offered, and the chapter concludes with the clinical implications of the research findings.
CHAPTER 1: Conceptualisation of Psychosis and Schizophrenia

1.1 Background and Diagnostic Features

Schizophrenia is a complex mental illness that remains to be fully understood in terms of aetiological factors and symptom development. It is considered to be a multifaceted disorder characterised by major disturbances in cognition, emotion and behaviour (Andreasen, 1997). Typical clinical symptoms include hallucinations, delusional beliefs, disordered thinking, and flattened or inappropriate affect. Such symptoms usually first appear in adolescence or young adulthood (Luna & Sweeney, 2001; Rössler, Salize, van Os, & Riecher-Rössler, 2005), with deficits in cognitive functioning also evident in the early course of illness, even before overt psychotic symptoms are displayed (Keefe et al., 2006). Social dysfunction is common in schizophrenia, however it remains unclear as to which specific factors underlie the poor social outcomes experienced by individuals with the disorder.

According to the latest version of the Diagnostic and Statistical Manual of Mental Disorders (DSM), (American Psychiatric Association [APA], 2013), schizophrenia is diagnosable if psychotic symptoms are present most of the time during a one-month period and signs of the disturbance persist for at least six months. There must also be significant impairment in social and occupational functioning. The more generic psychiatric term “psychosis” applies when certain symptoms are present, but the DSM criteria for schizophrenia are not fully met, for example, regarding symptom duration or frequency. Thus, schizophrenia is a specific disorder involving psychosis or psychotic
symptoms. Other organic brain disorders such as stroke, tumours and head trauma can also lead to psychosis, however for the purpose of this thesis, references to psychosis are limited in scope to schizophrenia. Individuals without a history of organic brain disorder who have suffered an initial episode of psychosis are at increased risk of developing schizophrenia, therefore it is important to investigate possible causal factors of the disorder as early as possible in order to develop and implement targeted intervention strategies before the illness becomes full-blown.

The conceptualisation of schizophrenia has changed considerably over the years, with continued debate regarding the reliability and validity of diagnostic categories. Emil Kraepelin’s description of ‘dementia praecox’ in the 19th century was an initial step toward understanding schizophrenia (Sanbrook & Harris, 2003). Kraepelin was the first to suggest a distinction between the organic psychoses and functional psychoses, with the former representing psychotic experiences in the presence of a physiological anomaly and the latter occurring in the absence of any such organic disturbances (Katona & Robertson, 2000). Dementia praecox was an example of the functional psychoses in that organically healthy individuals demonstrated signs of irreversible deterioration across a variety of mental processes (Katona & Robertson, 2000). The term ‘praecox’ was used to differentiate this type of earlier onset dementia from other forms of dementia that usually occurred in late adulthood (Kyziridis, 2005), and it is dementia praecox that corresponds to the current conceptualisation of schizophrenia.
Kraepelin identified four symptom subtypes: paranoid, marked by delusions and auditory hallucinations; hebephrenic (or disorganised), characterised by bizarre behaviour, thought incoherence, and blunted or inappropriate affect; catatonic, in which motor function is affected by either immobility or excessive activity; and simple, marked by slow decline accompanied by apathy and social withdrawal (Katona & Robertson, 2000). In addition to emphasising diversity of symptoms, Kraepelin described a generally chronic course of illness with poor prognosis (Andreasen & Carpenter, 1993), which likely contributed to the fact that early treatment and intervention strategies were not considered until decades later (Sanbrook & Harris, 2003). In more recent years, longitudinal studies of schizophrenia have indicated that the course of illness is not necessarily chronic for all sufferers and that prognosis can be improved with appropriate treatment and earlier interventions (Sanbrook & Harris, 2003).

Whilst Kraepelin's conceptualisation of schizophrenia was limited to dementia praecox, the subsequent work of Eugen Bleuler and John Hughlings-Jackson further developed the concept of schizophrenia. In fact, it was Eugen Bleuler who proposed the term schizophrenia, which has Greek origins in its terminology and literally means split mind (Kyziridis, 2005). Bleuler formulated the concept of two distinct types of psychopathology, specifically, fundamental and accessory symptoms. Fundamental symptoms comprised of four “A” categories: Affective incongruity, whereby affect is either blunted or inappropriate (e.g. smiling when discussing something is sad), as to suggest a dissociation between thoughts and feelings, or blunted; Associative Loosening, that is, the lack of logical connections between thoughts; Ambivalence, in which conflicting ideas and feelings coexist; and Autism, referring to a withdrawal from reality
into an inner fantasy world (Katona & Robertson, 2000). Bleuler’s four ‘A’s were all considered relatively stable features of the disorder (Andreasen & Carpenter, 1993). On the other hand, accessory symptoms comprised of hallucinations and delusions (Andreasen, 1997), symptoms that occurred transiently for various periods of time (Andreasen & Carpenter, 1993). In accordance with current day conceptualisations of schizophrenia psychopathology, Bleuler’s fundamental symptoms are akin to ‘negative’ symptoms, whereas accessory symptoms reflect ‘positive’ symptomatology.

Kraepelin and Bleuler’s depiction of psychosis placed more of an emphasis on negative symptomatology and disruptions to affect and cognition, however in the 1960’s and 1970’s, the focus shifted away from negative symptoms and cognitive impairment to positive-type symptoms. Kurt Schneider had a great influence in this regard, having purported that ‘first-rank’ symptoms (i.e. those typically considered positive symptoms) represented the core feature of the disorder in that these symptoms inhibited one’s ability to determine boundaries between the self and others and thus impacted on personal autonomy (Andreasen, 1997). Specific first-rank symptoms include: auditory hallucinations, thought withdrawal, thought insertion and broadcasting, or external thought control (Kyziridis, 2005). Schneider further described ‘second-rank’ symptoms, which were also considered characteristic features of the disorder however deemed less important regarding diagnosis. Examples of second-rank symptoms are: hallucinations (non auditory or somatic), flat or blunted affect, shifts in mood, and sudden or secondary delusional ideas (Kyziridis, 2005).
Schneider’s conceptualisation of psychosis has had a profound impact on the diagnostic criteria for schizophrenia in recent editions of the DSM. According to the current version of the DSM, i.e. DSM-5 (APA, 2013), the majority of the primary symptom criteria for schizophrenia as depicted in Criterion A (see Appendix A for a complete list of the diagnostic criteria) remain typically reflective of Schneider’s symptom conceptualisation. Specifically, for any diagnosis of schizophrenia, an individual must display two of the five characteristic symptoms, which include: delusions, hallucinations, disorganised speech, disorganised or catatonic behaviour, and negative symptoms. The recently superseded fourth edition of the DSM (DSM-IV-TR) (APA, 2000) included a clause that stated if delusions were bizarre, or if hallucinations were auditory and first-rank in nature (e.g. two or more voices conversing), then only one symptom was required for a diagnosis of schizophrenia. However, this clause was removed in the DSM-5 due to the nonspecificity of Schneiderian symptoms, and also the variability in clinical judgment regarding whether delusions are bizarre or non-bizarre (APA, 2013). This exclusion was one of two main updates from DSM-IV-TR to DSM-V in relation to Criterion A. The second notable change was that one of the two Criterion A symptoms required for a diagnosis of schizophrenia is now required to be “positive” in nature (e.g. hallucinations, delusions and/or disorganised speech), as positive symptoms are regarded as providing greater diagnostic validity than other symptoms of the disorder.

Despite the diagnostic significance of positive symptoms, a vast body of literature now exists that shows neurocognitive impairment is also a prominent feature of schizophrenia (Keefe et al., 2006) however it remains unaccounted for on a diagnostic level. In fact, it has been demonstrated that neurocognitive impairments can manifest
before the onset of positive symptoms, such as, hallucinations and delusions (Keefe et al., 2006; Klosterkötter, Albers, Steinmeyer, Hensen, & Saß, 1995). Furthermore, research has indicated that neurocognitive deficits, as well as negative symptoms, are more widely linked to social impairment in schizophrenia than positive symptoms (Ventura, Helleman, Thankes, Koellner, & Nuechterlein, 2009). In light of such findings, the classification of psychotic disorders has been debatable, with a particular emphasis on the need for re-evaluation of the diagnostic criteria of schizophrenia in the DSM. For example, Bora, Yücel, and Pantelis (2010) proposed inclusion of neurocognitive deficits in the DSM-5 diagnostic criteria perhaps as a specifier, or as a feature of the disorder within the proposed hybrid categorical-dimensional classification system. However, neurocognitive impairment was not acknowledged in the DSM-5 criteria for schizophrenia and it is too early to predict the influence this will have on the field.

Based on the research to date, there seems to be a definite need for the reconsideration of neurocognitive deficits in the diagnostic criteria of psychotic disorders such as schizophrenia. This may lead to earlier detection of illness, and therefore earlier and more targeted treatment interventions depending on symptom manifestation, which could possibly prevent disease progression and improve psychosocial outcomes.

1.2 Prevalence and Course of Illness

Although the lifetime prevalence of schizophrenia is generally low, a recent systematic review suggested that approximately 0.7% of the general population will suffer from the disease (McGrath, Saha, Chant, & Welham, 2008), and it has been rated as the third
highest cause of disability amongst other medical conditions (Ustun et al., 1999). In Australia specifically, schizophrenia ranked as the third highest cost to the mental health system (SANE, 2002). These astounding statistics highlight the considerable impact of the illness on functioning, especially that of a social nature, which emphasises the particular importance of research into the underlying factors of social impairment.

In terms of illness onset, males are typically aged 18-25 years when symptoms manifest, whereas females are usually between the ages of 25-30 years (APA, 2000). Psychosis generally affects males and females in the same way, however females are inclined to fare better during the course of illness (Rössler et al., 2005). For example, men have been found to have more hospitalisations and for longer admission periods than females (Usall, Ochoa, Araya, & Márquez, 2003). Furthermore, an Australian study found women had better premorbid functioning than men and also experienced a less severe course of illness, with lower levels of disability and better social reintegration following acute episodes of illness (Morgan, Castle, & Jablensky, 2008).

Although sex differences have been reported in terms of course of illness (Angermeyer, Kuhn, & Goldstein, 1990; Morgan et al., 2008), gender is not considered to be the only determinant as to prognosis and outcome. Many other factors are thought to influence the course of illness, for example, vulnerability to trait anxiety, duration of untreated psychosis, and an external locus of control (Jobe & Harrow, 2010). As such, there is considerable variability regarding course of illness; patients can experience multiple acute episodes with either good or partial recovery in between episodes, they can remain symptomatic long after an initial first episode with varying levels of
deterioration, or some even recover fully with or without antipsychotic medication (Jablensky, 2000; Jobe & Harrow, 2010). Jablensky (2000) reported that only 8% of patients achieved full symptom remission with good recovery after an initial episode of psychosis, which indicates that outcomes in schizophrenia are generally poor. However, the illness is not necessarily characterised by progressive deterioration as originally suggested by Kraepelin; its course is more likely to involve episodic periods of symptoms with variations in level of functioning and impairment (Harrow & Jobe, 2010).

In fact, symptom remission and recovery appears to be greater when treatment occurs at the time of the first-episode. In a longitudinal study of first episode psychosis patients, less than 9% of the sample with a specific diagnosis of schizophrenia/schizophreniform disorder (n = 158) displayed continual psychotic symptoms at 12 months post-treatment (Edwards, Maude, McGorry, Harrigan, & Cocks, 1998). Furthermore, in another seven-year follow up study of first-episode patients, 57% of individuals with a schizophrenia/schizophreniform diagnosis reported some paid employment in the preceding two years (Jackson & McGorry, 2010). These results suggest that early intervention can lead to better functional outcomes and reduce the likelihood of a chronic, enduring course of illness.

1.3 Burden of Disease

Impairments in functioning including self-care, interpersonal relationships and employment are considered to be fundamental aspects of schizophrenia, making the disorder a major source of disability. For individuals with a typical onset of illness, that is, in adolescence or early adulthood (Luna & Sweeney, 2001; Rössler et al., 2005), many
will continue to live with their family of origin as the illness can preclude development and maintenance of social skills and goal-directed activity thus making it difficult to live independently. For others who do not receive ongoing care from their families, community housing or support services are often utilised. In any case, the illness places a significant burden on carers, mental health services, and the welfare system.

The direct and indirect financial costs of schizophrenia in Australia are astounding. In a report issued by the mental health charity organisation SANE (2002), it was stated that the total cost of the illness in the Year 2001 was $1.85 billion. Direct cost to the health system was reported at $661 million, which included hospitalisation, community mental health treatment, pharmaceuticals, and other medical expenses (e.g. GP and specialist visits). Indirect costs were reported at $722 million, reflecting lost earnings from individuals unable to work due to the illness, carer costs, legal and forensic costs, and premature death. In addition, $274 million was afforded for welfare payments (e.g. disability support pensions) and $190 million reflected lost tax revenue. Even more astonishing, recent data from the 2010 Australian National Survey of Psychosis (see Neil, Carr, Mihalopoulos, Mackinnon, & Morgan, 2013) indicated that the illness costs Australian society $4.91 billion annually, and the Australian government $3.52 billion.

The burden on family is also an important issue, especially since the advent of deinstitutionalisation, with most affected individuals supported in the home whilst receiving community-based treatment rather than extended periods of hospitalisation (SANE, 2002). Perhaps an even greater burden on families are those individuals who have poor insight and will not seek treatment and/or resist treatment, and thus remain
in the family home debilitated by psychotic symptoms and social impairments. Numerous studies have demonstrated that carers of persons with schizophrenia experience significant stressors and feel a high level of burden (reviewed in Saunders, 2003). Family members can become highly stressed when subject to one's psychotic symptoms and dysfunctional behaviours, and also experience other negative emotions in dealing with the diagnosis and associated stigma (Rössler et al., 2005). They may also find it difficult to communicate and interact with the affected individual, especially as people with schizophrenia often withdraw and isolate themselves, which can make co-existence burdensome. Furthermore, it is not uncommon for carers to suffer emotionally and develop their own mental health problems as a result of having to live with a mentally unstable individual who cannot function independently. For example, McGrath and Davies (1999) stated that carers can experience depressive-type symptoms, such as, unreasonable guilt as a consequence of thinking they may have contributed to the development of the disorder, or feel grief for the fact that their child's quality of life and outcomes will likely be poor.

In order to decrease the heavy burden of schizophrenia, it is important to develop treatment targets that will not only ameliorate symptoms, but also improve social functioning and other outcomes for those with the illness. Early intervention programs have already shown relatively positive outcomes for people with first-episode psychosis in terms of symptom remission and social/vocational recovery (Edwards et al., 1998; Jackson & McGorry, 2010), thus treatment early in the course of illness seem imperative. Currently, the primary treatment goal is to stabilise patients by treating symptoms with the use of antipsychotic medication and to improve quality of life by
reintegration back into the community (Rössler et al., 2005). To improve outcomes further, particularly those associated with social and vocational functioning, a greater understanding of the underlying factors that contribute to social impairment in schizophrenia is required. This would enable development of targeted non-pharmacological interventions to aid rehabilitation.

1.4 Aetiological Considerations

As aforementioned, schizophrenia is a heterogeneous disorder with a variety of clinical features that affect individuals in a non-uniform manner. Despite extensive research efforts, the multifaceted nature of the illness has created difficulty in determining precise causal factors. Schizophrenia has been described from a number of theoretical perspectives, which are not central to this thesis thus will not be reviewed here, however the neurodevelopmental hypothesis (Murray & Lewis, 1987; Weinberger, 1987) is mentioned as it is in line with the fact that neurological and other developmental anomalies are present before the onset of symptoms. This view suggests that schizophrenia may result from pathological processes occurring in utero or perinatally, leading to substantial loss of neurons and abnormal neural circuitry in adolescence when the brain is reaching an adult level of maturity. Even though basic neural functions develop during childhood, brain maturation continues after puberty when more complex cognitive and social processes are acquired. It is thought that significant stress during this period of development in adolescence and early adulthood, when the disorder usually manifests, may trigger symptomatology (Weinberger, 1995). Cognitive and behavioural impairments have been observed prior to the onset of typical clinical symptoms providing evidence for the neurodevelopmental perspective
(Erlenmeyer-Kimling et al., 2000). In addition, a lack of marked progressive neurodegeneration in individuals with the illness (Marenco & Weinberger, 2000) lends further support for a neurodevelopmental basis.

Given the heterogeneity of schizophrenia, opinions differ regarding whether it is representative of a single disorder or numerous independent syndromes. It is more commonly thought the latter is the case, reflecting variable phenotypic expression of inherited genes, which possibly relate to different manifestations of the disorder. Twin studies have indicated that non-genetic influences such as environmental stressors and psychological processes may underlie or contribute to the development of schizophrenia; a notable proportion of monozygotic twins do not show any signs of the disorder (Sullivan, Kendler & Neale, 2003). As a result, cognitive functions and processes have been a focal point in schizophrenia research over the past two to three decades. The next chapter discusses cognition in psychotic disorders, with a focus on social cognition given the recent surge of interest in this field.
CHAPTER 2: Neurocognition and Social Cognition in Psychotic Disorders

2.1 Neurocognition in Early Psychosis and Schizophrenia

Although the DSM-5 diagnostic criteria for psychotic disorders does not make reference to cognitive deficits (APA, 2013), cognitive impairment has long been demonstrated in schizophrenia and first episode psychosis (FEP), and is considered to be a major contributor to the pathophysiology of psychotic disorders (Pantelis & Maruff, 2002). The cognitive abilities most consistently reported as impaired in individuals with psychotic illness include attention, memory, and executive functions (Heinrichs & Zakzanis, 1998; Hutton, et al., 2002; Pantelis & Maruff, 2002). Such abilities reflect non-social or “neurocognitive” skills, which involve processing of emotionally neutral information. Neurocognitive deficits are generally present during the first episode (Bilder et al., 1991; Nopoulos, Flashman, Flaum, Arndt & Andreasen, 1994) and appear to remain generally stable over the course of the illness (Hoff, et al., 1999).

Even though the majority of evidence indicates that neurocognitive impairment in schizophrenia is generalised, some studies have found that attentional functions are spared in early-onset schizophrenia (Øie, Rund, Sundet & Bryhn, 1998; Ueland, Øie, Landrø & Rund, 2004). These studies should be interpreted with caution for a number of reasons, including a) very small sample sizes, b) samples consisting of mixed diagnoses including schizotypal personality disorder (which is not a psychotic illness), c) inclusion of both medicated and medication-naïve participants, and d) potentially unmatched tasks regarding difficulty level. Conversely, as participants in these studies were under 18 years of age, attentional processes may remain unaffected in
schizophrenia until particular neurodevelopmental changes have occurred once the brain has fully matured post-adolescence.

Evidence from longitudinal studies has demonstrated that poor cognition in childhood, prior to the onset of any clinical symptoms, is a predictor of the development of schizophrenia (Cannon et al., 2002; Erlenmeyer-Kimling, et al., 2000). For example, Erlenmeyer-Kimling and colleagues (2000) found that offspring of individuals with schizophrenia who displayed deficits in verbal memory and attention in childhood were significantly more likely to present with a schizophrenia spectrum disorder in mid-adulthood, compared to children of parents with an affective mental illness or no psychiatric disorder at all. Such findings indicate that neurocognitive impairment is not an outcome factor but rather constitutes a predisposition to the illness with a probable genetic basis. The fact that neurocognitive deficits are present before the onset of any typical psychotic symptoms (Davidson et al., 1999; Mednick, Parnas & Schulsinger, 1987) suggests that different neural mechanisms may be responsible for the fundamental aspects of the disorder.

Evidence for neurocognitive dysfunction as an independent feature of schizophrenia is also provided by the fact that symptoms without a cognitive basis, e.g. certain negative symptoms such as blunted affect, have shown the weakest associations with neurocognitive variables (Blanchard, Kring & Neale, 1994). On the contrary, other studies that have assessed the relationship between neurocognition and symptomatology have found negative symptoms to be significantly related to neurocognitive deficits (Addington, Addington & Maticka-Tyndale, 1991; Keefe et al., 2006). The significant associations could be explained by the possibility of shared
neural mechanisms between neurocognition and certain negative symptoms, particularly those symptoms that include a high cognitive component. For example, one of the most commonly used measures of symptoms in schizophrenia and psychosis is the Positive and Negative Syndrome Subscale (PANSS; Kay, Opler, & Lindenmayer, 1989), which classifies two cognitive-based individual items (‘lack of abstract thinking’ and ‘stereotyped thinking’) as negative symptomatology. Thus, these particular symptoms may correlate to a higher degree with cognitive variables.

There has been great variability in the measures and tasks used to assess particular domains of neurocognition in schizophrenia. In an effort to establish a standardised cognitive battery primarily for use in clinical trials in schizophrenia, which was an initiative of the National Institute of Mental Health (NIMH), Nuechterlein and colleagues (2004) identified six distinct neurocognitive domains fundamentally impaired in schizophrenia that resulted from a review of factor analytic studies. The independent neurocognitive functions arising from the review included: processing speed, attention/vigilance, working memory, verbal learning and memory, visual learning and memory, and reasoning and problem solving (i.e. executive functioning). Specific tasks were then selected for inclusion in a test battery in order to measure the six neurocognitive domains commonly impaired in schizophrenia; this battery is known as the Measurement and Treatment Research to Improve Cognition in Schizophrenia (MATRICS) consensus cognitive battery (Green, et al. 2004). A seventh domain, social cognition, was also included in the battery due to increased interest in the field (Nuechterlein et al., 2004); associated measures were not included in the factor-analytic studies reviewed, as social cognition was a fairly new area of research in schizophrenia.
at the time. Furthermore, standardisation of social cognitive measures across different clinical and non-clinical groups had not yet been accomplished.

The MATRICS battery comprises ten individual tests each measuring one of the seven cognitive domains noted above. Selection of tests was based on good psychometric properties (e.g. test-retest reliability) in order to validly measure changes in cognition across time within clinical trials or referentially in non-intervention studies (Green et al., 2004). Given that the MATRICS battery is a relatively new standard in the assessment of cognition in schizophrenia, it has not yet been extensively used for research purposes. Nonetheless, methodological inconsistencies and use of non-standardised measures across studies may decrease with continued administration of the MATRICS, or other similar, cognitive test batteries in schizophrenia research.

It is important to note that neurocognitive dysfunction appears to predict social and functional impairment, even more so than clinical symptoms (Green, 1996). Specific neurocognitive deficits including verbal memory, working memory, attention/vigilance and executive functioning have been found to predict functional outcome in the areas of basic life skills (e.g. conversation and leisure skills), social problem solving, employment, and independent living (for reviews see Green, 1996; Green, Kern, Braff, & Mintz, 2000). However, the relationship between neurocognition and poor social functioning has only been moderate (Green, 1996; Green, et al., 2000), which has led to recent suggestions that other more specific factors, such as impaired social cognition, may also play a role in the development and maintenance of schizophrenia. The next section provides an overview of social cognition, including definitions of representative domains and a review of the literature in this field.
2.2 Social Cognition in Early Psychosis and Schizophrenia

Social cognition is a complex paradigm that comprises several distinct processes. Many definitions of the term exist, from Fiske’s (1995, p.151) basic conceptualisation that social cognition is "simply thinking about people", to broader descriptions such as “a domain of cognition that involves perception, interpretation, and processing of social information” (Ostrom, 1984, p.176). Both of these definitions are valid as a variety of theoretical constructs are thought to constitute social cognition, however Ostrom’s (1984) depiction reflects a more comprehensive view that implies a failure to correctly perceive social cues and information may produce misinterpretations and inaccurate responses in social settings, thus adversely affecting one’s social functioning and interactions.

Despite developments in the area of social cognition over the past decade and a half, progress has been limited due to inconsistencies in terminology and the way in which it has been assessed. In an attempt to reduce the variability regarding how it is defined in the literature, investigators affiliated with the National Institute of Mental Health (NIMH) convened to reach a consensus on the domains that comprise social cognition (Green et al., 2008). It was concluded that social cognition covers at least five different domains, namely: emotion processing, social perception, theory of mind, social knowledge, and attributional style.

It is important to investigate social cognition in the early course of illness, as potential intervention strategies are likely to produce better outcomes if applied before the development of a full-blown disorder. Studies that have investigated social cognition in the early course of illness, specifically FEP, have generally found deficits in various
social cognitive domains (e.g. Addington, Saedi & Addington, 2006; Bertrand, Sutton, Achim, Malla & Lepage, 2007; Edwards, Pattison, Jackson, & Wales, 2001; Inoue et al., 2006; Krstev, Jackson & Maude, 1999), however the nature and extent of such impairments remains unclear due to limited research with early psychosis populations compared to those with an established illness, and a lack of longitudinal studies. Based on the paper by Green et al. (2008), the following sub-sections include separate descriptions of five social cognitive domains that have been commonly assessed in schizophrenia, with reviews of the literature.

2.2.1 Emotion recognition

Emotion recognition is one of the most extensively studied domains of social cognition. It refers to one’s ability to identify emotions via processing of nonverbal cues, for example, through facial expression or tone of voice. The Diagnostic Analysis of Nonverbal Accuracy-2 (DANVA-2) scale for faces and voices (Nowicki & Carton, 1993; Nowicki & Duke, 1994) is an example of an assessment tool that measures emotion recognition through both facial affect and voice prosody. Specifically, there are two components to the task; one that involves the presentation of colour photographs of faces via computer, and another that consists of audio-recordings of two professional actors who say a neutral phrase in an emotional tone of voice. The task is easy to administer as it involves forced-choice responses, with respondents required to indicate whether they think the stimulus is depicting happiness, sadness, anger, or fear. Kerr and Neale’s (1993) Face Emotion Identification Test (FEIT) and Voice Emotion Identification Test (VEIT) are also similar in principle to the DANVA-2, and have also been commonly applied in schizophrenia research.
A variety of other emotion recognition tasks have also been used in empirical studies, which highlights the issue of methodological inconsistencies in the measurement of social cognition in schizophrenia. Furthermore, the majority of studies have only assessed emotion/affect recognition by using pictures of faces as stimuli (e.g. Aghevli, Blanchard & Horan, 2003; Comparelli et al., 2013; Edwards et al., 2001; Kohler, Bilker, Hagendoorn, Gur, & Gur, 2000; Mueser, Penn, Blanchard, & Bellack, 1997), without focusing on other nonverbal indications. Regardless of the assessment measure used, impairments in emotion recognition, particularly in facial affect recognition, have been consistently reported.

Although most studies have assessed chronic schizophrenia samples, a recent study by Comparelli et al. (2013) included three schizophrenia groups each reflecting a different stage of illness (ultra high-risk, first-episode, and multi-episode) and a control group, as to ascertain whether emotion recognition deficits are stable over the course of illness. They assessed six basic emotions (fear, sadness, anger, disgust, happiness and surprise) through a novel facial affect recognition task and found impaired ability in the identification of all negative emotions for both the first-episode and multi-episode group, with the prodromal group impaired only on sadness and disgust. A limitation to the study was the lack of a prosodic emotion recognition task, however another similar study that investigated ultra high-risk and first-episode psychosis patients found significant impairments in facial and prosodic emotion recognition in both clinical groups compared to controls (Amminger et al., 2012). Results from both of these studies indicate that emotion recognition deficits are present in the early phase of illness before the onset of full-blown psychosis, which provides support for a neurodevelopmental model of schizophrenia. Furthermore, no significant differences in impairment were
found between the clinical groups in either of the studies, suggesting emotion recognition is a stable deficit feature over the course of illness, and thus appears to be a marker of schizophrenia rather than an indicator of illness severity.

2.2.2 Emotion/social perception

Emotion or social perception refers to the ability to recognise and interpret social cues, such as gestures and affect, from other people's behaviour in a social environment (Green, Olivier, Crawley, Penn, & Silverstein, 2005). Reviews of social cognition in schizophrenia have classified social and emotion perception both within the same domain (e.g. Yager & Ehmann, 2006) and separately (e.g. Couture, Penn, & Roberts, 2006). The main distinction between the two is that judgements of social and emotion cues vary depending on the nature of the cue; emotion perception involves the recognition of emotions from a person's facial expressions or tone of voice in order to draw conclusions about mood and affect, whereas social perception involves not only cues relating to emotion but a broader range of social cues in one's environment. Moreover, measures of emotion perception concentrate only on mood state, while social perception tasks can assess additional areas such as social roles and goals (Green et al., 2005) and require individuals to use contextual information to infer events that led to particular behaviours (Penn, Ritchie, Francis, Combs & Martin, 2002; Sergi & Green, 2002; Trope, 1986).

The domain of emotion perception has been interchanged with the term “emotion processing” in the literature, however it is important to draw a distinction here based on conceptual issues. According to Salovey and Sluyter (1997), emotion processing is an extensive concept that comprises various neuropsychological functions, that is, it is not
necessarily reflective of one particular domain of social cognition. Salovey and Sluyter (1997) conceived a four-facet model of emotion processing, comprising the factors: ‘identifying emotions’, ‘facilitating emotions’, ‘understanding emotions’, and ‘managing emotions’. Identifying emotions is considered to be the most basic of the four model components, whereas the other three facets are thought to require more complex levels of processing. According to Salovey and Sluyter (1997), facilitating emotions involves the evaluation of the meaning and utility of emotions in order to effectively execute a particular behaviour, understanding emotions reflects the ability to comprehend changes in emotion, and managing emotions refers to one’s experience of emotions and the way they are expressed in response to a situation.

Conceptually and practically, it seems ‘emotion recognition’ (described and presented above as a particular domain of social cognition), is comparable to the ‘identifying emotions’ facet of the model described by Salovey and Sluyter (1997). As emotion perception is defined rather ambiguously in the social cognition literature and is a relatively broad concept in itself, the term could be considered to reflect the other more complex processes depicted in the four-factor model, i.e. facilitating emotions, understanding emotions, and managing emotions. As such, the measure of social cognition included in the MATRICS Consensus Cognitive Battery (i.e. the Managing Emotions subtest of the MSCEIT; Mayer, Salovey, & Caruso, 2002) could be referred to more specifically as a measure of emotion perception, rather than one of overall emotion processing. This would eliminate some of the ambiguity in the literature regarding how emotion perception/emotion recognition/emotion processing is depicted and subsequently measured in research studies.
For the purposes of this thesis, emotion perception and social perception are classified together, as they both involve processing of social cues and situational factors, broadly speaking. Schizophrenia patients have been shown to display impairments in the accurate perception of social cues compared to non-clinical populations and control groups, especially those in abstract form (for a review see Leonhard and Corrigan, 2001). Individuals with schizophrenia are more inclined to think concretely given extensive cognitive deficits that likely affect higher order processes (Corrigan, 1997). Concrete interpretations are exemplified by the fact that those with the illness seem to concentrate on what people say or do, rather than recognise other important social cues such as one’s emotional tone or affect (Trope, 1986). For example, the observation of someone crying may be construed as sadness if the person has just said goodbye to a loved one that they would not see for some time, or happiness if the behaviour was elicited in response to the loved one returning home to visit (Trope, 1986). This suggests that a tendency to think concretely affects the ability of patients with psychosis to processes contextual information.

Kring and Campellone (2012) have recently critiqued the emotion perception literature in schizophrenia and highlighted that the majority of the research in this domain has focused primarily on the perception of static faces displaying various emotions or expressions of affect. This is a major shortcoming in the literature as such perceptions are rather basic forms of emotion processing (i.e., as mentioned earlier, analogous to ‘emotion recognition’), and that emotion perception involves more than just facial or prosodic processing. In their review, Kring and Campellone (2012) emphasise that the role of context is crucial to emotion perception, and that contextual information actually facilitates judgment about emotions. Studies that have addressed the influence of
context on social/emotion perception have demonstrated that people with schizophrenia and schizoaffective disorder display various deficits in context processing (e.g., Cohen, Barch, Carter, & Servan-Schreiber, 1999; Penn et al., 2002). Furthermore, schizophrenia patients appear to lack the ability to readjust initial interpretations when contextual information becomes available following an initial response (Couture et al., 2006).

Only recently, however, have studies begun to examine how context impacts on emotion perception. Green and colleagues devised a task to explicitly assess how situational context impacts on emotion perception in schizophrenia patients. In this task, (the Social Context Appreciation Task; SCAT; unpublished) pictures of people depicting different emotions (both with and without contextual information) are shown to individuals. Initially, a picture of expressed emotion is presented on a clear backdrop without contextual information, which is then followed by presentation of the same picture although contextual information is included in the background. For example, a picture of a young boy crying is first presented without any contextual information, and then it is shown again in the context of receiving an autograph from a baseball player (Kring & Campellone, 2012). In a study that used the SCAT, it was found that people with schizophrenia tended to spend less time attending to contextual information compared to control subjects, and were therefore less accurate in understanding the emotional state of the target stimulus (Green, Waldron, Simpson, & Coltheart, 2008). Thus, deficits in other non-social cognitive abilities that are typically involved in attending to stimuli (e.g., attention/concentration or speed of processing), may impact on context processing in those with schizophrenia and this can have consequences in daily social functioning. In fact, support for this conjecture has recently been provided
by Chung and colleagues (2011), who found a positive correlation between measures of context processing (using a test of social inference) and attention, which was stronger in a schizophrenia sample compared to healthy controls.

2.2.3 Theory of mind

Theory of mind (ToM) is a social cognitive construct initially described by Premack and Woodruff (1978), involving the ability to acknowledge that other people act according to their own beliefs, thoughts and intentions, and these mental states may be different to one's own (Corcoran, 2001). Such “mentalising” allows inferences to be made about the intentions or behaviours of others; being able to sense that someone is lying or the interpretation of body language is only possible if the concept of theory of mind exists. The extent to which people can mentalise is thought to vary in the normal population (Corcoran, 2001), with most people displaying generally accurate and functional theories of mind however others can lack humour, be overly literal, or perceive cues incorrectly.

Strong empirical evidence supports the notion that autistic individuals and those with Asperger’s syndrome are most affected by ToM deficits (for a review see Baron-Cohen, 1995), with ‘first order’ and ‘second order’ tasks both impaired. A first order task typically involves acknowledgement of another person’s thoughts about an external event, whereas second order tasks may require higher order cognitive abilities such as understanding irony or metaphor. Individuals with schizophrenia also display deficits in ToM skills (for a review see Brüne, 2005a), however impairments are less severe than those seen in autism as schizophrenia patients can generally complete first order tasks.
(Doody, Götz, Johnstone & Frith, & Cunningham Owens, 1998; Frith & Corcoran, 1996; Pickup & Frith, 2001). Furthermore, mentalising abilities are more variable in individuals with schizophrenia compared to those with autism as patients with delusions still have the capacity to acknowledge that beliefs can be false (Langdon, Coltheart, Ward & Catts, 2001).

Despite differences in the nature and severity of ToM deficits between autistic individuals and those with a psychotic illness, a substantial body of evidence now exists to support the view that ToM impairment is a feature of schizophrenia. ToM deficits have been found across different phases of psychotic illness, for example, in chronic patients (Brüne, 2005a, Sprong, Schothorst, Vos, Hox, & van Engeland, 2007), first-episode psychosis patients (Bertrand et al., 2007), and in remitted patients (Inoue et al., 2006; Sprong et al., 2007). This has raised the question as to whether ToM deficits are trait rather than state phenomena in schizophrenia-spectrum disorders. Furthermore, it is noted that most studies of ToM impairment have been conducted using chronic or multi-episode patients and have been cross-sectional in design, which has also created difficulty in determining whether ToM impairment is in fact a trait feature of the illness.

To address the issue of whether ToM represents a state or trait deficit, two meta-analyses (Bora, Yücel, & Pantelis, 2009; Sprong et al., 2007) provide some elucidation. Bora et al. (2009) reported continuous data on ToM performance from 36 studies of male schizophrenia patients and male control subjects. The results of the review indicated that whilst task specific differences and other state variables (e.g. acute versus non-acute phase) explained a large proportion of the variation in ToM findings across studies, the fact that remitted patients continued to display ToM deficits suggests poor
mentalising ability is more likely a trait characteristic in schizophrenia (Bora et al., 2009). Similarly, Sprong et al. (2007) reported a large overall effect size ($d = 1.25$) regarding ToM impairment across 29 studies, with a weaker level of impairment ($d = 0.69$) in remitted patients. Nonetheless, the effect remained significant in the remitted patients, which indicates that although acute psychosis may impact on ToM performance, mentalising deficits still appear to be trait phenomena in schizophrenia.

Although ToM has been assessed using a variety of measures, two assessment tools that have been more commonly used in schizophrenia research are the “Hinting Task” (Corcoran, Mercer, & Frith, 1995) and “Visual Jokes” task (Corcoran, Cahill, & Frith, 1997), both of which are second order tasks. The measures were developed and validated for use in adult clinical populations by Corcoran and others (1995; 1997), with the Hinting Task having since been widely applied in psychiatric populations (e.g. Bertrand et al., 2007; Bora et al, 2005), as it is quick and easy to administer and score. The Hinting Task involves 10 short passages read out loud by an examiner one at a time, with the respondent required to convey the intended meaning of a character’s statement, which may include metaphor or irony. The Visual Jokes task involves the interpretation of humour within cartoon illustrations, in order to determine a person’s ‘mentalising’ ability, i.e. if they understood the emotional state of the character(s) in the cartoon. Although a general scoring protocol exists for the Visual Jokes task (see Corcoran et al., 1997), it is much more difficult to score than the Hinting Task as interpretations of the cartoons can be highly variable between respondents, thus the mentalising score (on a scale of 0-3 for each cartoon) contains some level of subjective evaluation.
2.2.4 Social knowledge

Social knowledge refers to one’s understanding of the roles, rules and goals that characterise social situations and guide social interactions (Green et al., 2008). It has also been discussed as ‘social schema’, with the term schema referring to the cognitive representation of social information (Corrigan & Addis, 1995). As such, social knowledge is considered a specific type of social cognition as it encompasses one’s ability to comprehend situational stimuli (Corrigan & Addis, 1995; Green et al., 2008), which has implications for social skills and interpersonal effectiveness. Corrigan and Addis (1995) designed a task to specifically assess social knowledge, called the Schema Component Sequencing Task – Revised (SCST-R), which involves sequencing of particular actions in order to accurately portray a given social situation. This task was based on the notion that temporal information regarding a social scenario is necessary for correct inferences and predictions about the actions of others as individuals engage in situations (Galambos, 1986). To illustrate, when considering the example of eating in a restaurant, if someone has already “been shown to their table by the waiter” and “has eaten their main meal”, the assumption should be that the person has already ordered and the prediction should be that they will pay their bill (Corrigan & Addis, 1995).

The SCST-R comprises a total of 12 social scenarios, half with five component actions (‘short’) and the other half with nine component actions (‘long’). Both short and long sequences were included to assess whether the latter (i.e. when more information is involved in the form of an increased number of component actions) further hinders a patient due to higher cognitive load and increased task complexity (Corrigan & Addis, 1995). For each of the 12 situations, actions were displayed in writing on 5 by 8 inch
cards in a random order; the essential requirement of the task was to correctly sequence the cards in the correct order. The authors found that schizophrenia patients were significantly less able to temporally arrange the component actions of social situations than healthy controls, particularly for the long conditions. The findings implied that not only are schizophrenia patients impaired in social knowledge, but they also seem to experience increased difficulty with social cognitive tasks that are relatively more complex (Corrigan & Addis, 1995).

Corrigan and colleagues also developed two other tasks assessing social knowledge in schizophrenia: the Social Cue Recognition Task (SCRT; Corrigan & Green, 1993a), and its revised edition (SCRT-R; Corrigan et al., 1996), and the Situational Feature Recognition Test (SFRT; Corrigan & Green, 1993b). The latter is a paper-and-pencil task that requires individuals to demonstrate an awareness of social information (i.e. actions, roles, rules, and goals) that correspond to particular social situations (Corrigan & Green, 1993b), whereas the former tasks involve watching videotaped vignettes of social interactions in order to elicit awareness of concrete versus abstract social cues. These two tasks, in addition to the SCST-R, have been amongst the most commonly used assessment measures of social knowledge in schizophrenia research (Couture et al., 2006).

The overall literature to date demonstrates that social knowledge has been studied considerably less than other areas of social cognition in schizophrenia. A reason for this is likely due to the fact that, despite having been identified as a particular area of social
cognition (Green et al., 2008), it seems to overlap with the domain of social perception on a conceptual level. Specifically, successful social knowledge requires an awareness of the characteristic social cues that occur in particular social situations, which encompasses the perception and processing of social information (i.e. ‘social perception’). Subsequently, the two have been grouped together as representative of the same construct in certain reviews and meta-analyses of the social cognition literature (e.g. Couture et al., 2006; Fett, Viechtbauer, Dominguez, Penn, van Os, & Krabbendam, 2011). For the purposes of this thesis, and in accordance with descriptions of the main social cognitive domains in Green et al. (2008), social knowledge and social perception are presented and analysed separately.

2.2.5 Attributional style

Attributional style refers to the causal factors people attribute to positive and negative events (i.e. “attributions”). Three main types of attributions have been identified: i) external personal attributions, i.e. causes attributed to other people; ii) external situational attributions, i.e. causes attributed to situational factors; and iii) internal attributions, i.e. causes due to oneself (Green et al., 2008). Attributional style is most commonly measured by questionnaires, with the Attributional Style Questionnaire (ASQ; Peterson et al., 1982) and Internal Personal Situational Attributions Questionnaire (IPSAQ; Kinderman & Bentall, 1996), widely used to ascertain whether specific samples display a bias toward attributing outcomes to themselves, other people, or situational factors. The IPSAQ is considered to be one of the most valid and reliable measures of attributional style available (Martin & Penn, 2002).
In research involving both psychiatric and non-clinical samples, key distinctions in attributional style have been typically identified. When considering schizophrenia samples, individuals with persecutory delusions tend to display an exaggerated level of external personal attributions, thus mostly attributing negative outcomes to others rather than situations or themselves (Candido & Romney, 1990; Kaney & Bentall, 1989; Kinderman, Kaney, Morley, Bentall, 1992; Lyon, Kaney & Bentall, 1994; Martin & Penn, 2002). This propensity is known as a ‘personalising’ or self-serving bias (Bentall, Corcoran, Howard, Blackwood, & Kinderman, 2001), which has been theorised to underlie persecutory delusions (Bentall, Kinderman & Kaney, 1994). Specifically, it has been hypothesised that individuals with persecutory delusions are likely to have an underlying negative self-concept as a result of adverse experiences (e.g. real or perceived negative evaluations by others), thus persecutory delusions can develop as a means of self-preservation (Martin & Penn, 2002). That is, by attributing negative events to others and positive outcomes to oneself, the likelihood of self-blame is diminished and self-esteem is maintained (Kinderman et al., 1992).

2.3 General Limitations and Considerations in Social Cognition Research

One of the main challenges of social cognition research in psychosis and schizophrenia lies in the fact that the construct is broad and includes a multitude of psychological processes (Green et al., 2008; Penn, Corrigan, Bentall, Racenstein, & Newman, 1997). It has been suggested that social cognition comprises at least five different domains (Green et al., 2008), however given the variability in the literature regarding the classification of such domains, it has been unclear as to whether each identified area represents a distinct process. For example, some researchers have referred to emotion
perception and social perception as the same domain (e.g., Yager & Ehmann, 2006),
whereas others have grouped social perception and social knowledge together (e.g. Fett
et al, 2011). Moreover, the area of emotion perception is ambiguous in itself, with
investigators having included emotion recognition within this domain (e.g. Fett et al,
2011) and others drawing a distinction between the two given that emotion perception
encompasses contextual information rather than just facial affect recognition (e.g. Kring
& Campellone, 2012).

Progress in social cognition research has further been hampered by the use of a wide
range of assessment tools across studies, which introduces problems in terms of
comparing results and generalising findings. This highlights the need for standardised
measures of social cognition that tap into specific domains, in order for a universal
approach to be utilised amongst future studies in the assessment of social cognition.
The MATRICS Consensus Cognitive Battery (referred to earlier this Chapter) was
developed to provide a gold standard in the assessment of cognition in schizophrenia,
including social cognition, however it primarily assesses neurocognitive abilities and
only includes one task related to social cognition (i.e., the Managing Emotions subtest of
the MSCEIT; Mayer et al., 2002). This task was purported to measure the overall
construct of social cognition, however given the conceptual issues pertaining to the
construct, the MSCEIT task has been classified as a measure of emotion perception per
se in this thesis. Thus, the MSCEIT task used in the MATRICS is a measure of a particular
aspect of social cognition and does not provide information on various other social
cognitive processes (e.g., ToM, emotion recognition, social knowledge, etc.) that may be
impaired in FEP.
Before specific test batteries can be developed for comprehensive measurement of social cognition in schizophrenia and early psychosis, clarity is required regarding the classification of domains. In addition, more research is necessary to elucidate the particular social cognitive processes that are impaired in the early and later phases of schizophrenia. Penn and Corrigan (2001) suggested that studies should incorporate a variety of social cognitive measures into their study designs instead of assessing only one particular aspect of social cognition such as affect recognition (e.g. Addington et al., 2006) or ToM (e.g. Bora et al., 2005), in order to assist in determining whether social cognition embodies a generalised deficit within a particular clinical sample or if only specific functions are impaired. Bertrand et al. (2007) has provided some empirical evidence for the former in a sample of FEP patients who, compared to controls, displayed impaired performance on a variety of social cognitive tasks including the Hinting Task (relating to ToM) and the Four Factor Test of Social Intelligence, which assesses various other social cognitive domains such as affect recognition and social perception.

2.4 Neurocognition and Social Cognition: Distinct Constructs?

It has been suggested that social cognition and neurocognition are not representative of the same construct, given differences in the nature of the information that is processed. The former refers to perception and processing of social information such as verbal and non-verbal social cues (Fiske, 1995), whereas the latter involves non-social stimuli including words, numbers and objects that are affectively neutral (Corrigan & Toomey, 1995; Penn, et al., 1997). Furthermore, social cognition and neurocognition have been conceptualised differently regarding how that information is processed. Social cognition
has been described as involving “bidirectional” processing, that is, an individual responds to a social stimulus, which can also interact with the person (Fiske, 1995). Conversely, neurocognition represents unidirectional processing of inanimate objects that do not have any personal significance to the person (Fiske, 1995).

Even though the literature highlights the importance of social cognition in terms of underlying the effectiveness of social behaviour and functioning (Brüne, Abdel-Hamid, Lehmkämper, & Sonntag, 2007; Couture et al., 2006; Roncone et al., 2002), specific neurocognitive processes have also been shown to contribute to the execution of such behaviours (Green et al., 2000; Green, Kern, & Heaton, 2004; Milev, Ho, Arndt, & Andreasen, 2005). Using a practical example, engaging in a conversation not only requires the processing of social information contained in verbal speech and non-verbal cues, but also neurocognitive functions such as attention (to concentrate and comprehend what the other person is saying) and working memory (to briefly store incoming information whilst other available social cues are processed in order to generate an appropriate response) (Penn, et al., 1997).

The actual relationship between social cognition and neurocognition in schizophrenia has received some attention in the literature, for example, social cue perception (Corrigan, Green & Toomey, 1994) and emotion recognition (Kee, Kern & Green, 1998) have been significantly associated with early visual processing. Such correlations suggest that these particular functions, regardless of their cognitive nature, may reflect some common neural activity (Pinkham, Penn, Perkins, & Lieberman, 2003). Indeed, visual processing and emotion recognition are considered to be more basic than other cognitive skills (Brothers, 1990), thus it may be that lower-order cognitive functions
may share some of the same neural mechanisms. Furthermore, social perception has been identified as a mediator between visual processing and functional status in schizophrenia (Sergi, Rassoisky, Nuechterlein, & Green, 2006), which indicates that neural networks link certain neurocognitive and social cognitive processes, and that these connections may partially explain the overlap between the constructs. Comparably, significant associations have been demonstrated between social cognitive and neurocognitive abilities requiring higher-order processes, for example, ToM and executive functioning (Kinderman, Dunbar, & Bentall, 1998).

Despite evidence of some degree of overlap between particular social cognitive and neurocognitive functions (Corrigan et al., 1994; Fanning, Bell, & Fiszdon., 2012; Kee et al., 1998; Kinderman et al., 1998; Sergi et al., 2007; Ventura, Wood, & Helleman, 2011), it is still considered that the domains are relatively independent of one another (Fanning et al., 2012; Pinkham, Penn, Perkins & Lieberman, 2003; Sergi et al., 2007). Some studies have provided support for the view of independent constructs by indicating that social information processing and neurocognitive skills are regulated by semi-independent neural systems (Bozikas, Kosmidis, Anezoulaki, Giannakou, & Karavatos, 2004; Lee, Farrow, Spence & Woodruff, 2004; Pinkham et al., 2003).

Moreover, a study that utilised structural equation modelling to investigate the associations between the two overall constructs in a sample of schizophrenia/schizoaffective disorder outpatients found that a two-factor model that represented social cognition and neurocognition as separate constructs fit the data significantly better than a one-factor model, despite relatedness between the domains (Sergi et al., 2007). In that study, social cognition was represented by the domains of
emotion recognition and social perception, and a variety of neurocognitive variables were also included (e.g. attention, verbal working and secondary memory, fluency, processing speed, executive functioning). The relative independence of other domains of social cognition such as ToM remains elusive, therefore future studies should aim to examine the relationship between various social cognitive functions and the neurocognitive abilities already known to be impaired in psychosis. This would provide a more comprehensive understanding of the degree of associations between social cognition and neurocognition.
CHAPTER 3: The Relationship Between Symptomatology and Cognitive Variables in Schizophrenia

3.1 Psychotic Symptoms: A Three-Factor Model

The clinical features of schizophrenia have historically been categorised as either ‘negative’ or ‘positive’ symptoms (Crow, 1982). Negative symptoms reflect a loss or absence in functioning and include features such as avolition (lack of motivation and withdrawal from one’s environment), anhedonia (lack of interest or pleasure in social and recreational activities), alogia (amount of speech is reduced and may be elusive or repetitive), and flat or blunted affect (unchanging facial expression, poor eye contact and reduced spontaneous body movement) (Sims, 2002). Positive symptoms signify an increase or distortion of normal thinking and include the phenomena of delusions, hallucinations and disorganised speech; the clinical features perhaps most commonly associated with psychosis and those that are required for a diagnosis of schizophrenia (APA, 2013).

Delusions and hallucinations are regarded as the typical positive symptoms of schizophrenia (Crow, 1982). Delusions are beliefs of an improbable or unrealistic nature and are usually held with unusual conviction regardless of lack of proof or contradictory evidence. Delusional themes expressed by individuals with schizophrenia commonly include paranoia and persecution, religion (God or the Devil), grandiosity, somatic concern or ideas of reference (e.g. messages from the television or radio). Hallucinations can occur in visual, olfactory, gustatory, tactile, or auditory form, the latter being the most common. Auditory hallucinations are usually experienced as
hearing voices, which are perceived as different from the person's own thoughts or inner speech (Katona & Robertson, 2000).

Disorganised speech is also widely regarded as positive symptomatology (Andreasen & Olsen, 1982; APA, 2013) however a third symptom group, namely 'disorganised', has been described in the literature (e.g. Liddle, 1987) that includes such phenomena. Specifically, disorganised symptomatology can refer to confused or unclear speech, rapid or nonsensical thoughts, and disordered behaviour (Liddle, 1987). A number of factor-analytic studies have demonstrated that the three-factor model, which includes disorganisation, is a better representation of schizophrenia-spectrum symptomatology than the positive-negative paradigm (Bilder, Mukherjee, Rieder & Pandurangi, 1985; Drake et al., 2003; Emsley, Rabinowitz, & Torreman, 2003; Liddle, 1987). Furthermore, the three-factor model has been widely adopted in research studies to examine the relationship between specific symptoms and cognitive deficits (e.g. Brüne et al., 2007; Cameron et al., 2002; Smith, Hull, Huppert & Silverstein, 2002).

3.2 Neurobiology of Symptoms

From a neurobiological perspective, it has been thought for some time that increased levels of dopamine underlie psychotic symptoms. This idea largely arose from the unintentional discovery that psychotic symptoms could be reduced with a particular class of dopamine-blockers (Randrup & Munkvad, 1965). In addition, dopamine-increasing drugs such as amphetamines can produce psychotic symptoms, or exacerbate symptoms in individuals who already have schizophrenia (Laruelle et al., 1996). Hence the first line of treatment in mental health settings for individuals with
such symptoms is currently antipsychotic medication that primarily acts to reduce dopaminergic activity.

Hallucinogens and stimulant drugs, which affect neurotransmitters such as serotonin and glutamate, can trigger psychotic symptoms (Mueser, Yarnold & Levinson et al., 1990). Drugs that block glutamate such as ketamine can also produce the symptoms and cognitive dysfunction that are seen in schizophrenia (Lahti, Weiler, Michaelidis, Parwani & Tamminga, 2001). Furthermore, abnormally low levels of glutamate receptors have been found in post-mortem brains of individuals who had been diagnosed with schizophrenia (Konradi & Heikers, 2003), suggesting that various neurobiological systems may play a role in the complex disorder.

Even though antipsychotics are generally effective in treating positive symptoms, some individuals do not respond to such medication. This implies that other, perhaps non-neurochemical, factors may be involved in the aetiology of the disorder. Furthermore, the treatment effect of antipsychotics on negative symptoms is not nearly as profound as it is on positive symptoms, which also suggests that neural mechanisms may differ according to symptom subtype. Impairment in social cognition has also been proposed as a possible causal factor in the development of schizophrenia symptoms (Frith & Corcoran, 1996), however further research is required to address this hypothesis.
3.3 The Relationship Between Symptomatology and Neurocognition in Schizophrenia

There is a body of literature that demonstrates a definite correlation between neurocognitive impairment and psychotic symptoms in schizophrenia-spectrum disorders. Specifically, the overall empirical findings have indicated that negative symptoms are typically associated with a number of neurocognitive domains, including verbal fluency, memory and executive functioning (Liddle & Morris, 1991; Norman et al., 1997; Woodward, Ruff, Thornton, Moritz, & Liddle, 2003; Woodward, Thornton, Ruff, Moritz, & Liddle, 2004). Furthermore, neurocognitive deficits have been more strongly associated with negative symptoms than positive symptoms (e.g. Heslegrave, Awad, & Voruganti, 1997; Savilla, Kettler, & Galletly, 2008). For example, the results of the study conducted by Savilla et al. (2008) revealed a moderate association between negative symptoms and neurocognition, but the correlation between positive symptoms and neurocognition was smaller and not statistically significant.

In fact, positive symptoms have not been commonly related to neurocognition. Even though there has been some evidence of an association, for example, a meta-analytic review revealed a correlation between positive symptoms and executive functioning (Johnson-Selfridge & Zalewski, 2001), many studies have failed to show an association (e.g., Liddle, 1987; Norman, 1997). It has also been suggested that individuals with paranoid delusions may have better neurocognitive abilities than those with a non-paranoid subtype of the illness (Seidman, 1983), however a review of 32 studies comparing neurocognitive functioning between paranoid and non-paranoid
schizophrenia groups provided mixed results (Zalewski, Johnson-Selfridge, Ohriner, Zarrella & Seltzer, 1998).

In terms of disorganised symptomatology, the nature of the relationship with neurocognition is similar to that of negative symptoms. Characteristic disorganised symptoms such as formal thought disorder have been linked to deficits in attention (Harvey et al., 1998; Silverstein et al., 1991), memory (Harvey et al., 1998; Nestor et al., 1998) and executive functions (Barrera, McKenna, & Berrios, 2005; Nestor et al., 1998). Thus, it appears that disorganised thinking and negative symptoms are more consistently linked to deficits in neurocognition, and relating to similar cognitive domains, than the more typical positive symptoms.

3.4 The Relationship Between Symptomatology and Social Cognition in Schizophrenia

In terms of the relationship between psychotic symptoms and social cognition, the picture is less clear, especially in the early stage of illness given the relative paucity of research with FEP populations in this field. The literature has provided mixed findings and it is important to establish the nature of the relationship between psychopathology and the various facets of social cognition, given that social cognitive deficits have been implicated in the development and maintenance of psychotic symptoms (Couture et al., 2006; Frith & Corcoran, 1996).

3.4.1 Negative symptoms and social cognition

The existing literature provides some evidence for a relationship between negative symptoms and social cognitive deficits, however results have been inconsistent. Support
has been shown for an association between negative symptoms and various social
cognitive domains, for example, emotion recognition (Johnston et al., 2010), social
perception (Corrigan et al., 1994; Sergi et al., 2007) and ToM (Bora, Gokcen, Kayahan, &
Veznedaroglu, 2008; Corcoran et al., 1995; Shean & Meyer, 2009; Stratta et al., 2011),
whereas other studies have failed to demonstrate any relationship (Bertrand et al.,
2007; Mancuso, Horan, Kern, & Green, 2011; Toomey, Schuldberg, Corrigan & Green,
2002). Furthermore, the findings of some studies showing support for a relationship
between negative symptoms and the specific domain of ToM are interpreted with
cautions; one study was based on a very small sample size (Corcoran, et al., 1995), and
another study included individuals with non-paranoid positive symptoms in the
negative symptom subgroup (Pickup & Frith, 2001).

A distinction has been made in the literature between cognitive and affective empathy,
whereby the former refers to the ability to understand another person’s viewpoint and
the latter involves inferences specifically about the emotional experiences of others and
the capacity to share those feelings (Mehrabian & Epstein, 1972). Empathic abilities are
reflective of ToM processes (Montag, Heinz, Kunz & Galliant, 2007; Shamay-Tsoory,
Harari & Levkovitz, 2007) and recent evidence suggests that both cognitive and
affective empathy are impaired in schizophrenia and that the degree of dysfunction is
associated with severity of negative symptoms (Shamay-Tsoory et al., 2007).

3.4.2 Positive symptoms and social cognition

As delusions reflect false beliefs about the intentions and actions of others, it has been
hypothesised that impairments in ToM (i.e. the inability to acknowledge the mental
states of others) may contribute to such symptoms (Frith & Corcoran, 1996). The
findings relating to this assumption have been equivocal; some studies have provided evidence for the theory (Corcoran et al., 1995; Frith & Corcoran, 1996; Pickup & Frith, 2001), however a number of other studies have not found a relationship between ToM and paranoid symptoms (Drury, Robinson & Birchwood, 1998; Greig, Bryson & Bell, 2004; Langdon et al., 1997; Safari & Hardy-Baylé, 1999). The studies that did demonstrate an association should be considered in light of statistical shortcomings, for example, Pickup and Frith’s (2001) study provided only a weak relationship with a small effect size regarding the paranoid symptoms subgroup, further confounded by a small sample size. Positive symptoms have also been linked to impaired ability on a facial emotion recognition task (Hall et al., 2004) however the sample size was small in this study, as was the effect size.

Extensive research has been conducted with individuals possessing paranoid or persecutory delusions in terms of how they explain positive and negative social outcomes, known as attribution theory. As mentioned in Chapter 2, it has been suggested that an exaggerated self-serving bias (inflated internal attributions for positive events and external attributions for negative events) may underlie persecutory delusions (Bentall et al., 2001), with many studies having shown evidence of an association between this type of attributional bias and paranoid delusions (Candido & Romney, 1990; Fornells-Ambojo & Garety, 2009; Kaney & Bentall, 1989; Kinderman et al., 1992; Lyon et al., 1994; Martin & Penn, 2002). However, it is noted that some studies supporting this theory have been confounded by methodological concerns including a lack of clear diagnostic criteria when comparing groups. Martin and Penn’s (2002) results suggested that individuals without persecutory delusions may possess similar attributional biases to those with persecutory delusions, hence attributional style may
be common amongst individuals with delusions of varying content and requires further clarification.

3.4.3 Disorganised symptoms and social cognition

Most of the research examining the association between psychotic symptoms and social cognition has focused on the positive-negative symptom dichotomy, thus there is relatively limited research regarding the relationship between disorganised symptoms and social cognition. In their study of ToM and symptom dimensions, including disorganisation, Frith and Corcoran (1996) concluded that individuals with the disorganised symptom of formal thought disorder demonstrated the most pronounced deficits in ToM ability. A similar finding was reported by Greig and colleagues (2004), who also assessed ToM ability in relation to symptom dimensions and neurocognition; it was found that individuals with disorganised schizophrenia performed worse on ToM tasks than any other subgroup of patients, with verbal memory also significantly affected. Another study demonstrated that the disorganised symptom of conceptual disorganisation as measured by the PANSS (Kay et al., 1989) was significantly associated with social cue perception (Toomey et al., 2002).

In a study of stable outpatients with schizophrenia that investigated the association between social cognition, neurocognition, and two specific features of formal thought disorder (i.e., idiosyncratic and concrete thinking), impairments in both types of cognition were found to be related to disordered thinking (Subotnik et al., 2006). A variety of neurocognitive functions were examined in this study, thus providing
evidence that neurocognition may underlie thought disorder, however only one particular area of social cognition (i.e. social knowledge) was assessed.
CHAPTER 4: Social Functioning in Psychotic Disorders

4.1 Definition of Social Functioning

Analogous to cognitive dysfunction and clinical symptomatology, impaired social functioning is also considered to be a hallmark characteristic of schizophrenia. Social functioning refers to overall day-to-day performance across various social domains, for example, independent living, family and peer relationships, occupational role functioning, and leisure activities (Green, 1996; Harvey & Bellack, 2009). The term “functional outcome” is also used in the literature to denote such socially-driven roles and behaviours (e.g., Green, 1996), however it refers more to functioning with reference to the effects of the illness and/or treatment over time (Bartholomeusz, Killackey, Thompson, & Wood, 2011).

Impairments in social functioning are usually severe in schizophrenia (Harvey & Sharma, 2002), with enormous implications for those with the illness and society in general. Individuals with schizophrenia are often socially withdrawn, which consequently limits the potential to develop interpersonal skills and sustain relationships. Social deficits also predict poor outcome, for example, in relation to relapse and unemployment (Perlick, Stastny, Mattis & Teresi, 1992; Sullivan, Marder, Liberman, Donahoe & Mintz, 1990; Tien & Eaton, 1992). Regarding unemployment, rates are extremely high for people with schizophrenia, with as many as 70-92% of people with the illness not employed in the workforce (Bartholomeusz et al., 2011). For those who are in the workforce, employment is likely to be in a part-time or casual role (McGlashan, 1988). Levels of unemployment are also considerable in FEP; in Australia,
the unemployment rate amongst young individuals with FEP has been estimated at 40-50% (Killackey, Jackson, Gleeson, Hickie, & McGorry, 2006; Marwaha & Johnson, 2004; Schimmelmann et al., 2008), which is substantially higher than that of age-related peers (15-24 years old) in the general community (Robinson & Lamb, 2009).

Such outcomes impact on the public health system and community services, not to mention individuals with the disorder becoming reliant on government financial support. Moreover, dependence on public funding usually occurs early in the illness, with up to 50% of first episode patients receiving disability payments within the first six months of treatment (Ho, Andreasen & Flaum, 1997). Given the serious consequences of functional impairment, there is growing interest in the investigation of potential causal factors, in particular social cognition.

4.2 Factors Affecting Social Functioning in Psychotic Disorders

Poor social functioning in schizophrenia is believed to likely result from a complex interplay between neurocognition, social cognition, clinical features and environmental influences (Yager & Ehmann, 2006) however the extent of contributory factors is not fully understood. Neurocognitive variables including verbal learning, working memory and attention/vigilance (Green et al., 2000; Green et al., 2004; Miley et al., 2005; Smith et al, 2002), and negative and disorganised symptoms (Smith et al., 2002), have been reported as significant predictors of various domains of functioning in schizophrenia. Despite these predictive relationships, a large amount of variance still remains unexplained by neurocognition and symptomatology. As such, there has been recent interest in the investigation of social cognitive factors that may underlie social impairment in schizophrenia. Deficits relating to the social cognitive constructs of social
perception, emotion recognition and ToM have been significantly associated with social
dysfunction in schizophrenia and also early psychosis (Addington et al., 2006; Brüne,
2005b; Brüne et al., 2007; Mueser, et al., 1996; Pinkham & Penn, 2006; Sullivan et al.,
2013), however more research is required to further elucidate the nature of these
relationships. The association between social functioning and neurocognition, social
cognition and symptomatology are discussed in more detail below.

4.2.1 Neurocognition and social functioning

Several studies have suggested that neurocognitive variables are associated with
functional status (Green et al., 2000; Green et al., 2004; Milev et al., 2005), however the
relationship does not appear to be generalised as specific neurocognitive domains have
been associated with particular functional indices. Meta-analytic results indicate that of
all neurocognitive abilities, only declarative memory seems to be related to a variety of
functional domains such as social interactions, occupational functioning and
independent living (Green et al., 2000). Furthermore, the meta-analysis indicated
executive functions and working memory are specific correlates of occupational
functioning and independent living, whereas attention/vigilance was related to
occupational and interpersonal functioning. In accordance with these findings, Pinkham
and Penn (2006) did not detect any association between executive functions and
interpersonal skill in their regression analyses. On the other hand, Pinkham and Penn’s
(2006) study failed to demonstrate a significant relationship between working or verbal
memory and social functioning, which contradicts previous research. Given the
methodological inconsistencies across studies, particularly the high level of variability
in the actual cognitive and functional domains assessed (Pinkham and Penn (2006) only
measured one area of functioning), such research findings should be interpreted with caution.

The overall relationship between neurocognition and social functioning appears to be significant, however the variance explained by neurocognitive variables has only been moderate (Green, 1996; Green, et al., 2000). Despite one study indicating that neurocognitive composite scores account for up to 60% of the variance in functional outcome (Green, 1996), the majority of studies report variance in the range of 20-40%. This implies that 60-80% of the variance in functioning cannot be explained by neurocognitive deficits.

4.2.2 Social Cognition and social functioning

As perception and interpretation of social information is required to facilitate social interactions, there has been recent interest in examining how social cognition may relate to social functioning and outcomes. Couture and colleagues (2006) proposed a theoretical model of social cognition and its relationship with social functioning, which highlights that deficits in social/emotion perception, ToM impairment, and an externalising attributional bias may all impact on social behaviour and functional indices. The model posits that deficits in perception of emotion or social cues inevitably leads to misinterpretations, and subsequent events in one’s social environment are then explained by factors influenced by an self-serving attributional style (which may not be corrected if ToM deficits are present), resulting in poor social behaviour. There is emerging evidence to suggest that social cognitive deficits, particularly in emotion/social perception and ToM, are significantly related to social dysfunction in schizophrenia and early psychosis (Addington et al, 2006; Brüne, 2005b; Brüne et al,
2007; Horan et al., 2012; Mueser, et al., 1996; Pinkham & Penn, 2006; Sullivan et al., 2013).

Some studies have reported that social cognitive variables account for more unique variance in social functioning than neurocognition (Brüne et al., 2007; Brüne, 2005b; Lysaker et al., 2005; Pinkham & Penn, 2006; Roncone et al., 2002), which has important implications for intervention strategies in terms of targeting specific psychological processes to improve outcome. In Brüne and colleagues’ (2007) study, impaired ToM ability was identified as the strongest independent predictor of abnormal social behaviour of schizophrenia inpatients, explaining approximately 50% of the variance. When IQ was controlled for, this relationship was strengthened further; ToM deficits impacted even more greatly on social competence for patients with at least average intelligence, providing evidence that ToM is a distinct cognitive construct that has a significant effect on social functioning. Sullivan et al. (2013) also recently found ToM to be a significant predictor of social functioning in a sample FEP patients.

Previous studies have applied correlational analyses to examine the relationship between social cognition and social functioning (Green, 1996, Green et al., 2000; Pinkham & Penn, 2006) however such basic statistical techniques do not allow for any conclusions regarding causation, which consequently limits the understanding of the influence of social cognition on functional outcome. More recent research has utilised path analysis (Brekke, Kay, Lee, & Green, 2005) and structural equation modelling (Sergi et al., 2006) to investigate such associations, revealing that social cognition also
acts as a mediator between neurocognitive variables and various functional indices. Such analyses address the nature of the relationship between cognition and general functioning, that is, how they are related and not simply if they are related. The importance of social cognition as a determinant of functioning and outcome can be appreciated by Sergi and colleagues’ (2006) study, which showed early visual processing (a neurocognitive variable) directly predicted the functional domains of independent living, social functioning and occupational functioning, however this relationship was no longer significant when social perception was introduced into the model and acted as a mediating variable between the neurocognitive and outcome variables.

Results of some studies investigating the relationship between social cognition and functioning should be interpreted with caution for a number of reasons. A major pitfall is that many studies have only examined one particular area of these broad constructs (e.g. Brekke et al., 2005; Pinkham & Penn, 2006; Sergi et al., 2006), thus generalisability of findings to other areas within these domains is not possible. In addition, and as mentioned earlier in this thesis, differences in assessment batteries and statistical procedures pose difficulties in making direct comparisons between studies and drawing valid conclusions. Regarding the relationship between ToM and social functioning, examining the association between the two without analysing the effect of first-order versus second-order tasks separately may influence findings. As first-order ToM abilities are generally spared in schizophrenia (Doody et al., 1998; Frith & Corcoran, 1996; Pickup & Frith, 2001), failing to exclude or control for performance on such tasks
may result in less variance accounted for by mental state attribution than other studies that compare performance on these tasks to social functioning indices separately.

A number of other limitations are also apparent from the ToM literature. Lower intellectual functioning as measured by general IQ has been shown to adversely affect ToM abilities (Doody et al., 1998). Moreover, IQ is known to decline over time in individuals with schizophrenia (Brüne et al., 2007), hence it could be expected that social impairment as a function of ToM deficits may be less severe in early psychosis populations if certain aspects of IQ contribute to mental state attribution. Some studies have shown that deficits in ToM are a feature of schizophrenia irrespective of IQ (Brüne et al., 2007; Pickup & Frith, 2001), suggesting they are quite separate constructs, however the extent of the relationship between IQ and ToM ability is not fully known and failing to control for IQ (e.g. Brüne, 2005b; Lysaker et al., 2005; Roncone et al., 2002) is a potential confounding variable. Furthermore, using measures with poor psychometric properties (e.g. low reliability) may also confound results, and it is not uncommon for studies to avoid reporting these statistics. Pinkham and Penn (2006) did include reliability data relating to the social cognitive measures used in their study and reported that a particular ToM measure (vignettes) had low reliability (0.31), which effectively limits interpretations about the predictive value of ToM on social functioning.
4.2.3 Symptomatology and social functioning

In addition to both neurocognitive and social cognitive deficits, clinical features have also been implicated as contributory factors to poor social functioning in schizophrenia. Negative symptoms have been consistently linked to social dysfunction (Bellack, Morrison, Wixted & Mueser, 1990; Guaiana, Tyson, Roberts, & Mortimer, 2007; Smith et al., 2002; Ventura et al., 2009; Zuo et al., 2012), and reduced social contact has been associated with greater negative affect and anhedonia (Blanchard, Mueser & Bellack, 1998). It is possible, however, that robust associations between negative symptoms and functional status in such studies are a result of theoretical relatedness between negative symptomatology and measures of social functioning, for example, individuals with anhedonia and apathy will likely display social impairment.

Positive symptoms such as hallucinations and delusions have been linked to poor social functioning (Brüne et al., 2007; Mancuso, Horan, Kern, & Green, 2011; Zuo et al., 2012), although some studies have not found the relationship to be significant (e.g., Smith et al., 2002). A few studies have also provided evidence for an association between disorganised symptoms and social dysfunction in schizophrenia (Smith et al., 2002; Zuo et al., 2012), however research regarding the relationship between this symptom dimension and social functioning is quite limited.

4.3 Summary

Schizophrenia is a chronic and disabling mental illness that can have serious implications on individuals suffering from the disorder, as well as healthcare and welfare services. Clinical symptoms, neurocognitive impairment and social dysfunction
are all well-known features of schizophrenia however specific aetiological factors remain unknown. From a neuroscientific perspective, neurobiological abnormalities and genetic variations have been implicated as underlying causes, but more recently researchers have suggested that deficits in social cognition may contribute to the manifestations of the disorder. Before any specific conclusions can be drawn from the literature, a number of methodological limitations need to be addressed in order to substantiate the current findings.

Emerging evidence indicates social cognition is impaired in schizophrenia and that, similar to neurocognition, the nature of this impairment is generalised rather than domain-specific (Bertrand et al, 2007). However, social cognitive functions are not defined uniformly in the literature and the measures used to test such abilities vary considerably across studies, making cross-study comparisons difficult. In order to overcome such inconsistencies, future studies should: a) incorporate a multi-factorial design to investigate a variety of well-defined social cognitive functions that represent different constructs, and b) use standardised and well-validated cognitive assessment tools.

Research has shown that social cognitive and neurocognitive processes are related to some degree (Fanning et al., 2012; Kee et al., 1998; Kindermann et al., 1998; Sergi et al., 2007), however some studies (e.g. Brekke et al., 2005; Corrigan et al., 1994) have only assessed one particular social cognitive construct, thereby limiting the assumptions that can be made about the nature and extent of such relationships. Again, future studies should assess a variety of social cognitive and neurocognitive functions using
standardised measures in order to provide a clearer picture of the specific associations between the two broad cognitive domains.

Research is quite limited with regard to the relationship between cognition and symptomatology, however there is a clearer relationship between neurocognitive impairment and negative symptoms as opposed to positive symptoms. Similarly, performance deficits in social cognition, particularly theory of mind, are more commonly associated with negative symptoms and also the disorganised symptom dimension (Corcoran et al., 1995; Frith & Corcoran, 1996; Greig et al., 2004; Pickup & Frith, 2001; Shamay-Tsoory et al., 2007). Given the limited amount of research in this specific field, further investigation is required to clarify the nature of the relationships between the three symptom dimensions and particular social cognitive domains, especially in early psychosis populations as nearly all studies have used chronic samples.

Furthermore, research has indicated that deficits in social cognition account for independent variance in social dysfunction, even more so than neurocognitive impairments (Brekke et al., 2005), and also act as a mediator between neurocognition and functional status (Sergi et al., 2006). Much like any research involving social cognition, a major limitation of these studies was that only one aspect of social cognition (e.g. social perception) was assessed. Similar to social cognition, social functioning is a broad concept that constitutes a variety of domains and therefore relationships between specific facets of social cognition and functioning (e.g. emotion recognition and vocational functioning; theory of mind and peer relationships) requires further investigation in order to clarify the nature of associations. Moreover, this line of
research is limited in terms of generalisability of findings to early psychosis populations as the majority of studies have only included participants with an established form of the illness.

Many social functioning measures were developed for adults with chronic schizophrenia and therefore may not be suitable for first episode populations, which consist predominantly of adolescents and young adults. Future research with early psychosis samples should consider using social functioning measures that have been validated for use with adolescents or young adults, and which tap into functional domains relevant to individuals at this stage of the lifespan (e.g. high school or tertiary education, peer interactions and dating).
CHAPTER 5: The Current Research Project

5.1 Rationale for the Current Research

Cognitive impairment is considered a characteristic feature of psychotic disorders, especially schizophrenia. Strong empirical evidence also exists for cognitive dysfunction in first episode psychosis (FEP) samples, that is, even before an established illness has emerged. However, it is less clear whether such impairment includes the specific area of social cognition, particularly in the early phase of illness. This is due to the fact that the vast majority of research in this area has been conducted using established or chronic schizophrenia samples, thus while there is literature emerging in this area, less is known about the social cognitive functioning of individuals who have experienced psychosis without having necessarily met the criteria for a schizophrenia-spectrum or bipolar disorder. The current research was conducted to explore social cognition in the early phase of schizophrenia-spectrum disorders using a FEP sample, in order to add to the growing evidence that deficits exist even in the initial stage of illness, and to investigate whether such deficits are linked to the poor social functioning commonly experienced by FEP individuals. The ultimate objective of this research is to gain a better understanding of social cognition in FEP and its relationship to other features of the disorder, as to provide information that can aid the development of novel targeted treatment interventions in early psychosis in order to improve prognosis and outcomes.

As discussed earlier in this thesis, the methodological issues in the literature concerning assessment of social cognition have posed problems in the generalisability of findings.
As a specific example, most studies have investigated only one or two domains (for example, theory of mind or emotion recognition) and have subsequently drawn conclusions about “social cognition” in general, when in fact social cognition is considered to be multidimensional. The current research, which was initially conceptualised in 2007, aimed to address this matter by using a variety of tasks so that multiple domains of social cognition could be assessed within the same study. At that time, social cognition was still poorly defined and consensus regarding representative domains was not definitive. In fact, this continues to be an issue, which creates ambiguity for researchers in this field and consequently limits comparability of findings between studies. Based on the overall literature when the research project was first designed, four to five main areas fell under the umbrella of social cognition; emotion recognition, social/emotion perception, theory of mind, social knowledge, and attributional style (Green et al, 2008). As such, these domains (with the exception of attributional style) were subsequently selected for investigation in the current research. The researcher decided not to assess attributional style for a number of reasons, which are raised in the General Discussion of this thesis (see Chapter 10).

Even though social cognition as an overall construct has been generally regarded as distinct to neurocognition (Fanning et al., 2012; Harvey & Penn, 2010; Pinkham et al., 2003; Sergi et al., 2007), the evidence suggests that certain social cognitive and neurocognitive domains overlap with each other (Fanning et al., 2012; Sergi et al., 2007; Ventura et al., 2011). Thus, it was deemed important to also assess neurocognition in the current research, in order to delineate this issue and better inform treatment interventions that target both neurocognition and social cognition. The MATRICS
Consensus Cognitive Battery, which includes various standardised tests that measure the neurocognitive domains most consistently shown to be impaired in individuals with schizophrenia (Nuechterlein et al., 2004), was chosen to assess neurocognition. An overview of the MATRICS Consensus Cognitive Battery is provided in Appendix B and can also be viewed online (www.matricsinc.org) in more detail. The battery can be purchased through providers of psychological testing tools, for example, Psychological Assessment Resources (PAR).

The researcher also considered it important to assess schizophrenia-spectrum psychopathology, given reported associations between certain types of psychotic symptoms and cognitive variables. This raises the question as to whether impairments in social cognition play a role in the development of psychotic symptoms. The Positive and Negative Syndrome Scale (PANSS; Kay et al., 1989) (see Appendix C) was chosen to assess psychopathology, as it is a well-known and widely used measure of various psychotic and general psychiatric symptoms. Furthermore, it can indicate the severity of three main symptom subtypes in schizophrenia (i.e. positive, negative, and disorganised) using Liddle’s (1987) conceptualisation (see Appendix D for details of the methodology).

Furthermore, social cognition, neurocognition and symptomatology have all been implicated in underlying social impairment in schizophrenia. The degree to which each of these factors influences social functioning in FEP has not been previously investigated, thus comprehensive assessment of all three factors was necessary in order
to ascertain whether particular characteristics in psychosis have a more profound affect on functioning. The measurement of social functioning itself has been broad and varied in the literature, therefore the validated measures of functioning chosen for the purpose of this research (i.e., The Global Functioning Scales and the Multidimensional Adolescent Functioning Scale; see Appendices E and F) were matched to the sample based on criteria such as age-appropriate items reflecting anticipated functional status.

5.2 Research Aims and Hypotheses

Based on the rationale presented above, a research proposal was submitted to the Deakin University Human Research Ethics Committee to investigate the broad topic of social cognition in FEP. The overall purpose of the research was to determine whether social cognitive impairment is in fact a feature of FEP, and if social cognition is associated with other key clinical and characteristic features of the illness as a means of possibly informing psychosocial treatment interventions. The project received ethical approval in 2008 and recruitment of participants began thereafter at Orygen Youth Health (OYH); a public youth mental health service that contains a specialised FEP clinic, namely, the Early Psychosis Prevention and Intervention Centre (EPPIC). OYH services the north-western metropolitan region of Melbourne, Australia, and is available to those aged between 15 and 25 years. Recruitment of FEP outpatients was achieved via one of two avenues; i) clients of EPPIC were informed of the study by their treating clinician and subsequently asked if they were interested in participating in the research, or ii) interest was expressed by the client after seeing a flyer about the research (flyers were circulated around the service). A healthy control group matched on age-range was
also recruited from the same catchment area. Healthy controls were sourced via newspaper advertisement and flyers distributed in local areas.

Interested participants were provided with a Plain Language Statement and Consent Form (see Appendix G and Appendix H for patient and control copies, respectively) and were verbally informed of the requirements of participation. Any further queries from prospective participants were answered by the researcher, and for those agreeable to participating, the consent form was signed and an assessment session was scheduled either at OYH or a location convenient for the participant.

As the research project was multidimensional and comprised various specific aims and objectives, four separate studies were devised and each prepared for publication in peer-reviewed journals following data collection and analysis. The specific research aims and hypotheses pertaining to each study are summarised below.

**Article One: Social Cognition in Clinical “at risk” For Psychosis and First Episode Psychosis Populations (Andrew Thomopson, Alicia Papas, Cali Bartholomeusz, Kelly Allott, G. Paul Amminger, Barnaby Nelson, Stephen Wood, & Alison Yung).**

Published in Schizophrenia Research, 2012, Volume 141, Pages 204-209.

As social cognition essentially refers to the mental operations that underlie social interactions, behavioural scientists and clinical investigators proposed the idea that deficits in these processes may contribute to the symptoms and interpersonal
difficulties experienced by individuals with schizophrenia (Penn et al., 1997). Since that time some 15 years ago, interest in the area surged and social cognitive impairment is now considered one of the key features of the disorder. However, due to vague and varied descriptions of particular social cognitive domains in the literature and other methodological concerns (i.e. the multitude of tools available to measure various constructs; limited research in early psychosis versus established illness), it remains unclear as to whether particular social cognitive deficits exist in the earlier phase of illness.

The primary research aim was to determine if FEP patients exhibit impairments across a number of social cognitive domains, specifically: emotion recognition, theory of mind, social perception and social knowledge. A healthy control group was included in the study to provide a benchmark for performance. It was hypothesised that FEP patients would demonstrate poorer social cognition ability across all domains compared to age-matched controls (15-25 years olds), given that the overall literature has shown evidence of social cognitive impairment across various domains in schizophrenia. The results are presented in Article One (see Chapter 6), which has been published in *Schizophrenia Research*.

It is noted that the Article One includes additional data outside the scope of this thesis. Specifically, social cognition data from another clinical group, namely ‘ultra-high-risk’ (UHR) of psychosis, was included to add significance to the research for journal publication purposes. The UHR sample was also recruited and assessed by the author of
this thesis, as part of a parallel study conducted at OYH. The author of this thesis was working at OYH at the time the UHR research project was conceptualised, and was acknowledged as an Associate Investigator on that study. As an Associate Investigator, research duties included: review of the literature, study design and consideration of methodological issues such as task selection, scoring and management of data, consideration of statistical issues, interpretation of results, and preparation of Article One for publication. Although listed as the second author of Article One, the author of this thesis was considered as equal first author, given the depth of involvement in the UHR study.

Importantly, the multi-group comparison in Article One allowed for degree of impairment in social cognition to be assessed at different phases of illness, rather than simply addressing whether social cognition deficits exist in FEP or in UHR. For the purpose of this thesis, however, the reader’s attention in Article One should be focused on social cognitive performance between FEP patients and controls only, as the UHR data does not relate to the topic of the this thesis. The UHR data and associated results are not referred to outside of Article One.

**Article Two: Social Cognition and Neurocognition Represent Distinct Constructs in First Episode Psychosis and Control Cohorts (Alicia Papas, Linda Byrne, & Andrew Thompson). Submitted to Early Intervention in Psychiatry.**

As overall constructs, social cognition and neurocognition are generally considered as distinct from one another, however evidence exists to suggest they may represent
different expressions of the same construct given that moderate degrees of overlap between domains have been reported. Thus, the main aim of this study was to examine the relationships between four social cognitive domains and six neurocognitive domains to ascertain whether social cognition and neurocognition do, in fact, represent separate constructs.

A specific objective was to determine if the pattern of relationships are different between FEP and healthy controls, as almost all studies in the literature with a similar research purpose have only included a clinical group in their investigations without examining nonclinical individuals. Rather than to simply assess correlational relationships, a further objective of this study was to determine the degree of variance shared between each neurocognitive domain and each social cognitive domain. This would allow for unique variances to be systematically and quantitatively assessed. In line with previous research, it was hypothesised that the four social cognitive and six neurocognitive domains assessed would be relatively distinct from one another, as indicated by no more than small to moderate amounts of shared variance. Article Two (see Chapter Seven) presents the findings of this study, which has just been received back (July, 2013) from peer-review in *Early Intervention in Psychiatry*. The authors are in the process of making the suggested changes for resubmission.

**Article 3: The Relationship Between Symptomatology and Social Cognition in First Episode Psychosis (Alicia Papas, Linda Byrne, Cali Bartholomeusz, & Andrew Thompson). Submitted to Psychiatry Research.**
It has been speculated that certain cognitive processes, including social cognition, may underlie psychotic psychopathology. This hypothesis has been proposed in light of evidence linking areas of social cognition (in particular, theory of mind) to specific psychopathology, especially negative symptoms. Similarly, the symptom domain of disorganisation has shown some association to social cognitive impairment. Interestingly, positive symptoms have been relatively unrelated to social cognitive deficits in areas such as emotion recognition, ToM, and social perception. Overall, the literature has indicated mixed findings regarding the relationship between symptoms and social cognition. Moreover, a very limited amount of research exists concerning the early phase of illness. Thus, the purpose of this study was to examine the relationship between social cognition and psychotic symptoms in FEP, in order to ascertain if particular symptomatology is associated with social cognitive impairment in the early course of the illness. More specifically, this study aimed to investigate the relationship between four defined social cognitive domains and three symptom subtypes (i.e. negative, positive and disorganised). It was hypothesised that social cognitive deficits across the domains assessed would be most related to negative, and possibly disorganised symptoms, in FEP. Positive symptoms were not expected to show any association with the domains measured. The findings are presented in Article Three (see Chapter Eight), which has been submitted to Psychiatry Research for review.

As interpersonal difficulties and poor social functioning are common in individuals with psychosis, another goal of the current research was to investigate the contribution of three main characteristics of the disorders (i.e. social cognition, neurocognition and symptomatology) previously shown to influence outcomes in FEP. A specific objective was to determine the best predictor of poor social functioning. As recent findings have implicated social cognition as having the greatest impact on social outcomes, it was hypothesised that social cognition would account for more variance in social functioning than neurocognition or symptoms. Neurocognition was expected to predict social functioning to a lesser degree based, whereas symptoms were not anticipated to show any significant association given the nature of the sample (that is, stable outpatients in an early phase of illness). The results of this investigation are depicted in Article Four (see Chapter Nine) in an original journal article, which has been submitted to Psychiatry and Clinical Neurosciences for review.
CHAPTER 6

Article One: Social Cognition in Clinical “at risk” For Psychosis and First Episode Psychosis Populations

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Authors: Andrew Thompson, Alicia Papas, Cali Bartholomeusz, Kelly Allott, G. Paul Amminger, Barnaby Nelson, Stephen Wood, Alison Yung.
Abstract

**Background:** Social cognitive deficits have been demonstrated in first episode psychosis (FEP) and groups at high risk for developing psychosis but the relative degree of deficit between these groups is unclear. Such knowledge may further our understanding of the importance of these deficits in the development of psychosis. The study aimed to compare the degree of impairment in social cognition in three groups: FEP, those at "Ultra High Risk" (UHR) for psychosis and healthy controls. **Methods:** UHR and FEP patients were recruited from an established youth mental health service in Melbourne. Three domains of social cognition were assessed: ToM (Hinting Task and interpretation of Visual Jokes); facial and vocal emotion recognition (Diagnostic Assessment of Non Verbal Accuracy); social perception (Mayer-Salovey-Caruso Emotional Intelligence Test - managing emotions branch). Group differences were analysed using Analysis of Covariance with age, gender and IQ as covariates. **Results:** Data on 30 UHR, 40 FEP and 30 control participants were analysed. FEP patients performed significantly worse on all social cognition tasks compared to controls. For the UHR group, scores were intermediate between FEP and controls for all tasks, but only significantly different to controls for ToM tasks. Effects sizes were largest for the ToM tasks and the emotion recognition task for both patient groups. There were no significant differences between UHR and FEP patients in performance on any of the tasks. **Conclusions:** Social cognition is generally impaired in FEP patients but there are fewer deficits in a UHR group. Longitudinal research in larger samples is needed to investigate whether social cognition deficits, such as ToM are risk factors in UHR groups for subsequent transition to full-threshold psychosis.
1. Introduction

Social cognition has been defined as the domain of cognition that involves the perception, interpretation and processing of social information (Ostrum, 1984). Patients with schizophrenia have been consistently found to be impaired in a number of domains of social cognition including emotion perception/recognition; Theory of Mind (ToM); adaptive attributional styles; and social perception/knowledge (for review see (Penn et al., 2008)). The finding that these deficits appear to be present in patients in remission as well as in the acute phase of illness (Edwards et al., 2001; Janssen et al., 2003) raises the question as to whether such deficits are trait rather than state phenomena in individuals with schizophrenia-spectrum disorders.

In attempting to address this question, attention has turned to investigating social cognition skills earlier in the course of psychotic illness, both in those experiencing their first episode of psychosis (FEP), and more recently in “at risk” for psychosis groups. A number of studies have consistently demonstrated significant impairments in FEP (Krstev et al., 1999; Edwards et al., 2001; Addington et al., 2006; Inoue et al., 2006; Bertrand et al., 2007). One particular “at risk” group that has attracted recent research interest is the “ultra high risk” (UHR) or “clinical high risk” (CHR) (Yung et al., 1998) population. To date, there have been six published studies on social cognition deficits specifically in UHR populations which have reported deficits, though not consistently, in all domains of social cognition (for review, see (Thompson et al., 2011)).
Four of these studies have attempted to investigate the “degree” of deficit in UHR populations compared to early psychosis populations. These studies have generally demonstrated similar levels of deficit in social cognition domains in the “at risk” participants and the early psychosis participants (Addington et al., 2008; Couture et al., 2008; Amminger et al., 2011; Green et al., 2011). However, only two of these studies have compared the patient groups on more than one social cognitive domain. Of these two studies, one found deficits in one of the social cognitive domains tested but not the other (Couture et al., 2008). In addition to this, the early psychosis samples in these studies that have investigated more than one domain have been distinctly different (FEP and early schizophrenia spectrum illness or early schizophrenia) (Green et al., 2011), none of the studies have adequately accounted for IQ or other neuropsychological differences between the groups and the proportion of antipsychotic treatment in the “at risk” group (which may influence the results) was either relatively high (Green et al., 2011) or not stated (Addington et al., 2008; Couture et al., 2008).

In light of these findings and the methodological shortcomings in existing studies, the present study attempted to further address the question of whether social cognition deficits are present both in FEP and those at UHR. The aim of the current study was to investigate the degree of impairment in multiple domains of social cognition in three groups: FEP, UHR and healthy controls. It was hypothesized that both patient groups would perform worse than controls across all social cognitive domains.
2. Methods

2.1. Participants

The FEP and UHR patient groups were recruited from a public youth mental health service (Orygen Youth Health) for 15-25 year olds that serves northwestern Melbourne, Australia. The service consists of a number of specialist clinics including the PACE (Personal Assistance and Crisis Evaluation) clinic, which assesses and treats UHR patients, and EPPIC (Early Psychosis Prevention and Intervention Centre), which assesses and treats those experiencing a first episode of psychosis. The PACE clinic offers a comprehensive clinical service for UHR patients for a 6-month period and patients in the EPPIC clinic receive up to 2 years of treatment. Both clinics provide a comprehensive treatment package comprising cognitive behavioural case management and appropriate pharmacological and medical treatment (Edwards et al., 2002; Yung et al., 2007). Potential recruits were identified at clinical review meetings. An attempt was made to approach all new referrals to the PACE clinic during the recruitment period unless they met the exclusion criteria. FEP patients were approached at the early or late recovery stage of their treatment and were assessed when deemed clinically stable.

Non-psychiatric control participants were recruited via advertisements in the local newspaper and at local community health services in the same geographical catchment area as the clinical service.
2.2. Inclusion criteria

2.2.1. UHR individuals

The CAARMS (Yung et al., 2005) was used to assess whether individuals met UHR criteria. The UHR criteria consist of at least one of the following: (i) State and trait risk factors (ii) Attenuated psychotic symptoms (iii) Transient psychotic symptoms. The criteria are further described in Table 1. The rationale for these criteria has been previously described (Yung et al., 2004a).

Table 1. Ultra High Risk criteria: (1) must be aged between 15 and 25 years, (2) have been referred to a specialised service for help, (3) have experienced a drop in functioning of at least one month over the last year or sustained low functioning, and (4) meet the criteria for one or more of the following three groups.

<table>
<thead>
<tr>
<th>Group 1: Attenuated positive psychotic symptoms</th>
<th>• Presence of at least one of the following symptoms: ideas of reference, odd beliefs or magical thinking, perceptual disturbance, paranoid ideation, odd thinking and speech, odd behavior and appearance • Frequency of symptoms: at least several times a week • Recency of symptoms: present within the last year • Duration of symptoms: present for at least 1 week and no longer than 5 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 2: Brief limited intermittent psychotic symptoms</td>
<td>• Transient psychotic symptoms. Presence of at least one of the following: ideas of reference, magical thinking, perceptual disturbance, paranoid ideation, odd thinking or speech • Duration of episode: less than 1 week • Frequency of symptoms: at least several times per week • Symptoms resolve spontaneously • Recency of symptoms: must have occurred within the last year</td>
</tr>
<tr>
<td>Group 3: Trait vulnerability group</td>
<td>• Schizotypal personality disorder in the identified individual or a first-degree relative with a psychotic disorder</td>
</tr>
</tbody>
</table>

Note: See Yung et al (Yung et al., 2003; Yung et al., 2004b) for the full operationalised criteria.
2.2.2. FEP Individuals

Patients are accepted into the EPPIC program if they have experienced at least 1 week of daily psychotic symptoms based on CAARMS criteria and had less than 6 months previous treatment for a psychotic disorder (Edwards et al., 2002). All EPPIC patients were eligible for the study regardless of specific psychosis diagnosis. All patients were in either the early or late phase recovery of their psychotic illness.

2.2.3. Control Individuals

Control participants were included if they were in the same age range as the clinical samples (15-25 years) and lived in the same geographical area as that served by the clinic.

2.3 Exclusion criteria

For all groups, individuals with a documented history of intellectual disability (i.e., IQ less than 70) were excluded, as well as those individuals receiving treatment for significant neurological disorder (such as epilepsy). Those with impaired visual acuity (i.e., blurred vision or less than corrected 20/40 vision) or corrected auditory acuity were also excluded from the study. Other exclusion criteria included: intoxication with illicit drugs or alcohol during the testing and poor English language skills. FEP patients were not recruited if they were acutely unwell at the time they were approached, i.e., needing inpatient care or acute interventions by the service crisis team.
Additionally, control participants were excluded if, via the brief clinical interview based on the SCID-I and CAARMS, they were found to have a current or previous psychiatric disorder or met the CAARMS criteria for UHR. The study received full ethical approval from the local Research and Ethics committees (Melbourne Health HREC and Deakin University HREC).

A total of 32 UHR individuals, 40 FEP individuals and 34 healthy control participants were recruited. 2 UHR participants were excluded: 1 due to poor English comprehension skills and 1 due to very limited completion of the assessment battery before moving overseas. 4 controls were also excluded: 1 had a previously diagnosed depressive illness, 2 met criteria for attenuated psychotic symptoms on the CAARMS and 1 had poor English comprehension skills.

2.4 Measures

2.4.1 Social cognition measures

2.4.1.1 Theory of Mind (ToM) tasks. (i). The Hinting Task (Corcoran et al., 1995) was used to test the participants’ ability to infer the real intentions behind indirect speech. In this task, 10 short passages are read aloud one at a time. Each passage depicts an interaction between two characters, and ends with one of the characters dropping an obvious hint. A second obvious hint is given if response to the first is incorrect. The total score is out of 20. This task has been well validated by Corcoran and colleagues (Corcoran et al., 1995) for use with adult clinical populations.
(ii). The Visual Jokes task (Corcoran et al., 1997). This requires participants to interpret
cartoons as jokes. A set of five ToM jokes (mentalising jokes) and five jokes based on
physical events (physical jokes) were shown to each participant. The cartoons were
presented using a computer program, which randomized the order both within and
between categories. Each joke was shown to the participant for as long as they needed
to understand it. The participant was then asked to explain the humour in the joke and
their responses were audio-recorded. The score allocated to each joke depended on the
type of interpretation offered using a rating scale, which reflected the degree of ToM
sophistication: 0) no adequate answer to the joke; 1) purely “physical” explanation of
the joke; 2) explanation of the joke with indirect or implied reference to emotional
states or theory of mind and 3) answer that specifically referred to mental states. This
gave an overall “mentalising” score (maximum 30) for the 10 jokes and sub-scores
(maximum 15) for the ToM and physical jokes. Scoring was achieved by consensus
between three of the investigators (AT, AP and CB) after correspondence with the task
author (Corcoran, personal communication). This paradigm has been well validated for
use in the adult clinical population (Corcoran et al., 1997).

2.4.1.2 Emotion recognition task. The adult version of the Diagnostic Analysis of
Nonverbal Accuracy-2 (DANVA-2) scale for faces and voices (Nowicki and Carton, 1993;
Nowicki and Duke, 1994) was used. The DANVA-2 Adult Facial expressions (DANVA-2-
AF) consists of 24 colour photographs of an equal number of happy, sad, angry and
fearful facial expressions of high and low intensities (Nowicki and Carton, 1993). The
individual is required to choose the correct facial emotion expression from the four
options given. The DANVA-2, Adult Paralanguage (DANVA-2-AP) component consists of
audio recordings of two professional actors responding to vignettes designed to elicit happy, sad, angry, and fearful emotions by saying a neutral sentence, "I am going out of the room now, but I'll be back later". There are 24 presentations of an equal number of happy, sad, angry and fearful voices of high and low intensities. Both the faces and voice stimuli were presented using a computer program, which randomised the order. A total score for all errors on either task (maximum 24) was computed. The DANVA-2 AF and AP have good reliability and reasonable construct validity (Nowicki and Duke, 1994).

2.4.1.3 Social/emotion perception task. Social perception was assessed using the Managing Emotions module (branch 4) of the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT) (Meyer et al., 2002). This test was primarily chosen because it is the only social cognition task included in the MATRICS consensus cognitive battery (Kern et al., 2008; Nuechterlein et al., 2008). In this task the participant is asked a number of questions based on vignettes relating to managing their emotions and the emotions of others. The total managing emotions score on the MSCEIT is scored using a web-based scoring program (available from Multi-Health Systems, Inc, Toronto, Ontario, Canada) using unadjusted consensus norms from a large normative sample. Scores are automatically calculated and scaled with a mean of 100 (SD = 15), with higher scores reflecting better emotional management. Whilst strictly a task of managing emotions, the MSCEIT has a considerable social cognitive skills component in that most of the questions assess the perception of social or interpersonal situations and appropriate responses to those social situations. In this regard it is similar to tasks of social perception.
2.4.2. Neuropsychological measures

IQ was estimated using the two subtest (vocabulary and matrix reasoning) version of the Wechsler Abbreviated Scale of Intelligence ([WASI; Wechsler, 1999]). The FEP patients did not complete the WASI as IQ data had already been collected on a number of these patients prior to the inception of the three group study using a different measure; their premorbid IQ was estimated based on performance on the NART (National Adult Reading Test) (Nelson, 1982). Although this is a measure of premorbid IQ, the correlation between the WASI-derived IQ and the NART-derived IQ in the control sample (who completed both WASI and NART) was very high (Pearson's r = 0.79).

Verbal working memory was measured using the letter-number sequencing test (Gold et al., 1997). Visual working memory was measured using the Wechsler Memory Scale – Third Edition (WMS-III) Spatial Span test (Nelson, 1982; Wechsler, 1997b).

2.4.3. Psychopathology, social functioning and clinical measures

Psychotic illness pathology was measured using the Brief Psychiatric Rating Scale (BPRS) (Overall and Gorham, 1962). Negative psychotic symptoms were measured using the Scale for Assessment of Negative Symptoms (SANS) (Andreasen, 1983). Social functioning was measured using the Social and Occupational Functioning Assessment Scale (SOFAS) (APA, 1994). Data were also collected on basic demographics, psychosis diagnosis and antipsychotic medication treatment.
2.5 *Statistical Analysis*

Demographic, social functioning, psychopathology and neuropsychological variables were analysed using chi-square tests for categorical variables and independent samples t-tests or one-way analyses of variance (ANOVA) for continuous variables. Social cognitive performance between the three groups was analysed using Analysis of CoVariance (ANCOVA) with IQ, age and gender as covariates. These were chosen given that previous studies have shown a significant relationship between some aspects of social cognition, especially ToM, and estimates of IQ (Corcoran et al., 1995; Corcoran et al., 1997), and social cognition skills have also been shown to vary by age and gender (Blakemore, 2008). The social cognition scores used in the primary analyses were total scores (and where applicable sub-scores) for all of the social cognition measures. Post hoc analyses were undertaken comparing each of the patient groups to the control group and also comparing the patient groups to one another. All analyses controlled for multiple testing using the False Discovery Rate (FDR) correction for multiple comparisons (Benjamini et al, 1995: Storey, 2002). Effect sizes of the mean differences between controls and UHR/FEP groups on all social cognition tasks were also calculated using a method derived from Cohen (1988). All analyses were conducted using STATA version 11.

3. Results

3.1 Demographic, neuropsychological, functioning and psychopathology measures

Demographics, neuropsychological results, social functioning and psychopathology data for all three groups are summarized in Table 2. There were no significant differences in the three groups with respect to age, gender, IQ, years of education, visual working
memory performance, and verbal working memory performance. Levels of psychiatric and negative symptoms were similar in the two patient groups, with the FEP group having marginally lower functioning (see Table 2). At the time of assessment, 3 of the 30 UHR and 30 of the 40 FEP participants were prescribed antipsychotic medication. Of the UHR sample, the majority (22/30, 73.3%) met criteria for the attenuated symptoms group, with small numbers meeting both the trait and attenuated symptoms criteria, (5/30, 16.7%), the trait criteria alone (2/30 6.7%) or a combination of trait, attenuated symptoms and BLIPS (1/30, 3.3%).
Table 2. Comparison of the three groups on demographics, IQ, functioning and psychopathology.

<table>
<thead>
<tr>
<th></th>
<th>Controls (n=30)</th>
<th>UHR (n=30)</th>
<th>FEP (n=40)</th>
<th>Statistical comparison P value #</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age, mean (SD)</strong></td>
<td>19.3 (2.9)</td>
<td>19.1 (2.8)</td>
<td>20.5 (2.5)</td>
<td>0.06</td>
</tr>
<tr>
<td><strong>Females, n (%)</strong></td>
<td>18 (58.1)</td>
<td>16 (53.3)</td>
<td>15 (37.5)</td>
<td>0.19</td>
</tr>
<tr>
<td><strong>Years of education, mean (SD)</strong></td>
<td>13.2 (2.0)</td>
<td>12.0 (1.9)</td>
<td>12.5 (1.6)</td>
<td>0.06</td>
</tr>
<tr>
<td><strong>Current occupation, n(%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Unemployed</strong></td>
<td>3 (10.0)</td>
<td>7 (23.3)</td>
<td>21 (52.5)</td>
<td>0.001</td>
</tr>
<tr>
<td><strong>Part-time/casual work</strong></td>
<td>5 (16.7)</td>
<td>2 (6.7)</td>
<td>4 (10.0)</td>
<td></td>
</tr>
<tr>
<td><strong>Full time work</strong></td>
<td>2 (6.7)</td>
<td>4 (13.3)</td>
<td>7 (17.5)</td>
<td></td>
</tr>
<tr>
<td><strong>Student</strong></td>
<td>20 (66.7)</td>
<td>17 (56.7)</td>
<td>8 (20.0)</td>
<td></td>
</tr>
<tr>
<td><strong>Estimated IQ, mean (SD)</strong></td>
<td>104.9 (12.5)</td>
<td>103.3 (16.0)</td>
<td>106.4 (10.6)</td>
<td>0.62</td>
</tr>
<tr>
<td><strong>Verbal working memory score (letter-number sequencing), mean (SD)</strong></td>
<td>15.6 (2.8)</td>
<td>15.2 (3.8)</td>
<td>14.6 (2.7)</td>
<td>0.38</td>
</tr>
<tr>
<td><strong>Visual working memory score (spatial span), mean (SD)</strong></td>
<td>17.8 (2.3)</td>
<td>16.7 (2.1)</td>
<td>17.1 (2.6)</td>
<td>0.19</td>
</tr>
<tr>
<td><strong>Social Functioning:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>SOFAS score, mean (SD)</strong></td>
<td>84.1 (8.3)</td>
<td>60.7 (11.1)</td>
<td>56.0 (13.3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>Clinical ratings:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>BPRS score, mean (SD)</strong></td>
<td>N/A</td>
<td>45.5 (8.2)</td>
<td>44.1 (12.8)</td>
<td>0.61</td>
</tr>
<tr>
<td><strong>SANS score, mean (SD)</strong></td>
<td>N/A</td>
<td>23.6 (15.0)</td>
<td>25.9 (14.1)</td>
<td>0.51</td>
</tr>
</tbody>
</table>

* Abbreviations: BPRS, Brief Psychiatric Rating Scale; SANS, Scale for Assessment of Negative Symptoms; SOFAS, Social and Occupational Functioning Scale

# ANOVA or Chi-squared analyses
3.2 Social cognition performance

3.2.1 Overall between-group comparisons

Means and standard deviations of all task scores for the controls, UHR and FEP groups are shown in Table 3. One way between-group ANOVAs revealed significant between-group differences on total scores for all social cognitive tasks (see Table 3). These significant group differences remained after controlling for age, gender and IQ and after controlling for multiple testing.

3.2.2 FEP compared to controls

Post-hoc Tukey tests adjusting for multiple testing revealed that FEP participants performed significantly worse than controls for all tasks and in all social cognition domains (see Table 3 for means and standard deviations).

3.2.3 UHR compared to controls

Scores for UHR individuals were mostly intermediate to those of controls and FEP. Post-hoc tests revealed that the UHR group performed significantly worse than controls on two Theory of Mind tasks, the Hinting task (mean difference -1.34, p=0.04) and the physical jokes section of the visual jokes task (mean difference 1.37, p<0.01).

3.2.4 FEP compared to UHR

In post-hoc testing there were no significant differences in performance on social cognition tasks between the FEP and UHR groups. Effect sizes regarding the difference in scores between controls and each of the patient groups were also calculated. The effect sizes were generally medium to large for the differences between controls and FEP, but small to medium when comparing controls to UHR. The effect sizes were
largest for the ToM hinting task (FEP, 0.93; UHR, 0.64) followed by the emotion recognition Danvas task (FEP, 0.91; UHR, 0.48), then the ToM visual jokes task (FEP, 0.64; UHR, 0.37) and lastly the social perception MSCEIT task (FEP, 0.62; UHR, 0.29).
Table 3. Means and standard deviations of social cognition task scores for the three participant groups along with statistical comparison of group effect (ANOVA).

<table>
<thead>
<tr>
<th></th>
<th>Controls (n=30)</th>
<th>UHR (n=30)</th>
<th>FEP (n=40)</th>
<th>Group effect</th>
<th>Unadjusted</th>
<th>Adjusted #</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>F</td>
<td>P value</td>
<td>F</td>
</tr>
<tr>
<td><strong>Theory of Mind</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hinting task score, mean (SD)</td>
<td>18.0 (1.15)</td>
<td>16.5 (3.11)*</td>
<td>15.7 (3.31)**</td>
<td>6.06</td>
<td>&lt;0.01</td>
<td>7.28 &lt;0.01</td>
</tr>
<tr>
<td>Overall visual jokes total score, mean (SD)</td>
<td>19.6 (5.25)</td>
<td>17.7 (5.06)</td>
<td>16.8 (3.28)**</td>
<td>3.38</td>
<td>0.05</td>
<td>3.89 0.03</td>
</tr>
<tr>
<td>Mentalising jokes subscore, mean (SD)</td>
<td>10.1 (2.07)</td>
<td>9.9 (3.25)</td>
<td>9.0 (2.37)</td>
<td>1.85</td>
<td>0.16</td>
<td>2.55 0.08</td>
</tr>
<tr>
<td>Physical jokes subscore, mean (SD)</td>
<td>9.3 (1.78)</td>
<td>7.8 (2.31)**</td>
<td>7.8 (1.79)**</td>
<td>6.31</td>
<td>&lt;0.01</td>
<td>7.78 &lt;0.01</td>
</tr>
<tr>
<td><strong>Social Perception</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MSCEIT branch 4 total score, mean (SD)</td>
<td>92.0 (10.38)</td>
<td>89.0 (10.33)</td>
<td>85.8 (9.70)**</td>
<td>3.30</td>
<td>0.05</td>
<td>4.43 0.03</td>
</tr>
<tr>
<td><strong>Emotion recognition</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Combined emotion recognition score, mean (SD)</td>
<td>10.0 (3.66)</td>
<td>11.8 (3.85)</td>
<td>13.7 (4.43)**</td>
<td>7.38</td>
<td>&lt;0.01</td>
<td>6.13 &lt;0.01</td>
</tr>
<tr>
<td>Facial affect recognition score, mean (SD)</td>
<td>4.5 (2.01)</td>
<td>5.2 (2.47)</td>
<td>6.3 (3.08)*</td>
<td>4.32</td>
<td>0.03</td>
<td>2.94 0.07</td>
</tr>
<tr>
<td>Prosodic affect recognition score, mean (SD)</td>
<td>5.5 (2.68)</td>
<td>6.6 (2.42)</td>
<td>7.4 (2.54)**</td>
<td>4.82</td>
<td>0.02</td>
<td>4.24 0.03</td>
</tr>
</tbody>
</table>

* ANCOVA with age, gender and IQ as covariates, adjusting for multiple comparisons (FDR).

Asterisks refer to post hoc analyses comparing UHR to controls and FEP to controls respectively, adjusting for age, gender and IQ and controlling for multiple testing (FDR): *p<.05, **p<.01

* Italicics signify sub-scores of social cognitive measure
4. Discussion

4.1. Summary of the results

The results indicate that FEP participants were impaired on the social cognitive domains of ToM, emotion recognition and social perception when compared to healthy control individuals. Effect sizes were largest for the ToM and emotion recognition tasks. The significant overall group differences across all social cognition tasks in the one-way ANOVA’s appeared to be mostly driven by the comparison between FEP and controls. The performance of UHR participants, whilst intermediate on all social cognition tasks to that of FEP and controls, was only significantly different to control participants for some of the ToM tasks. Thus, the authors’ hypothesis was not fully supported with the results indicating that the degree of impairment on social cognition tasks is less pronounced in UHR than FEP patients compared to controls; the degree of deficit however, is not consistent for all social cognition tasks.

4.2. Comparison with previous studies comparing “at risk” and FEP groups

Other research groups have reported that UHR individuals have similar deficits in social cognition domains in terms of degree of impairment relative to FEP/first-episode schizophrenia patients (Addington et al., 2008; Couture et al., 2008; Green et al., 2011). However, this has not been consistent across studies (Pinkham et al., 2007; Couture et al., 2008). Results of the current study provide support for an intermediate level of deficit and not the same level of deficit in UHR individuals relative to those with FEP, with some domains possibly more affected than others. In this respect the results are similar to those found for neurocognitive impairments in the UHR group (Brewer et al,
2006). Direct comparison with these previous studies is somewhat difficult given that
different social cognitive tasks were used. The only task that is comparable to another
study is the managing emotions test of the MSCEIT where scores in our sample were
very similar to those of Green and colleagues (Green et al., 2011). One continued
problem in the field is the lack of consensus on which tasks to use and the variable
psychometric properties of these tasks. An initiative similar to the MATRICS working
group (Nuechterlein et al., 2008) for social cognition researchers might address some of
these issues.

It is worth highlighting that our results still make it difficult to distinguish whether the
deficits in social cognitive domains are simply a trait factors for psychosis, a specific risk
factor (variable or causal) or a combination of the two. It is reasonable to hypothesise
how poor social cognition may make an individual vulnerable to exposure to other risk
factors such as bullying, poor social functioning as well as predispose to symptom
formation. As the study was cross sectional we cannot address the issues of: a) within-
group differences exist for UHR individuals depending on whether they “transition” to a
psychotic disorder and b) the potential change (worsening) in social cognition with
progression from “prodromal” to FEP to chronic psychosis. We would encourage further
longitudinal research into the relationship between social cognition, transition to
psychosis and functioning in this population and other “at risk” populations to further
define the significance of ToM and other social cognition deficits in emerging psychotic
disorders.
4.3. Methodological considerations

The two main strengths of the study were that i) a broad battery of assessments was used to investigate various social cognitive domains, and ii) a priori identified confounders were adjusted for to address any potential relationship between the groups and social cognitive functioning, most notably IQ. All analyses were adjusted for multiple testing. Very few of the UHR individuals (only 10%) were receiving antipsychotic medication.

There were limitations to the study. Firstly, the relatively small number of participants in the three groups does raise the possibility of a Type II error for some of the social cognition tasks. Specifically, subtle deficits in the UHR group may be demonstrated in a larger sample. However, one of the aims of the study was to investigate the relative size of the deficit compared to FEP and the study suggests that deficits are generally more pronounced in FEP. Secondly, it is worth noting that although the study adjusted for IQ as a potential confounder, the measure of IQ was not identical in all groups, although the scores were highly correlated in those who completed both the NART and the WASI.

4.4. Clinical implications

Both FEP and UHR patients appear to have difficulties in social cognition. There are promising approaches to social cognition training in more established disorders (Roberts and Penn, 2009) and interventions to improve social cognitive deficits are being developed for individuals in the earlier stage of illness (Bartholomeusz et al., 2011). The present results provide some support for such initiatives.
References


Ogura (ed) Recent Advances in Early Intervention and Prevention in Psychiatric Disorders. Seiwa-Shoten, Tokyo, pp. 26-33.


CHAPTER 7

Article Two: Social cognition and neurocognition represent distinct constructs in first episode psychosis and control cohorts

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Authors: Alicia Papas, Linda Byrne, Andrew Thompson
Abstract

Aim: Social cognition is a broad concept and is thought to be multidimensional, however limited research exists regarding the interrelationships between identified domains. Furthermore, a moderate degree of overlap has been reported between social cognition and neurocognition in schizophrenia, yet whether this is the case for non-clinical groups is unknown. The aims of the study were to: i) establish whether emotion recognition, Theory of Mind (ToM), social knowledge and emotion perception represent independent domains of social cognition, ii) assess whether relationships between social cognition and neurocognition are similar between first episode psychosis (FEP) patients and healthy controls, and iii) determine how much variance neurocognitive variables account for in each of the social cognitive domains. Method: Six neurocognitive domains (as per the MATRICS Consensus Cognitive Battery) and four areas of social cognition were assessed in 40 FEP patients and 30 controls. Results: The social cognitive domains were not significantly related to each other in the control group, however ToM was associated with all other social cognitive domains in the FEP group. Neurocognition was more highly related to social cognition in the FEP group than the control group. In general, individual neurocognitive variables did not contribute substantial variance to the social cognitive constructs measured. Conclusions: Social cognition and neurocognition are themselves multidimensional, and represent distinct constructs in both FEP and control groups. Implications for remediation and rehabilitation in FEP are raised.
1. Introduction

It is well known that cognitive impairment is a feature of schizophrenia and psychotic disorders. Neurocognitive abilities such as attention, reasoning and problem solving, and learning and memory are affected across these disorders, and historically have been assessed using tasks containing emotionally-neutral information such as alphanumeric characters, symbols, or personally irrelevant content. The MATRICS Consensus Cognitive Battery (MCCB) contains various tasks of this nature and is considered the current gold standard in assessing cognition in schizophrenia research. Neurocognition is not only impaired, but has been identified as a predictor of social outcomes and reduced real-world functioning.

Social cognition, which differs to neurocognition in that it involves processing and interpretation of emotionally-laden information required for social interaction, has recently become a focus in schizophrenia research. Social cognition is comprised of several domains including emotion recognition, theory of mind (ToM), social/emotion perception, social knowledge, and attributional style. Deficits in social cognition have been extensively documented in schizophrenia and also linked to poor functional outcomes to a greater extent than neurocognition. Moreover, social cognition has been shown to exhibit mediatory effects between neurocognition and social functioning, suggesting representative domains may be interrelated.

In general, social cognition and neurocognition are considered to represent distinct constructs with some degree of overlap, however their interrelationships have not been
fully explored. Some studies have supported a distinction between the two dimensions in schizophrenia,\(^1\,\text{14-15}\) whereas others have reported associations,\(^1\,\text{16-18}\) A recent meta-analysis indicated that correlations between social cognitive and neurocognitive domains are usually in the moderate range,\(^1\,\text{19}\) thus it remains unclear whether social cognition and neurocognition are in fact separate entities, or different expressions of the same underlying construct.

A methodological issue in the existing literature possibly contributing to the heterogeneity in findings is that some studies have examined relationships between neurocognition and only one social cognitive domain (e.g. emotion recognition or theory of mind or social perception). This is problematic as social cognition is considered multidimensional\(^6\) and therefore possible interrelationships between domains should be investigated. Moreover, many studies have only used clinical populations without including a control group, which introduces the question as to whether differential patterns of relationships exist in clinical and non-clinical populations. One study\(^1\,\text{16}\) included a non-psychiatric control group in their investigation of attention and facial affect recognition in schizophrenia and found that the tasks representing the two domains were associated only in the patient group. The authors concluded that the different tasks may have tapped into distinct constructs in the control group, however the association between the tasks in the schizophrenia group task may have been indicative of a unitary process of impairment rather than separable deficits. This fits with the viewpoint that intact neurocognitive abilities are required for effective social cognition,\(^2\,\text{20-21}\) given that social cognition often involves potentially more complex processes.
The current study aimed to examine the interrelationships of four previously identified social cognitive domains, and their relatedness to six established neurocognitive domains (as per the MCCB) in a FEP sample. A control group was also included to investigate patterns of relationships between the groups. As the existing literature suggests social cognition is comprised of distinct domains, it was hypothesised that the four social cognitive domains (emotion recognition, ToM, social knowledge and emotion perception) assessed would be relatively unrelated to one another. It was further hypothesised that each of the social cognitive domains would be no more than moderately related to the neurocognitive functions assessed. An objective of the study was to examine the amount of variance in each of the social cognitive domains accounted for by each of the neurocognitive variables.

2. Method

2.1. Participants

All participants (N=70) were part of a larger study, in which social cognition was assessed across three groups; ultra-high-risk (UHR) of developing a psychotic disorder, first episode psychosis (FEP), and controls. All participants were aged 15-25 years, reflecting the age range of patients attending the mental health service (Orygen Youth Health) where the patients groups were recruited. Controls were sourced from the same geographical catchment area i.e. Western Melbourne metropolitan region. Neurocognition was thoroughly assessed in the FEP (N=40) and control (N=30) groups only, thus both neurocognitive and social cognitive data from these cohorts has been
analysed in the current paper. More detailed participant demographics and study inclusion/exclusion criteria have been previously reported\textsuperscript{22}.

2.2 Materials

\textit{2.2.1 Social cognition assessment measures}

\textbf{Emotion recognition}. Emotion recognition was measured by the Diagnostic Analysis of Nonverbal Accuracy-2 (DANVA-2).\textsuperscript{23} Both components (facial recognition and paralanguage) of the adult version of the DANVA-2 were administered on a laptop computer. The number of errors on each component was combined to yield an emotion recognition total error score.

\textbf{Theory of mind (ToM)}. Two separate tasks were administered to assess ToM: i) Hinting Task\textsuperscript{24} and ii) Visual Jokes task.\textsuperscript{25} The Hinting Task is verbal in nature and assesses one's ability to decipher actual intentions behind indirect speech. It contains 10 items, with a possible maximum score out of 20. The Visual Jokes task is non-verbal and requires interpretation of humour in cartoon illustrations. Responses to 20 jokes were given a ‘mentalising’ score between 0 and 3 (maximum score of 60), which reflected the participant’s ToM level. Theoretically, both the Hinting task and Visual Jokes are measures of ToM,\textsuperscript{24,25} thus the scores from each of these tasks were transformed into $z$-scores and combined to produce an overall ToM variable. It is worth noting that the correlation between the two ToM tasks was only significant for the FEP sample ($r = .459$, $p = .011$) and not for controls ($r = .300$, $p = .064$).

\textbf{Emotion perception}. The Managing Emotions module of the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT)\textsuperscript{26} is documented as a task of “managing emotions”
or emotion regulation. It is the only social cognition task included in the MATRICS Consensus Cognitive Battery (MCCB; administered in its entirety in the current study) however it has been classified more specifically as an emotion perception task here and elsewhere,\textsuperscript{21} as it requires processing of social information and an awareness of emotional states in hypothetical real-life scenarios.

**Social knowledge.** Social knowledge was assessed by the Social Component Sequencing Task – Revised (SCST-R).\textsuperscript{27} The task contained 12 common social scenarios, each of which comprised a series of actions that were displayed in writing on laminated cards. Scenarios were either “short” (six actions) or “long” (nine actions). Participants were advised of each scenario via a “header” card, and then presented with the series of cards from left to right in a predetermined incorrect order. Participants were asked to sequence the social situation by sorting the cards into the correct order as quickly as possible. Two of the original scenarios were replaced with more age and culture-appropriate situations to reflect the demographic of the study group. Some actions in the original task were also slightly modified for this reason (the first author can be contacted directly for details). The task contained an equal number of short and long conditions, which were counterbalanced, to control for order effects. Mean “juxtaposition scores” were determined for each sequence, that is, the number of cards correctly juxtaposed to neighboring cards divided by the total possible correct juxtapositions.

### 2.2.2 Neurocognition assessment measures

Nine neurocognitive tasks were administered as per the MCCB (MATRICS Assessment Inc). The MCCB includes the following neurocognitive tasks: Trail Making Test – Trails A (TMT-A), Brief Assessment of Cognition in Schizophrenia – Symbol Coding (BACS-SC),
Category Fluency: Animal Naming, Continuous Performance Test – Identical Pairs (CPT-IP), Wechsler Memory Scale-III: Spatial Span (SS), Letter-Number Sequencing (LNS), Hopkins Verbal Learning Test – Revised (HVLT-R), Brief Visuospatial Memory Test – Revised (BVMT-R), and the Neuropsychological Assessment Battery (NAB): Mazes. A description of the tasks can be viewed online at www.matricsinc.org. Six neurocognitive domains are assessed by the MCCB; speed of processing, attention/vigilance, working memory, verbal learning, visual learning, and reasoning and problem solving.³

Results from the nine tasks were used to generate six overall neurocognitive domain scores as follows: BACS-SC, TMT-A and Category Fluency scores were transformed into z-scores and then combined to produce the Processing Speed domain score; LNS and SS overall scores were also transformed into z-scores and combined to form the Working Memory domain score; the total number of correct word recalls across three learning trials on the HVLT-R corresponded to the Verbal Learning domain; the total score acquired across three learning trials on the BVMT-R reflected the Visual Learning domain; the total raw score on the NAB Mazes reflected the Reasoning and Problem Solving domain; and overall performance on the CPT-IP across all three conditions (two, three, and four-digit numbers) corresponded to the attention/vigilance domain.

2.2.3 Pre-morbid IQ assessment

The National Adult Reading Test (NART)²⁸ was administered to obtain a premorbid IQ estimate. In instances where less than 10 words were correctly read aloud (i.e. the minimum number of items required to calculate an IQ estimate), the Schonell Reading
Test\textsuperscript{29} was administered. There was no difference in IQ estimates ($F=.129$, $p=.721$) between patients (mean = 106.39, SD = 10.62) and controls (mean= 107.30, SD= 10.80).

2.3 Procedure

Participants were administered all social cognition and neurocognition tasks within the same day where possible, with some assessments split over two days due to time constraints. The first author administered all social cognition tasks to each participant and completed most MCCB assessments. A research assistant trained in administering the MCCB conducted some of these assessments for the FEP group.

2.4 Statistical analyses

Independent samples $t$-tests were conducted to compare the groups on all measures of social cognition and neurocognition for descriptive purposes. Given multiple comparisons and the relatively low number of participants in each group, a more conservative $\alpha$ value ($\leq .01$) was selected as to minimise Type I error.

\textit{2.4.1 Interrelationships between social cognitive domains}

Bivariate correlations examined whether the four social cognition domains were related or independent within the sample groups. Due to different patterns of associations between the groups, subsequent regression analyses were conducted separately for patients and controls.
2.4.2 Degree of overlap between social cognition and neurocognition

Four linear multiple regressions were conducted for each group, with each social cognitive domain acting as the dependent variable in each model. The six neurocognitive domains were entered simultaneously as independent variables. This determined if “neurocognition” was related to social cognition and how much variance neurocognition accounted for in each social cognitive domain.

A further series of regressions were performed in which each of the six independent variables were removed sequentially (and re-entered after each regression was conducted), creating six models for the social cognitive domain in question. The $R^2$ of each model (less one neurocognitive variable) was then subtracted from the $R^2$ of the regression model containing all six neurocognitive variables. This computed value was indicative of the unique variance of the neurocognitive domain excluded from each model.

3. Results

Group comparisons on task performance revealed patients performed significantly worse than controls across three of the four social cognitive domains assessed (for degree of social cognitive impairment see an earlier study\textsuperscript{22}). In terms of neurocognitive functioning, patients were no different to controls except for performance on one of the processing speed measures (BACS-SC). All mean scores and standard deviations, with group comparisons are summarised in Table 1.
Table 1. Neurocognitive and social cognitive tasks: Group comparisons (Independent samples t-tests)

<table>
<thead>
<tr>
<th></th>
<th>FEP mean (SD)</th>
<th>Controls mean (SD)</th>
<th>t value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Social Cognition</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Emotion Recognition</em></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Facial affect error score</td>
<td>6.33 (3.08)</td>
<td>4.50 (2.01)</td>
<td>2.99**</td>
</tr>
<tr>
<td>Paralanguage error score</td>
<td>7.37 (2.54)</td>
<td>5.47 (2.67)</td>
<td>3.04**</td>
</tr>
<tr>
<td>Combined error score</td>
<td>13.70 (4.43)</td>
<td>9.97 (3.66)</td>
<td>3.75**</td>
</tr>
<tr>
<td><em>Theory of Mind (ToM)</em></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hinting Task total score</td>
<td>15.70 (3.26)</td>
<td>18.00 (1.15)</td>
<td>-4.14**†</td>
</tr>
<tr>
<td>Visual Jokes overall score</td>
<td>34.87 (5.49)</td>
<td>39.43 (5.72)</td>
<td>-3.36**</td>
</tr>
<tr>
<td>ToM composite</td>
<td>-0.32 (0.84)</td>
<td>0.45 (0.60)</td>
<td>-4.28**</td>
</tr>
<tr>
<td><em>Emotion Perception</em></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>MSCEIT overall score</td>
<td>85.79 (9.70)</td>
<td>92.02 (10.38)</td>
<td>-2.58*</td>
</tr>
<tr>
<td><em>Social Knowledge</em></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SCST-R overall score</td>
<td>0.83 (0.10)</td>
<td>0.88 (0.06)</td>
<td>-2.31</td>
</tr>
<tr>
<td><strong>Neurocognition</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td><em>Processing Speed</em></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TMT-A (time in seconds)</td>
<td>28.13 (9.41)</td>
<td>25.00 (7.05)</td>
<td>1.53</td>
</tr>
<tr>
<td>BACS-SC total score</td>
<td>54.85 (8.27)</td>
<td>61.03 (10.55)</td>
<td>-2.75*</td>
</tr>
<tr>
<td>Category Fluency total score</td>
<td>22.60 (4.33)</td>
<td>24.83 (5.19)</td>
<td>-1.96</td>
</tr>
<tr>
<td>Processing Speed composite</td>
<td>-0.10 (0.51)</td>
<td>0.14 (0.52)</td>
<td>-1.99</td>
</tr>
<tr>
<td><em>Working Memory</em></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Letter-Number Sequencing</td>
<td>14.60 (2.69)</td>
<td>15.63 (2.80)</td>
<td>-1.56</td>
</tr>
<tr>
<td>Spatial Span</td>
<td>17.13 (2.60)</td>
<td>17.80 (2.30)</td>
<td>-1.13</td>
</tr>
<tr>
<td>Working Memory composite</td>
<td>-0.14 (0.90)</td>
<td>0.18 (0.82)</td>
<td>-1.54</td>
</tr>
<tr>
<td><em>Verbal Learning</em></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HVLT-R (total trials 1-3)</td>
<td>25.05 (4.71)</td>
<td>27.50 (3.46)</td>
<td>-2.40</td>
</tr>
<tr>
<td><em>Visual Learning</em></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BVMT-R (total trials 1-3)</td>
<td>26.63 (6.80)</td>
<td>29.17 (4.89)</td>
<td>-1.74</td>
</tr>
<tr>
<td><em>Reasoning &amp; Problem Solving</em></td>
<td></td>
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<tr>
<td>NAB Mazes total score</td>
<td>22.83 (3.06)</td>
<td>23.97 (2.28)</td>
<td>-1.72</td>
</tr>
<tr>
<td><em>Attention/Vigilance</em></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>CPT-IP (mean D-Prime value)</td>
<td>2.39 (0.70)</td>
<td>2.55 (0.59)</td>
<td>-1.05</td>
</tr>
</tbody>
</table>

* p ≤ 0.01  ** p ≤ 0.001
† Levene’s test significant (equal variances not assumed)

The relationships between the four social cognition domains were generally different for patient and control groups. Regarding the FEP group alone, Pearson’s correlations showed that ToM was significantly related to all other social cognitive domains (emotion recognition, r = -.502, p = .001; social knowledge r = .498, p = .001; emotion perception r = .370, p = .020). None of the social cognitive domains were significantly related to each other in the control group.
For each group, four standard multiple regressions were conducted to assess the degree of overlap between neurocognition and social cognition. In each regression model, the dependent variable was one of the four social cognitive domains, whereas the six neurocognitive domains were independent variables (see Table 2 for a summary of the analyses). Emotion recognition and social knowledge were significantly predicted by neurocognition for the FEP group. The amount of variance accounted for by neurocognition in each of these social cognitive domains was approximately 37-38% ($p \leq 0.01$). Neurocognition did not account for a significant amount of variance in either ToM or emotion perception, although shared variance values were 24% and 17%, respectively. Regarding the control group, neurocognition accounted for 41% of the variance in ToM ($p < 0.05$), with all other social cognitive domains sharing unaccounted for by neurocognition.

<table>
<thead>
<tr>
<th></th>
<th>R²</th>
<th>F value</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emotion Recognition</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FEP</td>
<td>0.38</td>
<td>3.32</td>
<td>0.01</td>
</tr>
<tr>
<td>Control</td>
<td>0.20</td>
<td>0.96</td>
<td>0.47</td>
</tr>
<tr>
<td>Theory of Mind</td>
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</tr>
<tr>
<td>FEP</td>
<td>0.24</td>
<td>1.67</td>
<td>0.16</td>
</tr>
<tr>
<td>Control</td>
<td>0.42</td>
<td>2.71</td>
<td>0.04</td>
</tr>
<tr>
<td>Social Knowledge</td>
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<tr>
<td>FEP</td>
<td>0.37</td>
<td>3.28</td>
<td>0.01</td>
</tr>
<tr>
<td>Control</td>
<td>0.20</td>
<td>0.98</td>
<td>0.46</td>
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<td>Emotion Perception</td>
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<tr>
<td>FEP</td>
<td>0.17</td>
<td>1.12</td>
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</tr>
<tr>
<td>Control</td>
<td>0.13</td>
<td>0.56</td>
<td>0.76</td>
</tr>
</tbody>
</table>

The six neurocognitive domains were examined independently in terms of their unique relatedness to each social cognition construct. The specific variances ($R^2$) accounted for
by each of the neurocognitive domains are displayed in Table 3. In general, the $R^2$ values were small to negligible (i.e. $\leq .111$) except for those associated with social knowledge in the control group, which were all between .247 and .299.

Table 3. Secondary regression analyses: Unique contributions ($R^2$) of each neurocognitive variable on social cognition

<table>
<thead>
<tr>
<th></th>
<th>PS</th>
<th>Verbal Learning</th>
<th>WM</th>
<th>R&amp;P</th>
<th>Visual Learning</th>
<th>Attention/Vigilance</th>
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<tr>
<td>FEP</td>
<td>0.01</td>
<td>0.11</td>
<td>0.06</td>
<td>0.08</td>
<td>$\leq 0.001$</td>
<td>0.04</td>
</tr>
<tr>
<td>Control</td>
<td>0.05</td>
<td>0.03</td>
<td>0.01</td>
<td>0.01</td>
<td>0.01</td>
<td>$\leq 0.001$</td>
</tr>
<tr>
<td>Theory of Mind</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FEP</td>
<td>$\leq 0.001$</td>
<td>0.09</td>
<td>0.02</td>
<td>0.04</td>
<td>0.02</td>
<td>$\leq 0.001$</td>
</tr>
<tr>
<td>Control</td>
<td>0.02</td>
<td>0.10</td>
<td>0.01</td>
<td>0.01</td>
<td>$\leq 0.001$</td>
<td>0.01</td>
</tr>
<tr>
<td>Social Knowledge</td>
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<td></td>
</tr>
<tr>
<td>FEP</td>
<td>0.02</td>
<td>0.01</td>
<td>0.06</td>
<td>0.04</td>
<td>0.10</td>
<td>0.08</td>
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<tr>
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<td>0.26</td>
<td>0.25</td>
<td>0.30</td>
<td>0.25</td>
<td>0.25</td>
<td>0.27</td>
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<tr>
<td>Emotion Perception</td>
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</tr>
<tr>
<td>FEP</td>
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<td>$\leq 0.001$</td>
<td>0.01</td>
<td>0.03</td>
<td>0.11</td>
<td>0.05</td>
</tr>
<tr>
<td>Control</td>
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<td>0.02</td>
<td>0.02</td>
<td>$\leq 0.001$</td>
<td>0.02</td>
<td>$\leq 0.001$</td>
</tr>
</tbody>
</table>

Abbreviations: PS, Processing Speed; WM, Working Memory; R&P, Reasoning and Problem Solving

4. Discussion

The main aim of the paper was to establish whether social cognition and neurocognition are separate constructs. Initially, the interrelationships between four areas of social cognition (emotion recognition, ToM, social knowledge, emotion perception) were examined to determine if they are indeed independent of one another. Differential relationships were revealed between the patient and control groups; none of the social cognitive domains were related to each other in the control group, however ToM was associated with all other social cognition domains in the FEP group. Relationships
between neurocognition and individual social cognitive domains were also found to be
different for FEP and control groups. Regression analyses showed neurocognition was
not predictive of social cognition for controls, yet was significantly associated with
certain social cognitive domains in the FEP group. Where specific neurocognitive
functions were examined individually to assess their unique contribution to each of the
social cognitive domains, variances were generally small and thus all domains were
considered distinct.

The fact that the four social cognitive domains were unrelated to each other in the
control group supports the idea that social cognition is multidimensional and that the
domains described in the literature\(^8\) represent independent functions. The finding that
ToM was at least moderately associated with the other social cognitive domains in the
FEP group may indicate that inferring the mental states of others is a highly complex
skill for people with a psychotic illness and more basic social cognitive processes (e.g.
emotion recognition) need to be intact. The results of the larger study\(^22\) associated with
the current investigation showed a generalised social cognitive deficit for FEP patients
compared to controls, suggesting a need to target multiple domains in remediation
efforts.

Overall neurocognition did not contribute significantly to the variance in social
cognition in the control group, except for 41\% in ToM however the associated statistical
significance level (i.e. >.01) was with not considered strong given the sample size. In
the FEP group, neurocognition accounted for 37-38\% of the variance in two of the four
social cognitive domains, namely emotion recognition and social knowledge. Similarly, a recent study found neurocogniton to account for 34% of the variance in affect recognition in a sample of schizophrenia and schizoaffective patients. It is unclear as to why neurocognition was not associated with ToM and emotion perception in the current study, as Fanning and colleagues also found a significant overlap between those domains and neurocognition. Given that neurocognition accounted for a notable 17-24% of the variance in these other two domains, perhaps a larger sample size may have led to statistically significant contributions. These results support the notion that intact neurocognition may play a role in social cognitive ability.

Examining unique variance by neurocognitive domain-type, only small or trivial contributions (0-11%) were found. However, there was an exception in the control group; all of the six neurocognitive domains contributed 25-30% of the variance in social knowledge. This overlap suggests the SCST-R task required neurocognitive input and therefore may not be a principally social cognition measure. In terms of the nature of the SCST-R, it is similar to the Picture Arrangement subtest of the WAIS-III, which is a perceptual reasoning task. The SCST-R and Picture Arrangement tasks are comparable in that both involve sequencing of information, requiring higher order neurocognitive abilities e.g. executive functioning. The small degree of overlap (< 10%) between social knowledge and neurocognition in the FEP group indicates the SCST-R was just as complex for patients as the other more difficult social cognitive tasks (i.e. ToM), thus individuals having experienced an episode of psychosis may find social cognition skills, like other cognitive abilities, compromised in general.
Given the low number of participants in each group, it is acknowledged that data analysis by group-type was a limitation to the study in terms of Type I error risk. However, the authors believed it was important to examine the data in this way, rather than collectively, as initial correlations between the tasks were different for each group. This limitation was accounted for by using a more conservative $\alpha$-value of .01, as opposed to the conventional .05 level commonly applied. The FEP group results provide valuable information that potentially informs attempts to develop suitable interventions for cognitive remediation in early psychosis populations. As suggested by Fanning and colleagues,$^{21}$ social cognition domain-type may need to be considered when interventions are being developed as targeting certain neurocognitive deficits via remediation may contribute to better social cognition outcomes. Cognitive interventions aimed at both neurocognitive and social cognitive abilities, e.g. Cognitive Enhancement Therapy (CET), have already proven effective in improving skills in early schizophrenia patients, especially with regard to social cognition.$^{32}$

In summary, this study supports the notion that social cognition is multidimensional and represents distinct domains as previously identified in the literature. Although speculative, the results suggest that ToM is particularly complex for FEP patients and individuals may be more likely to rely on other social cognition skills in order to mentalise effectively compared to non-clinical individuals. Furthermore, neurocognition and social cognition represent distinct constructs, as evidenced by only a minor degree of shared variance between specific neurocognitive functions and social cognitive domains. On a clinical level, it may be important to address social cognition deficits partly via remediation of neurocognitive functions based on the domain being targeted.
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CHAPTER 8

Article Three: The relationship between symptomatology and social cognition in first episode psychosis

Submitted to Psychiatry Research

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Abstract

The exact relationship between social cognition and psychotic symptoms is unclear. Generally, negative and disorganised symptoms have been linked to poorer social cognition in schizophrenia, with limited research having been conducted using First Episode Psychosis (FEP) cohorts. Moreover, there has been variability in symptom classification across studies. Using a three-factor symptom model (positive, negative and disorganised), the main objective of the study was to investigate whether negative and disorganised symptom clusters are linked to social cognitive deficits in FEP. A secondary objective was to examine the relationship of individual symptoms and social cognition. The Positive and Negative Syndrome Scale (PANSS) was used to assess clinical symptoms in a sample of 40 FEP outpatients aged 15-25 years. Four domains of social cognition were assessed; theory of mind (ToM), emotion recognition, social perception, and social knowledge. Bivariate correlations and multiple regressions were applied to investigate the relationships between symptoms and social cognition. Disorganisation was the only symptom cluster related to deficits in social cognition. On an individual symptom level, various negative and disorganised items were associated with poorer social cognition, with difficulty in abstract thinking predictive of ToM and emotion recognition, and stereotyped thinking predictive of social perception in FEP.
1. Introduction

Social cognition refers to the mental processing and interpretation of social and emotional information. It is significantly poorer in individuals with both established schizophrenia (Penn et al., 2008) and first-episode psychosis (FEP) (Addington et al., 2006; Bertrand et al., 2007; Edwards et al., 2001; Inoue et al., 2006; Thompson et al., 2012) compared to non-clinical control groups. This has real world functioning implications for those with psychotic disorders, as social cognition has been identified as a unique predictor of functional outcome (Sergi et al., 2007). Despite demonstrated deficits in overall social cognitive functioning across schizophrenia-spectrum disorders, and across all illness stages from early to late, one issue that remains unclear is whether certain psychotic symptoms are associated with poorer social cognition in FEP. This is an important question to investigate as researchers have proposed that social cognition deficits may be a particular risk factor for the development of psychotic symptoms (Frith & Corcoran, 1996).

The exact areas of cognition encompassing “social cognition” are the focus of continuing debate in the literature, however four to five separate domains have been typically proposed by a number of authors. These are: theory of mind (ToM), emotion recognition, social perception, social knowledge, and attributional bias (Couture et al., 2006; Green & Horan, 2010; Green et al., 2005). Psychotic symptoms have historically been classed as either ‘positive’ or ‘negative’ (Crow, 1982), however heterogeneity also exists within these broad classifications. Researchers have since proposed symptom paradigms containing three or more factors as better fits than the positive-negative model (Bilder et al., 1985; Drake et al., 2003; Emsley et al., 2003; Liddle, 1987). A three-
factor model of positive, negative and disorganised symptomatology has been
commonly used to examine the relationship between symptom-type and cognition and
behaviour (Cameron et al., 2002; Smith et al., 2002), whereby positive symptoms
typically include hallucinations and delusions, negative symptoms reflect phenomena
such as social withdrawal and blunted affect, and disorganisation represents disordered
thought and odd behaviour (Liddle, 1987).

Regardless of the whether a two or multi-symptom model is used to examine the
relationship between psychopathology and social cognition, the literature has certain
methodological problems. In particular, there are differences in the classification of
specific individual symptoms. According to some researchers, difficulty in abstract
thinking is classified as disorganised psychopathology (Emsley et al., 2003; Liddle,
1987) rather than negative symptomatology as per the widely used negative subscale of
Positive and Negative Syndrome Scale (PANSS; Kay et al., 1989). Stereotyped thinking, a
negative item on the PANSS, has been referred to as a positive factor symptom in
another study (Drake et al., 2003). Emsley and colleagues (2003) found stereotyped
thinking, similar to lack of abstraction, corresponded to the disorganised (or
“cognitive”) factor. Furthermore, whilst stereotyped thinking has not been included in
any of the three symptom clusters in certain studies (e.g. Cameron et al., 2002) it has
been identified as a significant predictor of social cognition in a sample of FEP and
schizophrenia patients (Piskulic & Addington, 2011).
The findings of existing research pertaining to psychosis symptomatology and social cognition in schizophrenia-spectrum disorders have been inconsistent. As an overview, negative symptoms have been linked to deficits in social perception (Sergi et al., 2007), ToM (Bora et al., 2008; Shean & Meyer, 2009; Stratta et al., 2011) and emotion/affect recognition (Johnston et al., 2010), however some studies failed to find any significant associations across these domains (Bertrand et al., 2007; Mancuso et al., 2011). With regard to positive symptoms, deficits in ToM have been linked to paranoia (Corcoran et al., 1995; Frith & Corcoran, 1996; Pickup & Frith, 2001), but the findings are far from universal (Drury et al., 1998; Greig et al., 2004; Safarti & Hardy-Baylé, 1999). Hall and colleagues (2004) also reported an association between higher levels of positive symptoms and impaired ability on a facial affect recognition task. Despite significant associations between positive symptoms and social cognition, these findings should be considered in light of statistical and methodological factors, such as relatively small sample sizes and effect sizes (Hall et al., 2004; Pickup & Frith, 2001). The only social cognitive domain that appears to be consistently related to paranoia and delusional beliefs is attributional style, with an externalising bias linked to such positive symptomatology (e.g. Fornells-Ambrojo & Garety, 2009).

Disorganised symptoms have been the least researched in terms of the relationship between psychotic symptomatology and social cognition, especially in FEP. Studies have shown ToM deficits associated with the disorganised symptom of “thought disorder” (Frith & Corcoran, 1996), and social perception difficulties related to the specific item of “conceptual disorganisation” as measured by the PANSS (Toomey et al., 2002).
Inconsistency in the findings may be influenced by methodological differences with regard to the assessment and categorisation of symptoms, but also a result of considerable variation in social cognition task selection across studies. Many different tasks have been used to assess a particular domain of social cognition across studies, leading to difficulty in generalising the findings (Green et al., 2005). In addition, most studies have only examined one or two social cognitive domains, rather than the four or five identified areas, thus it has been difficult to conclude if deficits are global or more specific to certain domains.

The overall aim of the current study was to examine the relationship between symptomatology and social cognition, taking into account inconsistencies in symptom classification and task variation in the current literature. Based on Liddle’s (1987) symptom dimensions, the main objective was to determine whether positive, negative and/or disorganised symptom clusters are linked to specific or global social cognitive deficits in FEP. It was hypothesized that negative and disorganised psychopathology would be associated with poorer social cognition across all measured domains, with little or no relation to positive symptoms. Based on variation in the literature regarding classification of specific PANSS items, a secondary objective was to investigate the relationship between social cognition and individual symptoms, including “stereotyped thinking” due to the cognitive nature of this specific symptom and previously reported significant association with social cognitive task performance (Piskulic & Addington, 2011).
2. Methods

2.1 Participants

Participants were recruited from Orygen Youth Health, a public mental health service for 15-25 year olds that provides assessment and intervention to individuals residing in the Western region of Melbourne, Australia. Orygen Youth Health is comprised of various specialized services, with participants of this study recruited specifically from the Early Psychosis Prevention and Intervention Clinic (EPPIC). The EPPIC service (for a thorough description see Edwards, Harris et al., 2002) provides up to two years of treatment for individuals with a diagnosed first episode psychotic disorder, based on evidence of psychotic symptoms for at least one week. Patients were eligible to participate in the study irrespective of illness phase and duration of treatment at EPPIC. The sample consisted of 40 FEP patients (65% male and 35% female), with a mean age of 20.5 years. All individuals were in a non-acute, recovery phase of illness at the time of assessment. Relevant institutional ethics approvals were obtained prior to the commencement of the study and all participants provided written informed consent (third party consent from a parent/guardian if the participant was a minor) to participate.

2.2 Exclusion criteria

Individuals with a known intellectual disability, or those who demonstrated an IQ less than 70 as per performance on the National Adult Reading Test (Nelson, 1982) during the assessment, were excluded from the study. Any individual receiving treatment for a significant neurological disorder such as epilepsy was also excluded. Other exclusion
criteria included intoxication with illicit drugs or alcohol during testing, and poor
English language skills due to the verbal demands of the test battery.

2.3 Measures

2.3.1 Psychopathology

Symptoms were assessed via interview using the Positive and Negative Symptom Scale
(PANSS; Kay et al., 1989), a commonly used 30-item clinical measure that indicates
severity of psychosis-spectrum and other general mental health symptomatology. Three
symptom sub-scores (positive, negative and disorganised) were computed using 16 of
the PANSS items; see Cameron and colleagues (2002) for a description of the rationale
for item selection. The items corresponding to each of the symptom clusters were:
positive (delusions, hallucinations, grandiosity, suspiciousness, unusual thought
content), negative (blunted affect, poor rapport, emotional withdrawal, motor
retardation, lack of spontaneity and flow of conversation, passive social withdrawal,
active social avoidance), disorganised (difficulty in abstract thinking, disorientation,
poor attention, conceptual disorganisation).

2.3.2 Social cognition

A number of tasks were administered to assess the four social cognitive domains of
emotion recognition, theory of mind, social perception and social knowledge. A
description of these tasks is provided below, according to the domain measured.

2.3.2.1 Emotion recognition. The Diagnostic Analysis of Nonverbal
Accuracy-2 (DANVA-2; Nowicki & Duke, 1994) was used to assess emotion recognition.
Both components of the adult version of the DANVA-2, i) facial expressions (DANVA-2-AF) and ii) paralanguage (DANVA-2-AP), were administered on a laptop computer. The DANVA-2-AF comprises 24 colour photographs of an equal number of happy, sad, angry and fearful faces of high and low intensities (Nowicki & Carton, 1993). Following a brief display period, individual are required to make a forced-choice response of one of the four emotions. The DANVA-2-AP consists of 24 audio recordings of professional actors saying the neutral phrase, “I’m going out of the room now and I’ll be back later”, with an intended tone of voice of happy, sad, angry or fearful. As with the faces component of the task, there are an equal number of happy, sad, angry and fearful voices of high and low intensities.

2.3.2.2 Theory of mind (ToM). Two separate ToM tasks were administered; the Hinting Task (Corcoran et al., 1995) and the Visual Jokes task (Corcoran et al., 1997). The Hinting Task is verbal in nature and assesses one’s ability to insinuate actual intentions behind indirect speech. Ten short passages, each portraying an interaction between two characters, are read aloud to participants one at a time. All passages end with one of the characters dropping an obvious hint, and participants are then asked what the character really meant. Participants are awarded two points for a correct answer, however if an incorrect response is given, another obvious hint is provided and the participant is asked what the character wants the other to do. A correct answer at this stage results in one point. Alternatively, the participant would receive a score of zero for an incorrect response.

The Visual Jokes task is presented visually and involves interpretation of humour in cartoon illustrations. A set of 10 ‘ToM’ jokes and 10 ‘physical’ jokes (i.e. based on
physical events) were shown to participants via computer in a randomised order. Display time was unlimited and participants’ responses were recorded on a portable voice-recorder. In addition to an explanation regarding the humour of the cartoon, participants were asked to give each individual joke a funniness score (on a 1-10 scale). Answers were given a “mentalising” score between 0 and 3, which reflected the participant’s ToM level. A score of 0 reflected that the participant did not understand the joke at all, a score of 1 corresponded to a physical explanation only, a score of 2 indicated that the joke was interpreted without specific reference to mental states however could be inferred, and a score of 3 was given if the answer included an accurate depiction of the character(s) mental states. Scoring was achieved via consensus ratings between the research investigators (AP, CB, AT).

2.3.2.3 Social perception. The Managing Emotions module (branch 4) of the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT; Mayer et al., 2002) was administered to assess social perception. The task was included as it is the only social cognition task included in the MATRICS cognitive consensus battery. Whilst originally documented as a task of “managing emotions”, the MSCEIT has been classified as a social perception task in the current study as it requires a similar cognitive skill set (for example, interpretation of situational cues and thinking about social outcomes). A series of vignettes are read out loud to participants, with a number of multiple-choice style questions asked after each story. Each response is given a score of 1-5 and entered into a software program (available from Multi-Health Systems, Inc, Toronto, Ontario, Canada), which then generates an overall score similar to an IQ result (mean = 100, SD = 15) based on a large U.S. normative sample.
Social knowledge was assessed by the Social Component Sequencing Task – Revised (SCST-R; Corrigan & Addis, 1995). The task contains 12 common social scenarios, each of which is comprised of a series of actions. Scenarios are either "short" (six actions) or "long" (nine actions), with each action identified in writing on laminated card. Participants are advised of each scenario via a "header" card, then presented with the series of cards from left to right in the incorrect order. Participants are asked to sequence the social situation by sorting the cards into the correct order as quickly as possible (the task is timed via stopwatch). Two of the original scenarios were replaced with more age and culture-appropriate situations to reflect the demographic of study group and some actions in the original task were also slightly modified for this reason. The task contains an equal number of short and long conditions, which were counterbalanced, to control for order effects. Mean "juxtaposition scores" were determined for each sequence, that is, the number of cards correctly juxtaposed to neighbouring cards divided by the total possible correct juxtapositions. For example, if a nine item scenario was ordered as C-B-D-E-A-I-F-G-H, three of eight neighbouring pairs were correctly sequenced (D-E, F-G, and G-H), equating to a score of 0.375 (Corrigan & Addis, 1995).

2.3.3 Pre-morbid IQ

The National Adult Reading Test (NART; Nelson, 1982) was administered to obtain a premorbid IQ estimate (mean = 106.39, SD = 10.62),
2.4 Procedure

Participants undertook a clinical interview, which included demographic questions and the PANSS. Following the psychopathology assessment, an approximate measure of IQ was obtained via completion of the NART. Participants then completed the battery of social cognition tasks. All assessments were conducted by the first author, except for 11 psychopathology interviews that were conducted by a well-trained research assistant. Both raters had considerable experience administering the PANSS and undertook reliability checks throughout the study period, including consensus ratings on training videos.

2.5 Statistical analyses

2.5.1 Primary analysis

Pearson’s bivariate correlations were used to examine the relationship between each of three symptom cluster scores (positive, negative and disorganised) and social cognition. All social cognition outcomes were treated as continuous variables and included: number of errors on the DANVA-2, total raw score on the Hinting Task, total mentalising score on the Visual Jokes task, overall MSCEIT score, and mean juxtaposition score on the SCST-R. The individual mean juxtaposition scores for the short and long conditions of the SCST-R were also analysed separately, given differing levels of task complexity.

2.5.2 Secondary analysis

Pearson’s bivariate correlations were conducted to further delineate the relationship between the individual PANSS items comprising the three symptom domains and social
cognition, in addition to the specific negative symptom of “stereotyped thinking”, given recent findings of a unique contribution to social cognitive deficits (Piskulic & Addington, 2011). Where multiple symptoms correlated significantly with any given social cognition variable, these individual symptoms were then entered into standard multiple regression analyses as predictor variables, with social cognition outcome scores as dependent variables. Age and IQ were included as covariates in the regression analyses. All analyses were conducted using SPSS for Windows version 19.

3. Results

3.1 Psychopathology

The mean PANSS total score for the sample was 59 (SD = 14.80). The average severity of individual PANSS symptoms included in the current study was mild (<3). The maximum possible scores for each of the symptom clusters were: 4 for disorganisation, 7 for negative symptoms, and 5 for the positive factor. The mean scores for each of the symptom clusters were: disorganisation 1.86 (SD = 0.62), negative 1.89 (SD = 0.72) and positive 2.11 (SD = 0.97).

3.2 Primary analysis: Symptom clusters and social cognition

All 40 participants completed the five social cognitive tasks selected for use in this study. Data for one participant on the visual jokes task was excluded from the analysis due to a technical issue with the task stimulus.
Of the three symptom groups, only disorganisation correlated significantly with social cognition task performance. Specifically, disorganised symptoms were associated with i) performance on the MSCEIT ($r = -0.32$, $p = 0.042$) and ii) ability on the long sequences of the SCST-R ($r = -0.313$, $p = 0.049$).

3.3. Secondary analysis: Individual PANSS items and social cognition

A second correlational analysis revealed that five individual PANSS items (difficulty in abstract thinking, lack of spontaneity and flow of conversation, stereotyped thinking, poor attention, and motor retardation) were associated with poorer social cognitive task performance (see Table 1). Difficulty in abstract thinking was the only symptom linked to deficits on several social cognition outcomes, specifically: poorer mentalising ability on the Visual Jokes task, poorer performance on the Hinting Task, more errors on the paralanguage component of the DANVA-2, a lower score on the MSCEIT, and poorer performance on the SCST-R (with a stronger correlation when only performance on long sequences was analysed). The other four symptoms each correlated significantly with only one social cognition task. Stereotyped thinking and poor attention were both related to lower scores on the MSCEIT. Lack of spontaneity and flow of conversation was associated with poorer performance on the paralanguage component of the DANVA-2, whereas motor retardation was linked to lower scores on the Hinting Task.
Table 1. Pearson’s correlations (r values) between individual PANSS items and social cognition variables

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Lack of abstraction</th>
<th>Poor attention</th>
<th>Lack of spontaneity &amp; conversation flow</th>
<th>Motor retardation</th>
<th>Stereotyped thinking</th>
</tr>
</thead>
<tbody>
<tr>
<td>Visual Jokes</td>
<td>-0.519**</td>
<td>-0.054</td>
<td>-0.003</td>
<td>-0.022</td>
<td>-0.192</td>
</tr>
<tr>
<td>[mentalising]</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hinting Task</td>
<td>-0.490**</td>
<td>-0.187</td>
<td>-0.293</td>
<td>-0.359*</td>
<td>-0.029</td>
</tr>
<tr>
<td>DANVA-2-PL errors</td>
<td>0.326*</td>
<td>0.121</td>
<td>0.329*</td>
<td>0.187</td>
<td>-0.148</td>
</tr>
<tr>
<td>MSCEIT</td>
<td>-0.350*</td>
<td>-0.370*</td>
<td>-0.155</td>
<td>-0.171</td>
<td>-0.416**</td>
</tr>
<tr>
<td>SCST-R [overall performance]</td>
<td>-0.362*</td>
<td>-0.002</td>
<td>-0.075</td>
<td>-0.156</td>
<td>-0.162</td>
</tr>
<tr>
<td>SCST-R [long sequences only]</td>
<td>-0.455**</td>
<td>-0.146</td>
<td>-0.134</td>
<td>-0.110</td>
<td>-0.103</td>
</tr>
</tbody>
</table>

* significant at .05 level    ** significant at .01 level

The five symptoms were then entered into a standard multiple regression analysis (see Table 2) as predictor variables, with the social cognition tasks that were significantly related to more than one specific symptom as outcome variables, that is, Hinting Task, MSCEIT and DANVA-2. Total errors on the DANVA-2-AF and DANVA-2-PL were combined in order to acquire an overall emotion recognition variable.

Difficultly in abstract thinking, lack of spontaneity and flow of conversation, motor retardation, poor attention and stereotyped thinking were good predictors of performance on the Hinting Task and MSCEIT. The five symptoms predicted 37.4% of the variance in the Hinting Task and 34.6% in the MSCEIT. With regard to the DANVA-2,
the symptoms did not predict performance, however they still accounted for 26.5% of the variance in emotion recognition. At the individual symptom level, difficulty in abstract thinking was a unique predictor of deficits on the Hinting Task and DANVA-2, whereas stereotyped thinking contributed to poorer MSCEIT performance.
<table>
<thead>
<tr>
<th>Table 2. Standard multiple regression for individuals symptoms as predictors of social cognition (adjusted for age and predicted IQ)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hinting Task</strong></td>
</tr>
<tr>
<td>Lack of abstraction</td>
</tr>
<tr>
<td>Lack of spontaneity</td>
</tr>
<tr>
<td>Motor retardation</td>
</tr>
<tr>
<td>Poor attention</td>
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<tr>
<td>Stereotyped Thinking</td>
</tr>
<tr>
<td><strong>MSCEIT</strong></td>
</tr>
<tr>
<td>Lack of abstraction</td>
</tr>
<tr>
<td>Lack of spontaneity</td>
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<tr>
<td>Motor retardation</td>
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<tr>
<td>Poor attention</td>
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<tr>
<td>Stereotyped Thinking</td>
</tr>
<tr>
<td><strong>DANVA-2 (total errors)</strong></td>
</tr>
<tr>
<td>Lack of abstraction</td>
</tr>
<tr>
<td>Lack of spontaneity</td>
</tr>
<tr>
<td>Motor retardation</td>
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<tr>
<td>Poor attention</td>
</tr>
<tr>
<td>Stereotyped Thinking</td>
</tr>
</tbody>
</table>

* significant at .05 level
4. Discussion

The aim of the study was to investigate whether symptom subtypes, in particular negative and disorganised, are associated with deficits in social cognition across various domains including theory of mind, emotion recognition, social perception, and social knowledge in FEP. A main finding was that disorganised psychopathology, based on Liddle’s (1987) symptom structure, was significantly related to deficits in social perception as per performance on the MSCEIT and poorer social knowledge when task complexity increased. These results are consistent with other studies that have found associations between disordered thinking and social cognition (e.g. Piskulin & Addington, 2011; Toomey et al., 2002; Frith & Corcoran, 1996).

As hypothesised, positive symptoms were not related to the social cognition domains assessed in the current study. However, it was an unexpected finding that the negative symptom domain was not associated with poorer social cognition. There is a general consensus in the literature that negative symptomatology is linked to social cognition deficits in psychotic disorders (e.g. Stratta et al., 2011; Johnston et al., 2010; Shean & Meyer, 2009; Bora et al., 2008; Sergi et al., 2007;), thus it was surprising that only the disorganised cluster related to poorer social cognition. However, when symptoms were assessed on an individual level, certain “negative” and “disorganised” symptoms were associated with deficits across all four social cognition domains assessed in the study. Furthermore, these symptoms (difficulty in abstract thinking, poor attention, lack of spontaneity and flow of conversation, motor retardation, stereotyped thinking) were collectively found to be predictors of ToM and social perception, specifically. These findings imply social cognition is a broad concept representing different domains that
require different skill sets, and it is the domains that perhaps necessitate more complex abilities or abstraction that are associated with certain negative/disorganised symptoms. The findings also suggest that the symptom clusters used in the study, which were identified using samples with predominantly established psychotic illness, are not necessarily applicable to FEP and therefore symptomatology classification may need to be revisited for these populations.

The most noteworthy finding was perhaps that two cognitive-based symptoms, that is, difficulty in abstract thinking and stereotyped thinking, were the only unique predictors of social cognition in the regression analyses. Lack of abstraction, classified as disorganised psychopathology in the current study, predicted both ToM and emotion recognition. Stereotyped thinking, classified as a negative symptom in the current study, accounted for a considerable amount of variance in social perception. These results provide empirical evidence for the proposal of Piskulic and Addington (2011) that it is typically negative symptoms reflecting obvious cognitive processes that are associated with social cognition. The nature of the association remains unclear, however it may be that these particular symptoms are consequences or direct expressions of social cognition problems.

There were a few limitations in the current study. Similar to other studies in this area, the sample size was relatively small. Secondly, attributional style, considered a distinct social cognitive domain (Penn et al., 2008) and previously associated with specific positive symptoms of paranoid thinking, was not investigated. However, all other social
cognitive domains as identified in the literature (Green & Horan, 2010) were examined. It is also worth mentioning that the severity of symptoms in this cohort was mild, as this may have had implications on the findings; it has previously been suggested that only moderate to severe negative symptoms impact on social cognition task performance (Mancuso et al., 2011).

Despite these limitations, the strength of the study is that the main social cognition domains identified in the literature were assessed. The majority of other studies tend to investigate only one or two domains, thus future research should aim to incorporate the four to five documented areas of social cognition in their assessments. This will enable further conclusions to be drawn regarding the nature of associations between certain symptoms and specific areas of social cognition. In terms of assessment and categorisation of psychosis symptomatology and its relationship with social cognition, studies should pay particular attention to negative and/or disorganised symptoms with a strong cognitive underpinning (e.g. lack of abstraction and stereotyped thinking) and use multi-dimensional models that include a specific cognitive factor (e.g. Emsley et al., 2003). The current results provide grounds for further factor-analytical studies using FEP or early psychosis samples, in order to determine a more conclusive symptom model for these populations.

5. Clinical Implications

In general, the findings indicate that FEP individuals with disorganised and negative psychopathology have poorer social cognition outcomes, particularly in situations
requiring more complex abilities such as abstraction and mentalising. It would be helpful to investigate this relationship further in order to better understand the formation of symptoms; longitudinal studies would provide insight into whether early social cognitive deficits are perhaps risk factors for these specific symptoms. In terms of treatment of social cognitive deficits, the findings suggest that FEP patients with disorganised/cognitive symptoms and perhaps more severe negative symptoms would be best targeted by psychosocial approaches such as the relatively new social cognition remediation therapies for psychotic disorders (e.g. Roberts & Penn, 2009). Such interventions could lead to improved real-world social and vocational functioning for these individuals.
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CHAPTER 9

Article Four: **Neurocognition, social cognition and psychopathology as predictors of social functioning in first episode psychosis**

*Submitted to Psychiatry and Clinical Neurosciences*

Authors: Alicia Papas, Andrew Thompson, Cali Bartholomeusz, Kelly Allott, Linda Byrne
Abstract

Aim: Various factors have been shown to impact on social functioning in schizophrenia, including neurocognition, social cognition and psychotic symptoms. However, the relative effect of each of these factors is unclear, especially in the early phase of illness. The purpose of this study was to investigate their contribution to functioning in first episode psychosis (FEP). Methods: Forty FEP outpatients were assessed. Six neurocognitive domains (as per the MATRICS Consensus Cognitive Battery) and four social cognitive domains (emotion recognition, theory of mind, emotion perception, social knowledge) were measured. Composite scores were computed for neurocognition and social cognition, which were both analysed as predictors of social functioning. The Positive and Negative Syndrome Scale (PANSS) was applied to assess symptom severity, with the total score also used as a predictor variable. Functioning was assessed by three measures previously used in adolescent and young adult populations. Results: Linear regression analyses revealed the overall PANSS score was the only factor significantly associated with functioning. Overall measures of neurocognition and social cognition were not associated with any of the functioning indices. Secondary analysis indicated that negative and positive symptoms, but not disorganised symptoms, significantly predicted various functional domains. Conclusion: This cross-sectional study emphasises the importance of psychotic symptoms on functioning in FEP.
1. Introduction

Poor social functioning is considered characteristic of schizophrenia and early psychosis. Social functioning refers to performance in daily activities that comprise a social component, e.g., family and peer interactions, occupational or educational roles, independent living, and leisure activities.\textsuperscript{1} Research has shown various factors are associated with impaired social functioning in schizophrenia, in particular, neurocognition,\textsuperscript{2-4} social cognition,\textsuperscript{5-8} and psychiatric symptoms.\textsuperscript{4,6,8-11} Whether one factor has a greater impact on social functioning than another remains unclear, especially in the early phase of illness.

Specific areas of neurocognition have been identified as predictive of social functioning in schizophrenia, e.g., immediate and secondary verbal memory, executive functioning, and attention/vigilance.\textsuperscript{2-4} Many studies have shown clear associations between neurocognitive impairment and poor functioning in both cross-sectional studies\textsuperscript{1-2,9,12} and reviews of longitudinal research.\textsuperscript{3} However, the vast majority of evidence has been derived from chronic schizophrenia samples. A recent review conducted by Allott and colleagues\textsuperscript{13} indicated that the relationship between neurocognition and functioning is less clear in early psychosis. Cognitive remediation programs aimed at improving neurocognition and consequently functioning have been developed (for a review see Wykes et al.\textsuperscript{14}), yet their effectiveness has been limited. These programs have been shown to improve performance on neurocognitive measures however gains have not usually translated to real-world functioning, which is likely due to neurocognitive deficits having generally accounted for only a moderate degree of variance in functional outcomes.\textsuperscript{2} It has therefore been important to investigate and identify other factors
contributing to poor social functioning in order to develop better targeted interventions that may lead to greater improvements.

More recently, social cognition has been identified as a unique contributor to poor social outcomes in schizophrenia, including: social behaviour, social problem solving, social skills and community functioning. Social cognition differs to neurocognition in that specific abilities are involved to process information about oneself, others, interactions and social situations.\textsuperscript{15} In fact, social cognition has been shown to account for more variance than both neurocognition and psychopathology in predicting community and social functioning.\textsuperscript{6,16-17} An association between social cognition and social functioning has been reported even in the early course of the illness at the time of the first episode.\textsuperscript{18} Other studies have identified various social cognitive domains, e.g. social perception, social knowledge and Theory of Mind (ToM), as mediators between neurocognition and functional behaviours.\textsuperscript{5,10,19-20}

Psychopathology has also been implicated in having an adverse impact on social functioning in schizophrenia. Negative symptoms are thought to be of particular relevance in predicting real-world functioning; low motivation and apathy are purported to affect one’s ability to effectively engage in social and vocational roles. Indeed, negative symptoms have consistently been shown as significantly associated with poor social and community functioning.\textsuperscript{5,9-11,21} Other types of psychotic symptoms have also been related to poor functioning in schizophrenia, e.g. disorganised\textsuperscript{6,21-22} and positive.\textsuperscript{21,23} Even though the vast majority of these studies have used established
schizophrenia samples, a select few have investigated individuals at an earlier phase of illness e.g. at-risk of psychosis$^{22}$ and initial acute onset.$^{11}$ Thus, it seems psychopathology may play a role in functional capacity at different stages of psychotic illness.

Various studies have compared psychopathology and neurocognitive variables in terms of their relationship with social functioning and outcomes. In general, neurocognition has been identified as having a stronger association with functioning than psychiatric symptoms.$^{2}$ A recent meta-analysis suggested that psychopathology, especially negative symptoms, at least partially mediates the relationship between neurocognition and functional outcome in schizophrenia.$^{12}$ Other studies have shown psychopathology and not neurocognition to be associated with functioning. Guaiana et al.$^{11}$ found negative symptoms to be the sole predictor of social functioning at 9-month and 18-month follow-up in a sample of both early and chronic schizophrenia patients, whereas Zuo et al.$^{21}$ demonstrated strong associations between social functioning and three major symptom domains but not neurocognition. The overall inconsistency in findings has created difficulty in determining whether psychopathology or neurocognitive factors have a more profound effect on functioning.

Moreover, social cognition has not been thoroughly investigated as a predictor of social functioning relative to neurocognitive and psychopathological factors,$^{13}$ which adds further ambiguity as to which variables are most influential. When social cognition, neurocognition and symptoms have been assessed within the same study, social
cognition appears to have the strongest impact on social functioning, although all participant samples consisted of established schizophrenia patients and only one or two social cognitive domains were assessed. It remains unknown as to which factors affect social functioning in the initial stage of psychotic illness.

To the authors’ knowledge, this is the first study to assess the relative impact of social cognition, neurocognition and psychopathology on various domains of functioning in FEP. The specific aim was to determine the best predictor of social functioning in a cross-sectional study design. Based on recent research, it was hypothesised that social cognition would have the greatest impact on social functioning. Neurocognition was also expected to be predictive of functioning, however to a lesser degree. Given that the FEP sample in the current study was obtained from an outpatient clinic and would likely demonstrate milder symptom severity compared to acute or established schizophrenia populations, it was anticipated that psychopathology would have minimal effect on social functioning.

2. Method

2.1 Participants

Forty outpatients with a diagnosed first episode of psychosis were recruited from the Early Psychosis Prevention and Intervention Clinic (EPPIC) at Orygen Youth Health in Melbourne, Australia. All participants were aged 15-25 years and in a stable, non-acute phase at the time of assessment. Participants were part of a larger study (see Thompson et al. for specific sample characteristics) in which social cognition ability was assessed
and compared between three groups: FEP, ultra-high-risk (UHR) of developing a psychotic disorder, and healthy controls.

2.2 Materials

2.2.1 Psychopathology assessment

Psychopathology was assessed using the Positive and Negative Syndrome Scale (PANSS), a 30-item measure that indicates severity of psychotic and other symptoms. Individual symptoms are rated on a scale of 1 (absent) to 7 (severe); the total score was used to indicate overall psychopathology severity. For the purpose of a secondary analysis that was conducted to ascertain whether specific psychotic symptoms were associated with poorer functioning, psychopathology was later divided into three symptom domains (i.e. negative, positive and disorganised). The three symptom clusters were generated based on Liddle’s depiction of the major syndromes in schizophrenia, which have been supported by other factor analytic studies of the PANSS.

2.2.2 Social cognition assessment measures

Various tasks were administered to assess four social cognitive domains: emotion recognition, theory of mind (ToM), emotion perception and social knowledge. In order to standardise scores across the different measures and generate a social cognition variable, all scores were first converted to z-scores then combined to form a composite score.
**Emotion recognition.** Emotion recognition was measured by the Diagnostic Analysis of Nonverbal Accuracy-2 (DANVA-2). Both components (facial recognition and paralanguage) of the adult version of the DANVA-2 were administered on a laptop computer. The number of errors on each component was combined to yield an emotion recognition total error score.

**Theory of mind (ToM).** Two separate tasks were administered to assess ToM: i) the Hinting Task and ii) the Visual Jokes task. The Hinting Task assesses one’s ability to decipher actual intentions behind indirect speech. It contains 10 items, with a possible maximum score out of 20. The Visual Jokes task is non-verbal and requires interpretation of humour in cartoon illustrations. Responses to 20 jokes were given a ‘mentalising’ score between 0 and 3 (maximum score of 60), which reflected the participant’s ToM level. The correlation between the ToM tasks was significant $r = .459, p = .011$. Raw scores on these tasks were transformed into z-scores and combined to produce an overall ToM variable.

**Emotion perception.** The Managing Emotions module of the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT) is the only task in the MATRICS Consensus Cognitive Battery (MCCB), which was administered in its entirety in this study, that assesses “social cognition”. Here and elsewhere, the MSCEIT has been classified more specifically as an emotion/social perception task as it requires processing of social information and an awareness of emotional states in hypothetical real-life scenarios. The MSCEIT was scored using software available from Multi-Health Systems Inc, Toronto, Ontario, Canada, which produces an overall score; higher scores indicate a greater ability to process and manage emotions.
**Social knowledge.** Social knowledge was assessed by the Social Component Sequencing Task – Revised (SCST-R). The task contained 12 common social scenarios, half ‘short’ (six actions) and half ‘long’ (nine actions). Each action was displayed in writing on laminated card and presented in a pre-determined, incorrect order. For each social situation, participants were required to sequence the actions in the correct order. Two of the original scenarios were replaced with more age and culture-appropriate situations to reflect the demographic of the study group. Some actions in the original task were also slightly modified for this reason (the first author can be contacted directly for details). The presentation of short and long scenarios was counterbalanced to control for order effects. Mean juxtaposition scores were determined for each sequence, that is, the number of cards correctly juxtaposed to neighbouring cards divided by the total possible correct juxtapositions. For example, if a nine-item scenario was ordered as C-B-D-E-A-I-F-G-H, three of eight neighbouring pairs were correctly sequenced (D-E, F-G, and G-H), equating to a score of 0.375.

### 2.2.3 Neurocognition assessment measures

Nine neurocognitive tasks were administered as per the MCCB (MATRICS Assessment Inc). The MCCB includes the following neurocognitive tasks: Trail Making Test (TMT) – Trails A, Brief Assessment of Cognition in Schizophrenia – Symbol Coding (BACS-SC), Category Fluency: Animal Naming, Continuous Performance Test – Identical Pairs (CPT-IP), Wechsler Memory Scale-III: Spatial Span (WMS-SS), Letter-Number Span (LNS), Hopkins Verbal Learning Test – Revised (HVLT-R), Brief Visuospatial Memory Test – Revised (BVMTR), and the Neuropsychological Assessment Battery (NAB): Mazes. A description of the tasks can be viewed online at [www.matricsinc.org](http://www.matricsinc.org).
Six neurocognitive domains are measured by the MCCB; speed of processing, working memory, verbal learning, visual learning, reasoning and problem solving, and attention/vigilance. Speed of processing corresponds to performance on the TMT-Trails A, BACS-SC, and Category Fluency tasks. The working memory domain is represented by the LNS and WMS-SS. The verbal learning domain reflected overall performance on three learning trials of the HVLT-R, whereas the visual learning domain corresponded to ability across three learning trials on the BVMT-R. The reasoning and problem solving domain was associated with performance on the NAB Mazes. The attention/vigilance domain corresponded to performance on the CPT-IP. All neurocognition raw scores were initially transformed into z-scores and combined to produce the neurocognition composite score.

2.2.4 Social functioning measures

Global Functioning (GF) scales – Role and Social

The GF Role scale assesses performance in role functioning such as work, school, or household duties. The GF Social scale assesses quantity and quality of peer relationships, age-appropriate intimate relationships, extent of peer conflict, and interaction with family members. Each of the GF scales (published36) are scored between 1 and 10, where 1 reflects extreme dysfunction and 10 indicates superior functioning with no difficulties or impairments.

Multidimensional Adolescent Functioning Scale (MAFS)

The MAFS is a 23-item self-report questionnaire that assesses various aspects of adolescent functioning. The measure was originally developed by mental health
professionals at Orygen Youth Health in Melbourne and has recently been validated\(^{37}\) in a youth population. In addition to a total score that reflects overall functioning, three subscales can be generated; general functioning (MAFS-general), peer-related functioning (MAFS-peer) and family-related functioning (MAFS-family). The MAFS-general subscale assesses a variety of functional domains, including: educational and vocational, physical health status, life satisfaction, and achievements. The MAFS-peer subscale reflects perceived approval and support by peers or friends, whereas the MAFS-family subscale represents perceived closeness to one’s family and parental support. Items are rated from 1 “not at all or rarely” to 4 “always or almost always” applicable. Where an item does not apply, a “not applicable” option (with a score of 0) can be selected. Higher scores represented better functioning. For a detailed description of the scoring procedure, see Wardenaar et al.\(^{37}\)

### 2.3 Procedure

Participants underwent a clinical interview in which demographics, psychopathology, and functioning were assessed. This was followed by completion of all social cognition and neurocognition tasks. The duration of the assessment was approximately 3.5 hours. A few assessments were split over two days due to time constraints. Two researchers completed the assessments; both had training and experience in administering the PANSS and undertook reliability checks throughout the study period, including consensus ratings on training videos.
2.4 Statistical analysis

Relationships between the two GF scales and the MAFS subscales were first analysed using Pearson's correlations in order to ascertain if each scale represented a distinct functional indicator. Linear multiple regression analyses were conducted to determine the significant predictors of functioning in FEP patients. Five indicators of functioning (GF Role, GF Social, MAFS-general, MAFS-peer and MAFS-family) were assessed as dependent variables in separate regression analyses. The social cognition composite score, neurocognition composite score and PANSS total score (which were not significantly correlated with one another) were entered as independent variables in all regression models.

3. Results

3.1 Sample characteristics

The gender demographic of the sample was 65% male (N = 26) and 35% female (N = 14), with 75% of all participants prescribed atypical antipsychotics at the time of assessment. The remaining 25% of participants were not taking any antipsychotic medication. A summary of the demographics is presented in Table 1, including: age, number of years education/formal training, level of role and social functioning, overall psychopathology and symptom-type severity, and performance on all cognitive tasks.
Table 1. Sample characteristics: demographics, psychopathology, functioning and cognition

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>20.52</td>
<td>2.47</td>
<td>16 – 25</td>
</tr>
<tr>
<td>Education (years)</td>
<td>12.53</td>
<td>1.60</td>
<td>10 – 17</td>
</tr>
<tr>
<td><strong>Psychopathology</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PANSS overall score</td>
<td>59.00</td>
<td>14.80</td>
<td>33 – 91</td>
</tr>
<tr>
<td>PANSS Negative (psychomotor retardation)</td>
<td>1.89</td>
<td>0.72</td>
<td>1 – 4.3</td>
</tr>
<tr>
<td>PANSS Positive (reality distortion)</td>
<td>2.11</td>
<td>0.97</td>
<td>1 – 4.4</td>
</tr>
<tr>
<td>PANSS Disorganised</td>
<td>1.86</td>
<td>0.62</td>
<td>1 – 4</td>
</tr>
<tr>
<td><strong>Functioning</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Global Functioning: Role scale</td>
<td>5.65</td>
<td>5.54</td>
<td>1 – 9</td>
</tr>
<tr>
<td>Global Functioning: Social scale</td>
<td>6.70</td>
<td>1.14</td>
<td>4 – 9</td>
</tr>
<tr>
<td>MAFS-general</td>
<td>23.63</td>
<td>7.33</td>
<td>8 – 36</td>
</tr>
<tr>
<td>MAFS-peer</td>
<td>15.43</td>
<td>5.89</td>
<td>0 – 23</td>
</tr>
<tr>
<td>MAFS-family</td>
<td>19.10</td>
<td>5.70</td>
<td>3 – 28</td>
</tr>
<tr>
<td><strong>Cognition</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neurocognition Composite (z-score)</td>
<td>-0.16</td>
<td>0.56</td>
<td>-1.39 – 0.91</td>
</tr>
<tr>
<td>Social cognition Composite (z-score)</td>
<td>-0.11</td>
<td>0.49</td>
<td>-1.72 – 1.07</td>
</tr>
</tbody>
</table>

Abbreviations: PANSS, Positive and negative Syndrome Scale; MAFS, Multidimensional Adolescent Functioning Scale
Pearson’s correlations were computed between five social functioning indices (GF Role, GF Social, MAFS-general, MAFS-peer and MAFS-family). MAFS-family was the only measure that did not correlate significantly with any of the other social functioning indicators. Excluding the MAFS-family subscale, correlations between the other social functioning measures were mostly moderate (between $r = .35, p<.05$ and $r = .626, p<.001$).

3.2 Relative contribution of three factors to functioning

Both of the GF (Role and Social) scales and the three subscales of the MAFS (i.e. MAFS-general, MAFS-peer and MAFS-family) were included as dependent variables in separate linear regression analyses. Three predictors (social cognition, neurocognition and psychopathology) were entered as independent variables to assess their relative contribution to functioning. All regression models were significant, except for that in which the MAFS-family subscale was the dependent variable (see Table 2 for a summary of the results). In this model, none of the three factors were significant predictors. Examining the independent variables in each of the significant models, psychopathology emerged as the only factor highly associated with the functioning indices, accounting for moderate to large amounts of variance. Specifically, higher psychopathology was associated with poorer functioning, despite the average severity of individual PANSS symptoms being mild (mean ≤ 3.15). Overall social cognition and neurocognition did not significantly predict functioning irrespective of the indicator used to assess functioning.
Table 2. Social cognition, neurocognition and psychopathology as predictors of functioning.

<table>
<thead>
<tr>
<th>Functioning measure</th>
<th>Model and Predictors</th>
<th>$R^2$</th>
<th>$F$ value</th>
<th>Beta</th>
<th>$t$ value</th>
<th>$p$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Global Functioning: Role</strong></td>
<td>Model</td>
<td>.313</td>
<td>5.310</td>
<td></td>
<td></td>
<td>.004</td>
</tr>
<tr>
<td>Social cognition</td>
<td></td>
<td>.124</td>
<td>.870</td>
<td>.390</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neurocognition</td>
<td></td>
<td>.187</td>
<td>1.306</td>
<td>.200</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychopathology</td>
<td></td>
<td>-.483</td>
<td>-3.427</td>
<td>.002</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Global Functioning: Social</strong></td>
<td>Model</td>
<td>.556</td>
<td>14.611</td>
<td></td>
<td></td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Social cognition</td>
<td></td>
<td>-.004</td>
<td>-.031</td>
<td>.976</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neurocognition</td>
<td></td>
<td>.141</td>
<td>1.228</td>
<td>.227</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychopathology</td>
<td></td>
<td>-.717</td>
<td>-6.331</td>
<td>&lt;.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>MAFS-general</strong></td>
<td>Model</td>
<td>.450</td>
<td>9.543</td>
<td></td>
<td></td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Social cognition</td>
<td></td>
<td>-.024</td>
<td>-.193</td>
<td>.848</td>
<td></td>
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</tr>
<tr>
<td>Neurocognition</td>
<td></td>
<td>-.011</td>
<td>-.088</td>
<td>.931</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychopathology</td>
<td></td>
<td>-.672</td>
<td>-5.328</td>
<td>&lt;.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>MAFS-peer</strong></td>
<td>Model</td>
<td>0.203</td>
<td>2.968</td>
<td></td>
<td></td>
<td>.045</td>
</tr>
<tr>
<td>Social cognition</td>
<td></td>
<td>-.003</td>
<td>-.020</td>
<td>.984</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neurocognition</td>
<td></td>
<td>-.185</td>
<td>-1.201</td>
<td>.238</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychopathology</td>
<td></td>
<td>-.431</td>
<td>-2.839</td>
<td>.007</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>MAFS-family</strong></td>
<td>Model</td>
<td>.079</td>
<td>1.001</td>
<td></td>
<td></td>
<td>.404</td>
</tr>
<tr>
<td>Social cognition</td>
<td></td>
<td>-.155</td>
<td>-.945</td>
<td>.351</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neurocognition</td>
<td></td>
<td>.246</td>
<td>1.490</td>
<td>.145</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychopathology</td>
<td></td>
<td>-.061</td>
<td>-3.376</td>
<td>.709</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: MAFS, Multidimensional Adolescent Functioning Scale
3.3 Secondary analysis: Investigation of the relationship between symptom domains and functioning

As overall psychopathology was the only factor associated with functioning in the primary regression models, a secondary analysis was conducted to investigate whether this finding was specific to negative, positive and/or disorganised symptomatology. The three symptom clusters were analysed as predictors in four separate linear multiple regressions; the functional indicators associated with significant models in the primary regression analyses (i.e. GF Role, GF Social, MAFS-general and MAFS-peer) were each analysed as dependent variables. Psychomotor poverty (i.e. negative symptomatology) and reality distortion (i.e. positive symptomatology) domain scores were significantly associated with various functional indices, however disorganised symptoms were not at all related to functioning. Positive and negative symptomatology each contributed the same amount of variance (40%) to social functioning as measured by the GF Social scale. A similar level of variance was accounted for by positive (42%) and negative (38%) symptoms in general functioning (MAFS-general subscale). Only negative symptoms were significantly associated with the MAFS-peer subscale, accounting for nearly 50% variance. The GF Role scale was not predicted by any of the three symptom clusters, although negative symptoms displayed trend-level significance. A summary of the analyses is provided in Table 3.
Table 3. Secondary analysis: Impact of symptom-type on functioning

<table>
<thead>
<tr>
<th>Functioning measure</th>
<th>Predictors</th>
<th>Beta</th>
<th>t value</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Global Functioning: Role</strong>*</td>
<td>Negative</td>
<td>-.263</td>
<td>-1.768</td>
<td>.086</td>
</tr>
<tr>
<td></td>
<td>Positive</td>
<td>-.167</td>
<td>-1.053</td>
<td>.299</td>
</tr>
<tr>
<td></td>
<td>Disorganised</td>
<td>-.271</td>
<td>-1.684</td>
<td>.101</td>
</tr>
<tr>
<td><strong>Global Functioning: Social</strong></td>
<td>Negative</td>
<td>-.407</td>
<td>-3.131</td>
<td>.003</td>
</tr>
<tr>
<td></td>
<td>Positive</td>
<td>-.400</td>
<td>-2.893</td>
<td>.006</td>
</tr>
<tr>
<td></td>
<td>Disorganised</td>
<td>-.086</td>
<td>- .611</td>
<td>.545</td>
</tr>
<tr>
<td><strong>MAFS-general</strong></td>
<td>Negative</td>
<td>-.375</td>
<td>-2.753</td>
<td>.009</td>
</tr>
<tr>
<td></td>
<td>Positive</td>
<td>-.422</td>
<td>-2.909</td>
<td>.006</td>
</tr>
<tr>
<td></td>
<td>Disorganised</td>
<td>-.006</td>
<td>- .038</td>
<td>.970</td>
</tr>
<tr>
<td><strong>MAFS-peer</strong>*</td>
<td>Negative</td>
<td>-.498</td>
<td>-3.388</td>
<td>.002</td>
</tr>
<tr>
<td></td>
<td>Positive</td>
<td>-.188</td>
<td>-1.204</td>
<td>.236</td>
</tr>
<tr>
<td></td>
<td>Disorganised</td>
<td>.185</td>
<td>1.163</td>
<td>.252</td>
</tr>
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Abbreviations: MAFS, Multidimensional Adolescent Functioning Scale

* regression model significant at $p \leq .01$  
** regression model significant at $p \leq .001$

4. Discussion

The aim of the study was to investigate the relative contribution of neurocognition, social cognition and psychopathology to multiple dimensions of functioning in FEP. The hypothesis that social cognition would be the strongest predictor of functioning was not supported; in fact, social cognition failed to account for any significant amount of variance in the functional domains assessed. Similarly, neurocognition was anticipated to have an impact on functioning however a relationship was not found. Psychopathology was not expected to have a great effect on functioning, yet it was the only factor predictive of a variety of functional indices. Given this unexpected finding, a
secondary analysis was undertaken to determine whether particular symptom domains were associated with functioning. Negative and positive symptoms were found to contribute to various dimensions of functioning, with disorganised symptoms having no impact.

It is unclear as to why social cognition and neurocognition did not contribute to concurrent functioning in the current sample. This finding is inconsistent with the majority of previous research; associations between social cognitive deficits and poor social functioning have been commonly reported,\(^7\) whilst neurocognition has been shown to account for up to 60% of variance in functional outcomes.\(^2\) However, in line with the current results, there have been a few studies that have not demonstrated strong relationships between neurocognition and functioning.\(^{21,38}\) It is important to note that these studies,\(^{21,38}\) as well as the current study, share the commonality of a modest sample size (N≤40) thus failure to detect a significant association may have been due to insufficient power. An alternative hypothesis is that non-cognitive variables may have a greater impact on functioning in the early course of illness, which is supported by the current findings in that psychopathology was the only significant predictor of functioning across multiple functional indices. Moreover, longitudinal studies have shown that neurocognition\(^{39}\) and social cognition\(^{18}\) have weak relationships with functioning at baseline, yet become stronger over time.

The secondary analysis suggests negative and positive (but not disorganised) symptoms contribute to social functioning in FEP. This finding was similar to that of another recent
study in which highly significant correlations between both negative and positive symptom domains and overall functioning were reported. However, unlike the current study, Zuo and colleagues also found that disorganised symptoms were associated with functioning. The reason for this disparity may be due to differences in sample characteristics; the stable outpatients in the current study displayed a milder level of disorganisation compared to the acute inpatients in the other study. Even though multiple symptom domains have been shown to affect functioning, the majority of research to date suggests negative symptoms are more highly linked to poorer social functioning. The secondary analysis in the current study should be treated as exploratory due to the modest number of participants and multiple testing issues, however future studies should aim to replicate the findings in a larger early psychosis sample.

The main strength of the study was that multiple domains of functioning were assessed and that the measures used (i.e. MAFS and GF scales) were age-appropriate for the sample. Even though these tools were developed fairly recently and have not been extensively used to date, they contain items of relevance to younger populations when compared to the other widely used measures of social functioning in schizophrenia research (e.g., Social Functioning Scale). The GF scales are essentially based on the well-known Social and Occupational Functioning Assessment Scale (SOFAS) and represent two advantages; i) they are more specific indicators of social and role functioning, as these constructs are assessed separately in the ratings, and ii) they were designed for use in adolescent and young adult at-risk populations and are thus more suited to FEP participants. Indeed, the GF scales have previously been used to assess
social and role functioning in FEP, with the authors having concluded that the measures were valid and useful indicators of functioning in this population. The MAFS was also designed for use in at-risk individuals, and although it has not been used in other FEP studies to date, it was recently validated in a general population sample. Interestingly, the MAFS-family subscale was the only functional indicator in this study not associated with symptoms, which suggests family-related functioning may be associated with factors independent of psychosis.

A methodological limitation of the study was the assessment of both neurocognition and social cognition as entire constructs, rather than by domain-type. Composite scores were computed to limit the number of predictors in the regression analyses, in order to minimise multiple testing issues and maximise statistical power. The decision to use composite scores was further justified as another study using the same sample reported social cognitive impairments across a variety of social cognitive domains. However, significant associations between specific neurocognitive and/or social cognitive domains and functioning may have still been masked, as past research has suggested certain social cognitive domains, i.e. ToM, have a stronger effect on functioning than other areas of social cognition. Similarly, particular neurocognitive processes (e.g. verbal memory and attention) have been shown as more predictive of functioning than other domains.

In summary, the findings indicate that overall psychopathology (especially negative and positive symptoms) is closely associated to concurrent social functioning in FEP.
Traditionally, the focus of treatment in FEP has been symptom management above and beyond other features of the disorder, however this study highlights the importance of addressing both psychopathology and social functioning as early as possible in the course of the illness. Neurocognition and social cognition as overall broad constructs were not concurrently related to functioning in this study. Despite this, past research has demonstrated associations between certain cognitive domains and not others, thus a subset of key neurocognitive and social cognitive processes may be significantly impacting on functioning in FEP. Rather than investigating neurocognition and social cognition as entire constructs, future research should be focused on assessing the cognitive domains previously associated with social outcomes in the literature. Ensuring a sufficient sample size to allow for statistical testing of multiple domains is also necessary. Furthermore, longitudinal studies will not only inform when to best treat neurocognitive and social cognitive deficits, but would also clarify whether specific symptom domains are predictive of functional outcome beyond the early stage of illness.
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CHAPTER 10: General Discussion

10.1 Chapter Overview

This chapter begins with an overview of the aims and objectives of this thesis, which related to the broad topic of social cognition in first episode psychosis (FEP). Particular research goals were investigated in four studies, each with the following respective aims: 1) to assess the social cognitive ability of individuals with FEP, as to ascertain whether deficits exist in this clinical population and if they are widespread, 2) to examine the relationships between different domains of social cognition and neurocognition, in order to determine if the two constructs are distinct from one another, 3) to assess whether particular psychiatric symptom subtypes are associated with social cognition, and 4) to identify which key factors (social cognition, neurocognition, or symptoms) best predict social functioning in FEP. The findings of each study are summarised and discussed, and associated methodological considerations are raised. Furthermore, the limitations of the overall research are highlighted, with suggestions for future research in this field. This chapter concludes with a discussion of the clinical implications of the research.

10.2 Review of the research aims

10.2.1 Primary research aim

Given the growing evidence of social cognition deficits across schizophrenia-spectrum disorders in the literature, the main aim of the research was to determine whether social cognitive impairment is present in FEP, a distinctive clinical group in the early phase of psychotic illness who are not easily sourced from community-based mental
health services or hospital settings. This research aim was investigated by comparing performance across four social cognitive domains between a sample of FEP outpatients recruited from a specialised community mental health service (Orygen Youth Health) that services the northwestern metropolitan region of Melbourne, and a group of healthy controls residing in the same geographical catchment area. The results of the study were reported in an original article entitled "Social cognition in clinical ‘at risk’ for psychosis and first episode psychosis populations", published by the journal *Schizophrenia Research*, and presented in Chapter 6 of this thesis.

As mentioned earlier in Chapter 5, it is brought to the reader’s attention again here that Article One (see Chapter 6) also includes data from an additional clinical group, specifically one comprising individuals classified as being at ultra-high-risk (UHR) of psychosis. This data does not correspond to the aims and objectives of the thesis itself; the scope of the thesis is limited to social cognition in FEP, with the inclusion of a healthy control sample used as a comparison group. The UHR sample completed the same battery of social cognition tasks as the FEP and control groups in a concurrent study also conducted at Orygen Youth Health Research Centre that aimed to investigate social cognition in UHR individuals, thus it was only logical to analyse the data from all three groups simultaneously and report the overall findings in one original research paper. Importantly, the author of this thesis was also involved in the UHR study on multiple levels beyond general data collection; from study conceptualisation and task selection to interpretation of results and manuscript preparation. It was therefore justifiable to include the original paper published in *Schizophrenia Research* in this
thesis, as a means of reporting the findings associated with the main aim of the current research, however results pertaining to the UHR sample are not further discussed.

10.2.2 Secondary research aims

A number of secondary aims were investigated in additional independent studies. As the empirical literature has provided evidence that social cognition and neurocognition are generally distinct entities with generally a moderate degree of overlap in schizophrenia (Fanning et al., 2012; Sergi et al., 2007; Ventura et al., 2011), the general aim of Study Two was to investigate whether the constructs are in fact separate in FEP by examining the relationships between a variety of domains. Furthermore, the actual amount of shared variance between specific social cognitive and neurocognitive domains remains debatable, especially in FEP. Thus, a specific objective of Study Two was to determine the unique contribution of six specific neurocognitive domains to four social cognitive domains. Another objective was to examine whether the relationships between social cognition and neurocognition are comparable between FEP patients and healthy controls, as most research has only investigated schizophrenia samples in isolation.

The purpose of Study Three was to assess whether particular psychiatric symptoms are more closely related to social cognition in FEP, in order to ascertain whether individuals experiencing certain symptomatology display poorer social cognitive functioning. Finally, various factors are thought to affect social functioning and functional outcomes in psychosis and schizophrenia. Thus, Study Four aimed to determine which of three
factors that have previously been linked to social dysfunction (i.e. social cognition, neurocognition and symptomatology) is the best predictor of social functioning in FEP.

10.3 Review of the Studies and Methodological Considerations

10.3.1 Chapter 6: Various domains of social cognition are impaired in FEP

It is widely accepted that social cognitive deficits are a feature of schizophrenia, and even FEP, however the specific nature and degree of impairment is less clear in the early phase of illness as most research to date has been conducted using chronic or established schizophrenia samples. Furthermore, many studies have only assessed performance on one or two social cognitive domains and have subsequently drawn conclusions about “social cognition” in general, when in fact the construct is multidimensional (Harvey & Penn, 2010) and degree of deficit may be variable according to domain-type. Therefore, based on a National Institute of Mental Health (NIMH) workshop that included definition of the terms used to describe social cognition in the literature and recognition of key domains (Green et al., 2008), the current research aimed to examine social cognitive ability in FEP thoroughly by assessing the specific areas that had been identified at that meeting (i.e. emotion recognition, social/emotion perception, theory of mind (ToM), and social knowledge). This aimed to overcome one of the major methodological limitations in the literature, that is, lack of comprehensive assessment of social cognition within studies.

In Study One of this thesis (see Chapter 6), the four social cognitive domains mentioned above were assessed in a sample of FEP patients aged between 15 and 25 who were
attending a specialised public mental health facility servicing the Western metropolitan region of Melbourne. Healthy control participants who were matched in age range and sourced from the same catchment area as those attending the mental health service also completed the social cognition assessment for comparative purposes. A fifth domain of social cognition was identified at the NIMH workshop (Green et al, 2008), namely attributional style, however it was not assessed in this thesis for a number of reasons. Firstly, the measure considered for the assessment of attributional style was the IPSAQ (Kinderman & Bentall, 1996), based on the fact that it is a highly reliable and valid measure of the construct (Martin & Penn, 2002). However, it would have taken approximately 30 minutes to administer, which was deemed too lengthy for just one assessment task given the extensive number of measures in the research protocol.

Secondly, previous research in this area has consistently shown that individuals with schizophrenia who experience paranoia and persecutory delusions demonstrate an attributional bias, that is, attribute negative outcomes to other people and implicate themselves in positive outcomes (Candido & Romney, 1990; Kaney & Bentall, 1989; Kinderman et al., 1992; Lyon et al., 1994; Martin & Penn, 2002). As such, it was considered that adding the IPSAQ to the assessment procedure would not only cause an excessive time burden on participants, but that replication of a personalising bias would add little value to the research. Furthermore, it was anticipated that a relatively modest number of FEP patients (N = 40) in the study might not have been large enough to yield a sufficient sub-sample of individuals with paranoia at the persecutory delusion level, especially as FEP represents a heterogeneous clinical group. Based on all of these
concerns, it was concluded that assessment of attributional style was not warranted within the constraints of this thesis.

The overall results depicted in Article One revealed that FEP patients performed significantly worse than control participants in various areas of social cognition, specifically: emotion recognition, ToM, and social/emotion perception. In terms of the magnitude of the difference in test scores between FEP and control groups, effect sizes (cohen’s $d$) were generally medium to large (in the range of 0.62 to 0.93). Social cognitive deficits were most pronounced in the domains of ToM and emotion recognition, which suggests degree of social cognitive impairment in FEP is somewhat domain-dependent.

It is noted that the social cognitive domain of social knowledge, which was assessed in both participant groups using the Schema Component Sequencing Task – Revised (SCST-R; Corrigan & Addis, 1995), was not referred to in Article One. As the aim of the study was to assess four domains of social cognition, including social knowledge, the reason for excluding the associated results warrants explanation. The data analyses revealed that social knowledge was the only domain not associated with a significant group difference, which was a negative finding, as it did not support the hypothesis that all social cognitive domains would be impaired in the FEP sample. The SCST-R results were initially included in an earlier version of the article, however they were subsequently omitted following the peer-review process based on reviewer recommendations. Despite the rigorous guidelines of most journals, it is still important
to communicate all findings from research activities where possible and to highlight potential reasons for negative findings. This also provides fellow researchers with useful information that can serve to inform future research approaches. Thus, the SCST-R results reflecting social knowledge are presented here, as calculated in the original data analyses.

Scoring of the SCST-R involved determining juxtaposition scores for each of the 12 specified situations. The juxtaposition score reflected the number of cards correctly aligned to neighbouring cards divided by the total possible correct juxtapositions. For example, if a nine-item scenario was ordered as C-B-D-E-A-I-F-G-H, three of eight neighbouring pairs were correctly sequenced (D-E, F-G, and G-H), equating to a score of 0.375 (Corrigan & Addis, 1995). Thus, for each item, the total possible score was one. In terms of the FEP group, the mean juxtaposition score across the 12 scenarios was 10.0 (standard deviation = 1.15). The control group performed slightly better overall with a mean score of 10.5 (standard deviation = 0.78), but the difference between the groups was not significant ($p = 0.12$). When age, gender and IQ were controlled for, and the analysis was adjusted for multiple comparisons by applying the False Discovery Rate (FDR) correction, the significance level changed to $p = 0.27$. Although the group difference failed to reach statistical significance, the effect size was still fairly moderate ($d = 0.50$).
10.3.2 Chapter 7: Social cognition and neurocognition represent different constructs

Even though social cognition is still a relatively new field of research, it is considered a multidimensional construct that represents several specific domains (e.g. emotion recognition, social perception, ToM, social knowledge). However, to date, little is known about the interrelationships between these various domains. This raises questions as to whether the identified domains in the literature are actually distinct from one another. Neurocognition, which is considered a separate construct from social cognition, has been extensively researched and the various components (e.g. visual processing speed, memory functions, reasoning and problem solving, attention) are widely accepted as independent from each other. In terms of comparisons between social cognition and neurocognition, a moderate degree of overlap has been documented between the two constructs in schizophrenia research (Ventura et al., 2011). It is unclear, however, whether this overlap is only applicable to established schizophrenia samples or whether this relationship is seen in early illness and non-clinical groups. Furthermore, the amount of variance accounted for by specific neurocognitive variables in the various areas of social cognition is unknown.

Study Two was a thorough investigation of: i) the interrelationships between four defined social cognitive domains, and ii) the relationship between each of these four areas of social cognition and six neurocognitive domains commonly impaired in schizophrenia. Regarding the construct of social cognition, there were no significant associations between the four domains in the control group. However, ToM was moderately associated with all other social cognitive domains in the FEP group. This
finding may suggest that individuals in the early phase of psychotic illness may find inferring the mental states of others a highly complex skill, and that other more basic social cognitive processes are essentially relied upon in order to complete ToM tasks. In line with this speculation, facial recognition has previously been referred to as a lower-level subprocess of social cognition (Brothers, 1990), which implies that emotion recognition and perhaps some of the other more basic social cognitive processes are essentially building blocks for the higher-order abilities, such as, ToM (Pinkham et al., 2003). The fact that ToM was essentially unrelated to the other social cognitive domains in the control group, however, may indicate that inferring the mental states of others is normally not so cognitively demanding, and perhaps nonclinical groups use different strategies to complete high-order social cognitive tasks.

The pattern of relationships between social cognitive and neurocognitive domains was also found to be different between the FEP and control groups. For the control group, the only social cognitive domain that correlated significantly with several of the neurocognitive domains was ToM. Conversely, emotion recognition and social knowledge were associated with various neurocognitive functions in the FEP group. Linear multiple regression analyses revealed that overall neurocognition accounted for 41% of variance in ToM in the control group, and 37-38% of variance in each of emotion recognition and social knowledge in the FEP group. Results were only statistically significant for the FEP group at a more conservative α level of .01, likely due to a higher number of individuals in that sample compared to controls.
In terms of specific amount of overlap between the individual neurocognitive and social cognitive domains, shared variance was negligible to very small (up to 11% shared variance) regardless of the nature of the participant group. The only exception to this general finding involved the domain of social knowledge in the control group; each of the neurocognitive domains contributed 25-30% of the variance in social knowledge. Although this was a greater amount of variance compared to the other social cognitive domains, it was still only small to moderate. The reason for the neurocognitive variables contributing a higher amount of variance to social knowledge than the other areas of social cognition may have been due to the nature of the assessment task, that is, the SCST-R (essentially a card-sorting task) likely required a higher amount of perceptual reasoning skills than the other social cognition tasks used in the research. If social knowledge is to be assessed in future studies, alternative tasks should be sourced or developed that do not necessitate various neurocognitive abilities such as sequencing of information.

The overall results of this study suggested that the individual domains assessed were independent of one another, indicated by only a relatively small degree of overlap, thus providing evidence that social cognition and neurocognition are distinct, multidimensional constructs in FEP and non-clinical populations. Neurobiological perspectives offer support for this distinction; brain imaging studies have generally demonstrated that certain neural activity is more specific to particular social cognitive processes (e.g. ToM), whereas the same brain regions are not as prominently linked to non-social cognitive functions (e.g. reasoning and problem solving ability) (Pinkham et al., 2003). Specifically, the medial prefrontal cortex (mPFC) has been consistently shown
as a key region for social cognitive processes, especially ToM (Abdi & Sharma, 2004; Blakemore, 2008; Brunet-Gouet & Decety, 2006) and also emotion processing (Brunet-Gouet & Decety, 2006). Regarding identification of basic emotions such as fear and disgust, imaging studies have also implicated multiple other brain areas including the amygdala, anterior insula, and posterior superior temporal sulcus (pSTS) (Dolan, 2002). Other areas involved in social information processing include the right parietal cortex and basal ganglia (Adolphs, 2001), as well as the temporal-parietal junction and the temporal poles (Frith, 2001). Even though some of these neural structures also play a role in neurocognition, they tend to be most consistently activated in response to social stimuli (Pinkham et al., 2003).

Conversely, specific neurocognitive functions are commonly linked to other areas of the brain. For example, ventricular size abnormalities in schizophrenia are associated with deficits in abstraction/flexibility, language, and attention/concentration (Antonova, 2004). The role of the hippocampus and amygdala have also been highlighted with overall learning and memory ability (Benes, 2009), whereas working memory deficits in schizophrenia have been specifically linked to dysfunction of the dorsolateral prefrontal cortex (DLPFC) (Goldman-Rakic, 1999). Deficits in the DLPFC have also been identified in schizophrenia patients performing executive functioning tasks, with additional dysfunction in the anterior cingulate cortex (ACC) and mediodorsal nucleus of the thalamus (Miznenberg, Laird, Thelen, Carter, & Glahn, 2009). Furthermore, the temporal lobe has been correlated with speed of processing (Antonova, 2004). All in all, the differences in the neural substrates that subserve particular social cognitive and
neurocognitive processes may explain why relationships between domains were relatively small in the current research.

10.3.3 Chapter 8: The relationship between symptomatology and social cognition in FEP

The literature regarding the relationship between psychotic symptoms and social cognition in early psychosis has been somewhat ambiguous. The majority of research investigating this relationship has been conducted using established schizophrenia samples and has generally found an association between deficits in social cognition and negative symptoms (Bora et al., 2009; Sergi et al., 2007; Shean & Meyer, 2009; Stratta et al., 2011). Some studies, however, have reported no association between negative symptoms and social cognition (Bertrand et al., 2007; Mancuso et al., 2011). Findings relating to positive symptoms have also been inconsistent, however an externalising attributional bias has been more or less unequivocally associated with paranoia and persecutory delusions (e.g. Fornells-Ambrojo & Garety, 2009). A third psychotic symptom domain (i.e. disorganisation) has also been investigated, however not on as large a scale as negative and positive symptoms. Again, a clear picture is not yet apparent, although the existing evidence suggests an association between disorganised symptoms and social cognitive deficits (Frith & Corcoran, 1996; Subotnik et al., 2006; Toomey et al., 2002). It is important to examine the relationship between psychopathology and social cognition, especially in the early phase of illness, as social cognitive deficits could play a role in the development of psychotic symptoms.
Study Three sought to clarify the nature of the relationship between three main psychosis-related symptom clusters and various social cognitive domains in FEP. The fact that a number of social cognitive domains were investigated was a particular strength of the study, as most other studies have only assessed one or two areas of social cognition. Liddle’s (1987) three-factor symptom model was used to categorise psychopathology, as it depicts the major symptom clusters in schizophrenia-spectrum disorders i.e. negative, positive and disorganised. Furthermore, this symptom paradigm has been used in other research (e.g. Cameron et al., 2002; Smith et al., 2002; Zuo et al., 2012). Based on the social cognitive domains assessed in the study and the overall findings in the literature, it was hypothesised that negative and disorganised symptoms would be associated with social cognition however positive symptoms would show no significant relationship. This hypothesis was partially supported; disorganisation was significantly related to social cognition, however the positive and negative symptom clusters were unrelated. When symptoms were assessed on an individual level, various disorganised and negative symptoms correlated with poorer social cognition. Only two particular symptoms contributed a significant amount of variance to social cognition; abstract thinking accounted for a significant amount of variance in ToM and emotion recognition, whereas stereotyped thinking predicted poorer social perception. Both of these symptoms are cognitive-based, which suggests symptoms reflecting clear cognitive processes (regardless of whether they are more broadly classified as negative or disorganised phenomena) are associated with social cognition.

Given the cross-sectional nature of the study, conclusions could not be drawn as to whether social cognitive deficits are actually risk factors for certain psychopathology, in
particular disorganised and/or negative symptoms with an underlying cognitive component. Alternatively, symptoms of this type may be direct expressions of social cognition problems. Longitudinal studies would help to clarify the nature of the associations and better inform treatment interventions.

10.3.4 Chapter 9: Contributions of neurocognition, social cognition and psychopathology to social functioning in FEP.

It is well known that social and role functioning are affected in individuals with schizophrenia and psychosis, however it remains uncertain as to which syndromes of the disorder have the greatest impact. Research has shown that both social and non-social cognitive variables are significantly associated with social functioning. In fact, neurocognition has previously been shown to account for 40-60% of variance in functional outcomes (Green et al., 2000) and it has been suggested that social cognition may have a greater effect than neurocognition (Brüne et al., 2007; Lysaker et al., 2005; Pinkham & Penn, 2006). Psychopathology, in particular negative symptoms, has also been linked to poor functioning (Ventura et al., 2009). As most research in this field has been conducted in established and chronic schizophrenia samples to date, the impact of these factors on functioning in the early phase of illness is less clear.

The focus of Study Four was to examine the relative contribution of neurocognition, social cognition and psychopathology to various areas of functioning in the FEP sample. It was found that psychopathology was the only factor predictive of functioning, with a secondary analysis conducted to determine whether particular symptom domains
(negative, positive, or disorganised) were associated with poorer functioning. Results of this secondary investigation revealed that both negative and positive symptoms were associated with poor functioning, whereas disorganised symptomatology was not.

As the overall literature has indicated the importance of neurocognition and social cognition on functional capacity in schizophrenia and psychosis, it was surprising to find that these factors did not account for any significant amount of variance in social and role functioning in FEP. The findings of Study Four suggest cognitive variables are not as influential on functioning in the early phase of psychotic illness as negative and positive symptoms, however the lack of association between cognition and functioning may have been due to the way the data was analysed. In particular, the use of a neurocognition composite score may have masked significant contributions of certain neurocognitive domains. The neurocognition composite reflected performance on six domains known to be impaired in schizophrenia, however past research has demonstrated strong associations between some of these areas (e.g., verbal memory, attention, and executive functions) but not others (Green et al., 2000; Green et al., 2004; Milev et al., 2005). Similarly, the social cognition composite score may have also overshadowed particular domains having a more significant effect on social functioning than others. For example, ToM has previously been shown to have a greater impact on social functioning than other social cognitive domains (Brüne et al., 2007).

Nonetheless, the use of cognitive composite scores in Study Four was justified from a statistical standpoint, that is, to limit the number of independent variables and minimise
multiple comparison issues in the regression analyses. This statistically based decision was further supported by the results of Study One in that all three of the social cognitive domains reported (that is, emotion recognition, ToM, social/emotion perception) were significantly impaired in the FEP group compared to the control sample. Hence, combining the scores on the social cognition tasks seemed empirically valid. It is pointed out that the social cognitive domain of social knowledge, which was assessed but not found to be different between FEP patients and controls, was included in the social cognition composite. Thus, a possible hypothesis as to why overall social cognition did not significantly impact on functioning is that the inclusion of social knowledge in the composite perhaps masked any effects of the other social cognitive domains shown to be impaired in the clinical sample. This hypothesis was tested by removing social knowledge from the composite and re-running the regression analyses; this did not affect the results reported in Study Four in any way, thus social knowledge was kept in the social cognition composite.

10.4 Further Limitations, Critique, and Future Research Directions

In addition to the study-specific methodological considerations already raised in this chapter, there were a few generic limitations of the current research in terms of overall study design and statistical analyses. The main limitation related to sample size. Each participant group contained a modest number of individuals; 40 in the patient sample and 30 in the control sample. This may have limited statistical power in each respective study, which perhaps affected the results. Had the sample size been larger, the results may have emerged differently and depicted a more accurate picture in terms of: i) the magnitude of difference in social cognition ability between patients and controls, ii)
degree of relatedness between the various social cognitive and neurocognitive domains investigated in this research, iii) the nature of the relationship between symptom clusters and social cognitive domains, and iv) the actual significance of the various factors impacting on various areas of functioning.

The current study was still able to demonstrate that FEP patients performed significantly worse than controls in several social cognitive domains, which provided evidence that social cognition is impaired in the early course of psychotic illness. The modest FEP sample size also led to findings that support previous studies having shown a relationship between negative/disorganised symptoms and social cognition (Corrigan et al., 1994; Frith & Corcoran, 1996; Piskulic & Addington, 2011; Subotnik et al., 2006). Similar to findings reported by Piskulic and Addington (2011), individual negative and disorganised symptoms of a cognitive nature (i.e. lack of abstract thinking and stereotyped thinking) were found to have a more profound effect on social cognition.

The relatively small number of participants within each of the samples limited the way in which the data could be analysed. For example, regarding Study Four, the data was examined using linear regression analyses to ascertain the specific contribution of various key factors of psychosis (i.e. neurocognition, social cognition and psychopathology) on social functioning in participants with FEP. The study showed that only psychopathology significantly contributed to poor functioning in FEP, however had the FEP sample been larger, the neurocognition and social cognition data could have been analysed in a more comprehensive way, for example, by examining the contribution of the ten individual cognitive domains to functioning rather than as two overall constructs. A higher number of independent variables would have magnified
multiple testing issues and further affected statistical power with the size of the current sample, however if individual neurocognitive and social cognitive domains were examined independently in regression analyses in terms of their effect on various functional indices, certain domains may have been found to account for a significant amount of variance on functioning. As other studies have suggested, impairment in particular cognitive domains (e.g. verbal memory, attention, ToM, emotion recognition) may have a greater effect on social functioning than others.

In general, a larger FEP sample would have allowed for more flexibility in data analysis methods where statistical issues would be minimal or absent. Regarding the study depicted in Article Four, an ideal way to analyse the data would have been through the use of a path model such as structural equation modelling (SEM). This would have allowed both direct contributions and mediator effects of the various neurocognitive, social cognitive and symptom variables on functioning to be examined. Some studies have used path analysis or SEM and found that social cognition acts as a mediator between neurocognition and functioning (e.g. Sergi et al., 2006), whereas a recent review showed negative symptoms play a mediatory role between neurocognition and functioning (Ventura et al., 2009). However unlike Study Four in the current thesis, none of these studies investigated the effect of all three factors (i.e. neurocognition, social cognition and psychopathology) on social functioning. Thus, future studies should aim to replicate the aim of Study Four by examining the impact of multiple factors on social functioning in a comprehensive manner, i.e. examining several domains of cognition (neurocognitive and social cognitive) and different symptoms clusters, and to
ensure sample sizes are large enough to conduct SEM-type analyses to assess relationships.

The cross-sectional nature of the research was a limitation in terms of preventing cause-and-effect conclusions. Article Three provides useful information in that cognitive-based negative or disorganised symptoms (specifically, lack of abstract thinking and stereotyped thinking) predict poorer social cognition in FEP, however it is also possible that social cognition deficits contribute to the development of these symptoms. Longitudinal research is needed to provide insight into whether early social cognitive deficits are perhaps risk factors for specific symptoms. Similarly, the results in Article Four raise questions as to whether: i) specific symptom domains are predictive of functional outcome beyond the early stage of illness, and ii) social cognition and/or neurocognition are associated with poorer social functioning with illness progression, rather than at the first episode. Follow-up studies are required to clarify these questions, however in line with the current research, some longitudinal studies have already provided evidence that social outcomes are more strongly affected by cognitive deficits at follow-up rather than at baseline (e.g. Carlsson, Nyman, Ganse, & Cullberg, 2006; Horan et al., 2012).

Finally, the task chosen to assess social knowledge (i.e. the SCST-R; Corrigan & Addis, 1995) is raised as a methodological concern that may have impacted various aspects of the current research. As aforementioned in this Chapter, there was no significant difference between FEP patients and controls in terms of social knowledge. As this was
the only social cognitive domain found unimpaired in FEP compared to controls, the
task itself may have influenced this result. Even though the SCST-R is a measure of
social knowledge in terms of its content, it is essentially a card-sorting task and various
neurocognitive abilities such as visual processing and executive functioning (i.e.
ordering and sequencing information) are also simultaneously required to complete the
task. The results in Article Two support this in that social knowledge was notably more
related to various neurocognitive domains compared to the other social cognitive
domains in the control group. Thus, the SCST-R was perhaps a relatively poor absolute
measure of social knowledge, thus future studies should consider selecting or
developing a task that measures the construct without being highly influenced by other
cognitive abilities.

It is further noted that the researcher modified the original version of the SCST-R
developed by Corrigan and Addis (1995) due to the fact that the original task contained
scenarios and actions that were either culturally and/or age inappropriate for the
samples tested in the current thesis. For a list of the original and updated items, see
Appendix I. The main changes pertained to two of the long conditions, which were
replaced with entirely different scenarios as follows: ‘getting a raise’ was changed to
‘assignment extension’, and ‘going to church’ was superseded with ‘washing clothes’.
Given the age range of the participants in the current research, that is, 15-25 years, the
situation of getting a raise was not considered age-appropriate and therefore changed
to a situation that was comparable in terms of context and more suited to the sample
demographic. Similarly, as the sample was Australian and given cultural differences and
attitudes in this country, it was assumed that some of the participants in the current
research would not routinely go to church, if at all. Thus, the scenario was changed to a one that reflected social functioning in terms of self-care and routine daily living activities; in this regard, it was similar to the scenario of ‘taking a shower’, which was another item in the task.

10.5 Clinical implications of the research

The current research demonstrated that various social cognitive domains are impaired in FEP, in particular, emotion recognition, ToM, and social/emotion perception. Furthermore, evidence was provided that different neurocognitive and social cognitive domains are relatively independent of one another, thus interventions targeting multiple neurocognitive and social cognitive domains in remediation efforts is implicated. The findings also suggested that mentalising ability is particularly difficult for individuals with FEP, given that: i) ToM was the domain most impaired in FEP compared to controls as measured by the Hinting Task, and ii) ToM overlapped moderately with the other various social cognitive domains in the FEP group only, possibly indicating that FEP patients require partial dependence on other social cognitive skills to be able to infer mental states. As such, ToM should be a particular target area for remediation in FEP.

A relatively new intervention for social cognitive impairment in schizophrenia spectrum disorders, namely, Social Cognition and Interaction Training (SCIT; Penn, Roberts, Combs, & Sterne, 2007), has already shown efficacy in terms of remediation of social cognitive deficits, including ToM, and also improvements in social functioning (Roberts
& Penn, 2009; Roberts, Penn, Labate, Margolis, & Sterne, 2010). However, all of these studies have been conducted using inpatients and outpatients with established schizophrenia, thus little is known about the effectiveness of SCIT in remediating ToM and other social cognitive deficits in the early phase of illness. The feasibility of SCIT has recently been pilot-tested in a sample of FEP patients at Orygen Youth Health, with some evidence of improvement in social cognition and social functioning (Bartholomeusz et al., 2013). Although the authors concluded that SCIT appears to be a feasible intervention for use in FEP, the small sample size (N=12 recruited, with only 9 included in the analysis due to non-completers and those that could not be followed-up) limited the significance of the results. Furthermore, regarding social cognition specifically, significant improvements were only seen in emotion recognition and not in the other domains it is aimed to directly target (i.e. ToM and attributional style) (Bartholomeusz et al., 2013). Nonetheless, the results were promising and a larger trial is required in FEP populations to assess the effectiveness of SCIT-based programs.

The current research showed that various neurocognitive domains were moderately associated with social cognition in FEP, especially emotion recognition and social knowledge. These findings were similar to another recent study (Fanning et al., 2012), and suggest that social cognitive deficits may be better treated if neurocognition is also targeted in remediation programs. One such existing intervention is Cognitive Enhancement Therapy (CET) (Hogarty, Greenwald, & Eack, 2006), which is a comprehensive group-based rehabilitation program that directly targets both social cognitive and neurocognitive abilities. CET is comprised of two main components; i) 45 weekly social cognitive group sessions that include relevant psychoeducation topics,
exercises, feedback and homework, and ii) 16 graduated, computer-assisted training sessions aimed to improve the neurocognitive functions of attention, memory, and problem solving. Although a relatively new approach to improving functioning in schizophrenia, CET has already demonstrated sustained improvement in cognitive skills, especially with regard to social cognition, in established schizophrenia (Hogarty et al., 2006) and early schizophrenia (Eack et al., 2009) populations. Furthermore, the cognitive gains observed in the latter study seem to have led to improved social/vocational functioning and negative symptoms, as individuals who had received the alternative treatment (i.e. Enriched Supportive Therapy; EST) in the randomised-controlled trial did not show the same improvements in cognition or functioning.

In contrast to the literature that shows impaired neurocognition (Bowie, Reichenberg, Patterson, Heaton, & Harvey, 2006; Green et al., 2000; Green et al, 2004; Ventura et al., 2009) and social cognition (Brüne et al., 2010; Fett et al., 2011; Horan et al., 2012; Sullivan et al., 2013) contribute significantly to poor social functioning in psychosis and schizophrenia, the current research indicated otherwise. The present findings showed that psychopathology, and not cognition, predicted concurrent social functioning in FEP. This finding is more in line with two longitudinal studies that have implied neurocognition (Carlsson et al., 2006) and social cognition (Horan et al., 2012) are weakly associated with functioning in the early course of illness, however the relationships strengthen over time. As overall social cognition and neurocognition were not associated with the functional indicators in the current research, there is an implication that both symptoms and various areas of functioning (social, role, general) need to be assessed and treated in the early course of illness, rather than just a primary
focus on stabilisation of symptoms (as is the current first-line treatment approach for schizophrenia-spectrum disorders) and cognitive remediation programs. Even though the current research suggests social cognitive abilities are impaired in FEP, remediation efforts may be better placed as secondary interventions following treatment of symptoms and social dysfunction perhaps using other interventions. This research suggests that methods to improve social functioning, for example, initiatives such as social skills training through role-play or other psychosocial approaches, may be more effective in the earlier course of illness than specific cognitive training interventions.
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* References listed here correspond to the literature cited in Chapters 1, 2, 3, 4, 5 and 10 only. Chapters 6 to 9 contain their own reference lists as per manuscript submission.
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Original and Modified Versions
Appendix A: DSM-5 Diagnostic Criteria for Schizophrenia
DSM-5 Diagnostic Criteria for Schizophrenia

(Code: 295.90)

**Criterion A.** Two (or more) of the following, each present for a significant portion of time during a one-month period (or less if successfully treated). At least one of these must be (1), (2), or (3):

1. Delusions.
2. Hallucinations.
3. Disorganized speech (e.g., frequent derailment or incoherence).
4. Grossly disorganized or catatonic behavior.
5. Negative symptoms (i.e., diminished emotional expression or avolition).

**Criterion B.** For a significant portion of the time since the onset of the disturbance, level of functioning in one or more major areas, such as work, interpersonal relations, or self-care, is markedly below the level achieved prior to the onset (or when the onset is in childhood or adolescence, there is failure to achieve expected level of interpersonal, academic, or occupational functioning).

**Criterion C.** Continuous signs of the disturbance persist for at least 6 months. This 6-month period must include at least one month of symptoms (or less if successfully treated) that meet Criterion A (i.e., active-phase symptoms) and may include periods of prodromal or residual symptoms. During these prodromal or residual periods, the signs of the disturbance may be manifested by only negative symptoms or by two or more symptoms listed in Criterion A present in an attenuated form (e.g., odd beliefs, unusual perceptual experiences).

**Criterion D.** Schizoaffective disorder and depressive or bipolar disorder with psychotic features have been ruled out because either: 1) no major depressive or manic episodes have occurred concurrently with the active-phase symptoms, or 2) if mood episodes have
occurred during active-phase symptoms, they have been present for a minority of the total duration of the active and residual periods of the illness.

**Criterion E.** The disturbance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition.

**Criterion F.** If there is a history of autism spectrum disorder or a communication disorder of childhood onset, the additional diagnosis of schizophrenia is made only if prominent delusions or hallucinations, in addition to the other required symptoms of schizophrenia, are also present for at least 1 month (or less if successfully treated).

*Specify if:*

The following course specifiers are only to be used after a one-year duration of the disorder and if they are not in contradiction to the diagnostic course criteria.

**First episode, currently in acute episode:** First manifestation of the disorder meeting the defining diagnostic symptom and time criteria. An *acute episode* is a time period in which the symptom criteria are fulfilled.

**First episode, currently in partial remission:** *Partial remission* is a period of time during which an improvement after a previous episode is maintained and in which the defining criteria of the disorder are only partially fulfilled.

**First episode, currently in full remission:** *Full remission* is a period of time after a previous episode during which no disorder-specific symptoms are present.

**Multiple episodes, currently in acute episode:** Multiple episodes may be determined after a minimum of two episodes (i.e., after a first episode, a remission and a minimum of one relapse).

**Multiple episodes, currently in partial remission**

**Multiple episodes, currently in full remission**
**Continuous**: Symptoms fulfilling the diagnostic symptom criteria of the disorder are remaining for the majority of the illness course, with subthreshold symptom periods being very brief relative to the overall course.

**Unspecified**
Appendix B: MATRICS Consensus Cognitive Battery: Summary
<table>
<thead>
<tr>
<th>Cognitive Test</th>
<th>Cognitive domain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trail Making Test - Trails A</td>
<td>Processing speed</td>
</tr>
<tr>
<td>Brief Assessment of Cognition in Schizophrenia (BACS) – Symbol Coding</td>
<td>Processing speed</td>
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<tr>
<td>Category Fluency: Animal Naming</td>
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</tr>
<tr>
<td>Continuous Performance Test – Identical Pairs (CPT-IP)</td>
<td>Attention/vigilance</td>
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<tr>
<td>Wechsler Memory Scale – 3rd Edition (WMS-III): Spatial Span</td>
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<tr>
<td>Letter Number Span</td>
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<tr>
<td>Hopkins Verbal Learning Test – Revised (HVLT-R)</td>
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<tr>
<td>Neuropsychological Assessment Battery (NAB): Mazes</td>
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</tr>
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<td>Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT):</td>
<td>Social cognition</td>
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<td>Managing Emotions</td>
<td></td>
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</tbody>
</table>
Appendix C: Positive and Negative Syndrome Scale (PANSS)
THE POSITIVE AND NEGATIVE SYNDROME SCALE (PANSS)

Each of the 30-items of the PANSS (see below for descriptions and rating criteria) is rated on a seven point scale that represents increasing levels of psychopathology.

Positive Scale (P)

P1. DELUSIONS - Beliefs which are unfounded, unrealistic and idiosyncratic.

Basis for rating - Thought content expressed in the interview and its influence on social relations and behaviour.

1  Absent - Definition does not apply
2  Minimal - Questionable pathology; may be at the upper extreme of normal limits
3  Mild - Presence of one or two delusions which are vague, uncrystallised and not tenaciously held. Delusions do not interfere with thinking, social relations or behaviour.
4  Moderate - Presence of either a kaleidoscopic array of poorly formed, unstable delusions or a few well-formed delusions that occasionally interfere with thinking, social relations or behaviour.
5  Moderate Severe - Presence of numerous well-formed delusions that are tenaciously held and occasionally interfere with thinking, social relations and behaviour.
6  Severe - Presence of a stable set of delusions which are crystallised, possibly systematised, tenaciously held and clearly interfere with thinking, social relations and behaviour.
7  Extreme - Presence of a stable set of delusions which are either highly systematised or very numerous, and which dominate major facets of the patient’s life. This frequently results in inappropriate and irresponsible action, which may even jeopardise the safety of the patient or others.

P2. CONCEPTUAL DISORGANISATION - Disorganised process of thinking characterised by disruption of goal-directed sequencing, e.g. circumstantiality, loose associations, tangentiality, gross illogicality or thought block.

Basis for rating - Cognitive-verbal processes observed during the course of interview.

1  Absent - Definition does not apply
2  Minimal - Questionable pathology; may be at the upper extreme of normal limits
3  Mild - Thinking is circumstantial, tangential or paralogical. There is some difficulty in directing thoughts towards a goal, and some loosening of associations may be
evidenced under pressure.

4 Moderate - Able to focus thoughts when communications are brief and structured, but becomes loose or irrelevant when dealing with more complex communications or when under minimal pressure.

5 Moderate Severe - Generally has difficulty in organising thoughts, as evidenced by frequent irrelevancies, disconnectedness or loosening of associations even when not under pressure.

6 Severe - Thinking is seriously derailed and internally inconsistent, resulting in gross irrelevancies and disruption of thought processes, which occur almost constantly.

7 Extreme - Thoughts are disrupted to the point where the patient is incoherent. There is marked loosening of associations, which result in total failure of communication, e.g. “word salad” or mutism.

P3. HALLUCINATIONS - Verbal report or behaviour indicating perceptions, which are not generated by external stimuli. These may occur in the auditory, visual, olfactory or somatic realms.

Basis for rating - Verbal report and physical manifestations during the course of interview, as well as reports of behaviour by primary care workers or family.

1 Absent - Definition does not apply

2 Minimal - Questionable pathology; may be at the upper extreme of normal limits

3 Mild - One or two clearly formed but infrequent hallucinations, or else a number of vague abnormal perceptions, which do not result in distortions of thinking or behaviour.

4 Moderate - Hallucinations occur frequently but not continuously, and the patient’s thinking and behaviour are only affected to a minor extent.

5 Moderate Severe - Hallucinations occur frequently, may involve more than one sensory modality, and tend to distort thinking and/or disrupt behaviour. Patient may have a delusional interpretation of these experiences and respond to them emotionally and, on occasion, verbally as well.

6 Severe - Hallucinations are present almost continuously, causing major disruption of thinking and behaviour. Patient treats these as real perceptions, and functioning is impeded by frequent emotional and verbal responses to them.

7 Extreme - Patient is almost totally preoccupied with hallucinations, which virtually dominate thinking and behaviour. Hallucinations are provided a rigid delusional interpretation and provoke verbal and behavioural responses, including obedience to command hallucinations.
P4. **EXCITEMENT** - Hyperactivity as reflected in accelerated motor behaviour, heightened responsivity to stimuli, hypervigilance or excessive mood lability.

**Basis for rating** - Behavioural manifestations during the course of interview, as well as reports of behaviour by primary care workers or family.

1. **Absent** - Definition does not apply
2. **Minimal** - Questionable pathology; may be at the upper extreme of normal limits
3. **Mild** - Tends to be slightly agitation, hypervigilant or mildly overaroused throughout the interview, but without distinct episodes of excitement or marked mood lability. Speech may be slightly pressured.
4. **Moderate** - Agitation or overarousal is clearly evident throughout the interview, affecting speech and general mobility, or episodic outbursts occur sporadically.
5. **Moderate Severe** - Significant hyperactivity or frequent outbursts of motor activity are observed, making it difficult for the patient to sit still for longer than several minutes at any given time.
6. **Severe** - Marked excitement dominates the interview, delimits attention, and to some extent affects personal functions such as eating or sleeping.
7. **Extreme** - marked excitement seriously interferes in eating and sleeping and makes interpersonal interactions virtually impossible. Acceleration of speech and motor activity may result in incoherence and exhaustion.

P5. **GRANDIOSITY** - Exaggerated self-opinion and unrealistic convictions of superiority, including delusions of extraordinary abilities, wealth, knowledge, fame, power and moral righteousness.

**Basis for rating** - Thought content expressed in the interview and its influence on behaviour.

1. **Absent** - Definition does not apply
2. **Minimal** - Questionable pathology; may be at the upper extreme of normal limits
3. **Mild** - Some expansiveness or boastfulness is evident, but without clear-cut grandiose delusions.
4. **Moderate** - Feels distinctly and unrealistically superior to others. Some poorly formed delusions about special status or abilities may be present but are not acted upon.
5. **Moderate Severe** - Clear-cut delusions concerning remarkable abilities, status or power are expressed and influence attitude but not behaviour.
6. **Severe** - Clear-cut delusions of remarkable superiority involving more than one parameter (wealth, knowledge, fame, etc.) are expressed, notably influence interactions and may be acted upon.
7 Extreme - Thinking, interactions and behaviour are dominated by multiple delusions of amazing ability, wealth, knowledge, fame, power and/or moral stature, which may take on a bizarre quality.

P6. SUSPICIOUSNESS/PERSECUTION - Unrealistic or exaggerated ideas of persecution, as reflected in guardedness, a distrustful attitude, suspicious hypervigilance, or frank delusions that others mean harm.

Basis for rating – Thought content expressed in the interview and its influence on behaviour.

1 Absent - Definition does not apply
2 Minimal - Questionable pathology; may be at the upper extreme of normal limits
3 Mild - Presents a guarded or even openly distrustful attitude, but thoughts, interactions and behaviour are minimally affected.
4 Moderate - Distrustfulness is clearly evident and intrudes on the interview and/or behaviour, but there is no evidence of persecutory delusions. Alternatively, there may be indication of loosely formed persecutory delusions, but these do not seem to affect the patient’s attitude or interpersonal relations.
5 Moderate Severe - Patient shows marked distrustfulness, leading to major disruption of interpersonal relations, or else there are clear-cut persecutory delusions that have limited impact on interpersonal relations and behaviour.
6 Severe - Clear-cut pervasive delusions of persecution which may be systematised and significantly interfere in interpersonal relations.
7 Extreme - A network of systematised persecutory delusions dominates the patient’s thinking, social relations and behaviour.

P7. HOSTILITY - Verbal and nonverbal expressions of anger and resentment, including sarcasm, passive-aggressive behaviour, verbal abuse and assaultiveness.

Basis for rating – Interpersonal behaviour observed during the interview and reports by primary care workers or family.

1 Absent - Definition does not apply
2 Minimal - Questionable pathology; may be at the upper extreme of normal limits
3 Mild - Indirect or restrained communication of anger, such as sarcasm, disrespect, hostile expressions and occasional irritability.
4 Moderate - Presents an overtly hostile attitude, showing frequent irritability and direct expression of anger or resentment.
5 **Moderate Severe** - Patient is highly irritable and occasionally verbally abusive or threatening.

6 **Severe** - Uncooperativeness and verbal abuse or threats notably influence the interview and seriously impact upon social relations. Patient may be violent and destructive but is not physically assaultive towards others.

7 **Extreme** - Marked anger results in extreme uncooperativeness, precluding other interactions, or in episode(s) of physical assault towards others.

**Negative Scale (N)**

**N1. BLUNTED AFFECT** - Diminished emotional responsiveness as characterised by a reduction in facial expression, modulation of feelings and communicative gestures.

**Basis for rating** - Observation of physical manifestations of affective tone and emotional responsiveness during the course of the interview.

1 **Absent** - Definition does not apply

2 **Minimal** - Questionable pathology; may be at the upper extreme of normal limits

3 **Mild** - Changes in facial expression and communicative gestures seem to be stilted, forced, artificial or lacking in modulation.

4 **Moderate** - Reduced range of facial expression and few expressive gestures result in a dull appearance

5 **Moderate Severe** - Affect is generally ‘flat’ with only occasional changes in facial expression and a paucity of communicative gestures.

6 **Severe** - Marked flatness and deficiency of emotions exhibited most of the time. There may be unmodulated extreme affective discharges, such as excitement, rage or inappropriate uncontrolled laughter.

7 **Extreme** – Changes in facial expression and evidence of communicative gestures are virtually absent. Patient seems constantly to show a barren or ‘wooden’ expression.

**N2. EMOTIONAL WITHDRAWAL** - Lack of interest in, involvement with, and affective commitment to life’s events.

**Basis for rating** - Reports of functioning from primary care workers or family and observation of interpersonal behaviour during the course of the interview.

1 **Absent** - Definition does not apply

2 **Minimal** - Questionable pathology; may be at the upper extreme of normal limits
3 Mild - Usually lack initiative and occasionally may show deficient interest in surrounding events.

4 Moderate - Patient is generally distanced emotionally from the milieu and its challenges but, with encouragement, can be engaged.

5 Moderate Severe - Patient is clearly detached emotionally from persons and events in the milieu, resisting all efforts at engagement. Patient appears distant, docile and purposeless but can be involved in communication at least briefly and tends to personal needs, sometimes with assistance.

6 Severe - Marked deficiency of interest and emotional commitment results in limited conversation with others and frequent neglect of personal functions, for which the patient requires supervision.

7 Extreme – Patient is almost totally withdrawn, uncommunicative and neglectful of personal needs as a result of profound lack of interest and emotional commitment.

N3. POOR RAPPORT - Lack of interpersonal empathy, openness in conversation and sense of closeness, interest or involvement with the interviewer. This is evidenced by interpersonal distancing and reduced verbal and nonverbal communication.

Basis for rating - Interpersonal behaviour during the course of the interview.

1 Absent - Definition does not apply

2 Minimal - Questionable pathology; may be at the upper extreme of normal limits

3 Mild - Conversation is characterised by a stilted, strained or artificial tone. It may lack emotional depth or tend to remain on an impersonal, intellectual plane.

4 Moderate - Patient typically is aloof, with interpersonal distance quite evident. Patient may answer questions mechanically, act bored, or express disinterest.

5 Moderate Severe - Disinvolvement is obvious and clearly impedes the productivity of the interview. Patient may tend to avoid eye or face contact.

6 Severe - Patient is highly indifferent, with marked interpersonal distance. Answers are perfunctory, and there is little nonverbal evidence of involvement. Eye and face contact are frequently avoided.

7 Extreme - Patient is totally uninvolved with the interviewer. Patient appears to be completely indifferent and consistently avoids verbal and nonverbal interactions during the interview.

N4. PASSIVE/APATHETIC SOCIAL WITHDRAWAL - Diminished interest and initiative in social interactions due to passivity, apathy, anergy or avolition. This leads to reduced interpersonal involvements and neglect of activities of daily living.
**Basis for rating** – Reports on social behaviour from primary care workers or family.

1. **Absent** - Definition does not apply
2. **Minimal** - Questionable pathology; may be at the upper extreme of normal limits
3. **Mild** - Shows occasional interest in social activities but poor initiative. Usually engages with others only when approached first by them.
4. **Moderate** – Passively goes along with most social activities but in a disinterested or mechanical way. Tends to recede into the background.
5. **Moderate Severe** - Passively participates in only a minority of activities and shows virtually no interest or initiative. Generally spends little time with others.
6. **Severe** - Tends to be apathetic and isolated, participating very rarely in social activities and occasionally neglecting personal needs. Has very few spontaneous social contacts.
7. **Extreme** – Profoundly apathetic, socially isolated and personally neglectful.

**N5. DIFFICULTY IN ABSTRACT THINKING** - Impairment in the use of the abstract-symbolic mode of thinking, as evidenced by difficulty in classification, forming generalisations and proceeding beyond concrete or egocentric thinking in problem-solving tasks.

**Basis for rating** - Responses to questions on similarities and proverb interpretation, and use of concrete vs. abstract mode during the course of the interview.

1. **Absent** - Definition does not apply
2. **Minimal** - Questionable pathology; may be at the upper extreme of normal limits
3. **Mild** - Tends to give literal or personalised interpretations to the more difficult proverbs and may have some problems with concepts that are fairly abstract or remotely related.
4. **Moderate** - Often utilises a concrete mode. Has difficulty with most proverbs and some categories. Tends to be distracted by functional aspects and salient features.
5. **Moderate Severe** - Deals primarily in a concrete mode, exhibiting difficulty with most proverbs and many categories.
6. **Severe** - Unable to grasp the abstract meaning of any proverbs or figurative expressions and can formulate classifications for only the most simple of similarities. Thinking is either vacuous or locked into functional aspects, salient features and idiosyncratic interpretations.
7. **Extreme** - Can use only concrete modes of thinking. Shows no comprehension of proverbs, common metaphors or similes, and simple categories. Even salient and
functional attributes do not serve as a basis for classification. This rating may apply to those who cannot interact even minimally with the examiner due to marked cognitive impairment.

N6. LACK OF SPONTANEITY AND FLOW OF CONVERSATION - Reduction in the normal flow of communication associated with apathy, avolition, defensiveness or cognitive deficit. This is manifested by diminished fluidity and productivity of the verbal interactional process.

Basis for rating - Cognitive-verbal processes observed during the course of interview.

1. **Absent** - Definition does not apply
2. **Minimal** - Questionable pathology; may be at the upper extreme of normal limits
3. **Mild** – Conversation shows little initiative. Patient’s answers tend to be brief and unembellished, requiring direct and leading questions by the interviewer.
4. **Moderate** – Conversation lacks free flow and appears uneven or halting. Leading questions are frequently needed to elicit adequate responses and proceed with conversation.
5. **Moderate Severe** - Patient shows a marked lack of spontaneity and openness, replying to the interviewer’s questions with only one or two brief sentences.
6. **Severe** - Patient’s responses are limited mainly to a few words or short phrases intended to avoid or curtail communication. (e.g. “I don’t know”, “I’m not at liberty to say”). Conversation is seriously impaired as a result and the interview is highly unproductive.
7. **Extreme** - Verbal output is restricted to, at most, an occasional utterance, making conversation not possible.

N7. STEREOTYPED THINKING - Decreased fluidity, spontaneity and flexibility of thinking, as evidenced in rigid, repetitious or barren thought content.

Basis for rating - Cognitive-verbal processes observed during the interview.

1. **Absent** - Definition does not apply
2. **Minimal** - Questionable pathology; may be at the upper extreme of normal limits
3. **Mild** - Some rigidity shown in attitude or beliefs. Patient may refuse to consider alternative positions or have difficulty in shifting from one idea to another.
4. **Moderate** - Conversation revolves around a recurrent theme, resulting in difficulty in shifting to a new topic.
5. **Moderate Severe** - Thinking is rigid and repetitious to the point that, despite the interviewer’s efforts, conversation is limited to only two or three dominating topics.
6 **Severe** – Uncontrolled repetition of demands, statements, ideas or questions which severely impairs conversation.

7 **Extreme** - Thinking, behaviour and conversation are dominated by constant repetition of fixed ideas or limited phrases, leading to gross rigidity, inappropriateness and restrictiveness of patient’s communication.

**General Psychopathology Scale (G)**

**G1. SOMATIC CONCERN** - Physical complaints or beliefs about bodily illness or malfunctions. This may range from a vague sense of ill being to clear-cut delusions of catastrophic physical disease.

**Basis for rating** - Thought content expressed in the interview.

1 **Absent** - Definition does not apply

2 **Minimal** - Questionable pathology; may be at the upper extreme of normal limits

3 **Mild** - Distinctly concerned about health or bodily malfunction, but there is no delusional conviction and over-concern can be allayed by reassurance.

4 **Moderate** - Complains about poor health or bodily malfunction, but there is no delusional conviction and over-concern can be allayed by reassurance.

5 **Moderate Severe** - Patient expresses numerous or frequent complaints about physical illness or bodily malfunction, or else patient reveals one or two clear-cut delusions involving these themes but is not preoccupied by them.

6 **Severe** - Patient is preoccupied by one or a few clear-cut delusions about physical disease or organic malfunction, but affect is not fully immersed in these themes, and thoughts can be diverted by the interviewer with some effort.

7 **Extreme** – Numerous and frequently reported somatic delusions, or only a few somatic delusions of a catastrophic nature, which totally dominate the patient’s affect or thinking.

**G2. ANXIETY** - Subjective experience of nervousness, worry, apprehension or restlessness, ranging from excessive concern about the present or future to feelings of panic.

**Basis for rating** - Verbal report during the course of interview and corresponding physical manifestations.

1 **Absent** - Definition does not apply

2 **Minimal** - Questionable pathology; may be at the upper extreme of normal limits

3 **Mild** - Expresses some worry, over-concern or subjective restlessness, but no somatic and behavioural consequences are reported or evidenced.
4 Moderate - Patient reports distinct symptoms of nervousness, which are reflected in mild physical manifestations such as fine hand tremor and excessive perspiration.

5 Moderate Severe - Patient reports serious problems of anxiety, which have significant physical and behavioural consequences, such as, marked tension, poor concentration, palpitations or impaired sleep.

6 Severe - Subjective state of almost constant fear associated with phobias, marked restlessness or numerous somatic manifestations.

7 Extreme - Patient’s life is seriously disrupted by anxiety, which is present almost constantly and at times reaches panic proportion or is manifested in actual panic attacks.

G3. GUILT FEELINGS - Sense of remorse or self-blame for real or imagined misdeeds in the past.

Basis for rating - Verbal report of guilt feelings during the course of interview and the influence on attitudes and thoughts.

1 Absent - Definition does not apply

2 Minimal - Questionable pathology; may be at the upper extreme of normal limits

3 Mild - Questioning elicits a vague sense of guilt or self-blame for a minor incident, but the patient clearly is not overly concerned.

4 Moderate - Patient expresses distinct concern over his responsibility for a real incident in his life but is not preoccupied with it and attitude and behaviour are essentially unaffected.

5 Moderate Severe - Patient expresses a strong sense of guilt associated with self-deprecation or the belief that he deserves punishment. The guilt feelings may have a delusional basis, may be volunteered spontaneously, may be a source of preoccupation and/or depressed mood, and cannot be allayed readily by the interviewer.

6 Severe - Strong ideas of guilt take on a delusional quality and lead to an attitude of hopelessness or worthlessness. The patient believes he should receive harsh sanctions as such punishment.

7 Extreme - Patient’s life is dominated by unshakable delusions of guilt, for which he feels deserving of drastic punishment, such as life imprisonment, torture, or death. There may be associated suicidal thoughts or attribution of others’ problems to one’s own past misdeeds.

G5. MANNERISMS AND POSTURING – Unnatural movements or posture as characterised be an awkward, stilted, disorganised, or bizarre appearance.
**Basis for rating** - Observation of physical manifestations during the course of interview as well as reports from primary care workers or family.

1. **Absent** - Definition does not apply
2. **Minimal** - Questionable pathology; may be at the upper extreme of normal limits
3. **Mild** - Slight awkwardness in movements or minor rigidity of posture
4. **Moderate** – Movements are notably awkward or disjointed, or an unnatural posture is maintained for brief periods.
5. **Moderate Severe** - Occasional bizarre rituals or contorted posture are observed, or an abnormal position is sustained for extended periods.
6. **Severe** - Frequent repetition of bizarre rituals, mannerisms or stereotyped movements, or a contorted posture is sustained for extended periods.
7. **Extreme** - Functioning is seriously impaired by virtually constant involvement in ritualistic, manneristic, or stereotyped movements or by an unnatural fixed posture which is sustained most of the time.

**G6. DEPRESSION** - Feelings of sadness, discouragement, helplessness and pessimism.

**Basis for rating** - Verbal report of depressed mood during the course of interview and its observed influence on attitude and behaviour.

1. **Absent** - Definition does not apply
2. **Minimal** - Questionable pathology; may be at the upper extreme of normal limits
3. **Mild** - Expresses some sadness of discouragement only on questioning, but there is no evidence of depression in general attitude or demeanor.
4. **Moderate** - Distinct feelings of sadness or hopelessness, which may be spontaneously divulged, but depressed mood has no major impact on behaviour or social functioning and the patient usually can be cheered up.
5. **Moderate Severe** - Distinctly depressed mood is associated with obvious sadness, pessimism, loss of social interest, psychomotor retardation and some interference in appetite and sleep. The patient cannot be easily cheered up.
6. **Severe** - Markedly depressed mood is associated with sustained feelings of misery, occasional crying, hopelessness and worthlessness. In addition, there is major interference in appetite and or sleep as well as in normal motor and social functions, with possible signs of self-neglect.
7. **Extreme** - Depressive feelings seriously interfere in most major functions. The manifestations include frequent crying, pronounced somatic symptoms, impaired
concentration, psychomotor retardation, social disinterest, self neglect, possible depressive or nihilistic delusions and/or possible suicidal thoughts or action.

G7. **MOTOR RETARDATION** – Reduction in motor activity as reflected in slowing or lessening or movements and speech, diminished responsiveness of stimuli, and reduced body tone.

**Basis for rating** - Manifestations during the course of interview as well as reports by primary care workers as well as family.

1. **Absent** - Definition does not apply
2. **Minimal** - Questionable pathology; may be at the upper extreme of normal limits
3. **Mild** - Slight but noticeable diminution in rate of movements and speech. Patient may be somewhat underproductive in conversation and gestures.
4. **Moderate** - Patient is clearly slow in movements, and speech may be characterised by poor productivity including long response latency, extended pauses or slow pace.
5. **Moderate Severe** – A marked reduction in motor activity renders communication highly unproductive or delimits functioning in social and occupational situations. Patient can usually be found sitting or lying down.
6. **Severe** - Movements are extremely slow, resulting in a minimum of activity and speech. Essentially the day is spent sitting idly or lying down.
7. **Extreme** - Patient is almost completely immobile and virtually unresponsive to external stimuli.

G8. **UNCOOPERATIVENESS** - Active refusal to comply with the will of significant others, including the interviewer, hospital staff or family, which may be associated with distrust, defensiveness, stubbornness, negativism, rejection of authority, hostility or belligerence.

**Basis for rating** - Interpersonal behaviour observed during the course of the interview as well as reports by primary care workers or family.

1. **Absent** - Definition does not apply
2. **Minimal** - Questionable pathology; may be at the upper extreme of normal limits
3. **Mild** - Complies with an attitude of resentment, impatience, or sarcasm. May inoffensively object to sensitive probing during the interview.
4 Moderate: Occasional outright refusal to comply with normal social demands, such as making own bed, attending scheduled programmes, etc. The patient may project a hostile, defensive or negative attitude but usually can be worked with.

5 Moderate Severe: Patient frequently is incompliant with the demands of his milieu and may be characterised by other as an “outcast” or having “a serious attitude problem”. Uncooperativeness is reflected in obvious defensiveness or irritability with the interviewer and possible unwillingness to address many questions.

6 Severe: Patient is highly uncooperative, negativistic and possibly also belligerent. Refuses to comply with the most social demands and may be unwilling to initiate or conclude the full interview.

7 Extreme: Active resistance seriously impact on virtually all major areas of functioning. Patient may refuse to join in any social activities, tend to personal hygiene, converse with family or staff and participate even briefly in an interview.

G9. Unusual Thought Content: Thinking characterised by strange, fantastic or bizarre ideas, ranging from those, which are remote or atypical to those, which are distorted, illogical and patently absurd.

Basis for rating: Thought content expressed during the course of interview.

1 Absent: Definition does not apply

2 Minimal: Questionable pathology; may be at the upper extreme of normal limits

3 Mild: Thought content is somewhat peculiar, or idiosyncratic, or familiar ideas are framed in an odd context.

4 Moderate: Ideas are frequently distorted and occasionally seem quite bizarre.

5 Moderate Severe: Patient expresses many strange and fantastic thoughts, (e.g., being the adopted son of a king, being an escapee from death row), or some which are patently absurd (e.g., having hundreds of children, receiving radio messages from outer space from a tooth filling).

6 Severe: Patient expresses many illogical or absurd ideas or some, which have a distinctly bizarre quality (e.g. having three heads, being a visitor from another planet).

7 Extreme: Thinking is replete with absurd, bizarre and grotesque ideas.

G10. Disorientation: Lack of awareness of one’s relationship to the milieu, including persons, place and time, which may be due to confusion or withdrawal.

Basis for rating: Responses to interview questions on orientation.

1 Absent: Definition does not apply
2 Minimal - Questionable pathology; may be at the upper extreme of normal limits

3 Mild - General orientation is adequate but there is some difficulty with specifics. For example, patient knows his location but not the street address, knows hospital staff names but not their functions, knows the month but confuses the day of the week with an adjacent day, or errs in the date by more than two days. There may be narrowing of interest evidenced by familiarity with the immediate but not extended milieu, such as ability to identify staff but not the mayor, governor, or president.

4 Moderate - Only partial success in recognising persons, places and time. For example, patient knows he is in a hospital but not its name, knows the name of the city but not the borough or district, knows the name of his primary therapist but not many other direct care workers, knows the year or season but not sure of the month.

5 Moderate Severe - Considerable failure in recognising persons, place and time. Patient has only a vague notion of where he is and seems unfamiliar with most people in his milieu. He may identify the year correctly or nearly but not know the current month, day of week or even the season.

6 Severe – Marked failure in recognizing persons, place and time. For example, patient has no knowledge of his whereabouts, confuses the date by more than one year, can name only one or two individuals in his current life.

7 Extreme - Patient appears completely disorientated with regard to persons, place and time. There is gross confusion or total ignorance about one’s location, the current year and even the most familiar people, such as parents, spouse, friends and primary therapist.

G11. POOR ATTENTION - Failure in focused alertness manifested by poor concentration, distractibility from internal and external stimuli, and difficulty in harnessing, sustaining or shifting focus to new stimuli.

Basis for rating – Manifestations during the course of interview.

1 Absent - Definition does not apply

2 Minimal - Questionable pathology; may be at the upper extreme of normal limits

3 Mild - Limited concentration evidenced by occasional vulnerability to distraction and faltering attention toward the end of the interview.

4 Moderate - Conversation is affected by the tendency to be easily distracted, difficulty in long sustaining concentration on a given topic, or problems in shifting attention to new topics.

5 Moderate Severe - Conversation is seriously hampered by poor concentration, distractibility, and difficulty in shifting focus appropriately.
6  **Severe** - Patient’s attention can be harnessed for only brief moments or with great effort, due to marked distraction by internal or external stimuli.

7  **Extreme** - Attention is so disrupted that even brief conversation is not possible.

**G12. LACK OF JUDGEMENT AND INSIGHT** - Impaired awareness or understanding of one’s own psychiatric condition and life situation. This is evidenced by failure to recognise past or present psychiatric illness or symptoms, denial of need for psychiatric hospitalisation or treatment, decisions characterised by poor anticipation or consequences, and unrealistic short-term and long-range planning.

**Basis for rating** – Thought content expressed during the interview.

1  **Absent** - Definition does not apply

2  **Minimal** - Questionable patholgy; may be at the upper extreme of normal limits

3  **Mild** - Recognises having a psychiatric disorder but clearly underestimates its seriousness, the implications for treatment, or the importance of taking measures to avoid relapse. Future planning may be poorly conceived.

4  **Moderate** - Patient shows only a vague or shallow recognition of illness. There may be fluctuations in acknowledgement of being ill or little awareness of major symptoms which are present, such as delusions, disorganised thinking, suspiciousness and social withdrawal. The patient may rationalise the need for treatment in terms of its relieving lesser symptoms, such as anxiety, tension and sleep difficulty.

5  **Moderate Severe** - Acknowledges past but not present psychiatric disorder. If challenged, the patient may concede the presence of some unrelated or insignificant symptoms, which tend to be explained away by gross misinterpretation or delusional thinking. The need for psychiatric treatment similarly goes unrecognised.

6  **Severe** - Patient denies ever having had a psychiatric disorder. He disavows the presence of any psychiatric symptoms in the past or present and, though compliant, denies the need for treatment and hospitalisation.

7  **Extreme** - Empathic denial of past and present psychiatric illness. Current hospitalisation and treatment are given a delusional interpretation (e.g. as punishment from misdeeds, as persecution by tormentors, etc), and the patient thus refuse to cooperate with therapists, medication or other aspects of treatment.

**G13. DISTURBANCE OF VOLITION** – Disturbance in the wilful initiation, sustenance and control of one’s thoughts, behaviour, movements and speech.

**Basis for rating** - Thought content and behaviour manifested in the course of interview.

1  **Absent** - Definition does not apply
2 **Minimal** - Questionable pathology; may be at the upper extreme of normal limits

3 **Mild** - There is evidence of some indecisiveness in conversation and thinking, which may impede verbal and cognitive processes to a minor extent.

4 **Moderate** - Patient is often ambivalent and shows clear difficulty in reaching decisions. Conversation may be marred by alteration in thinking, and in consequence, verbal and cognitive functioning are clearly impaired.

5 **Moderate Severe** - Disturbance of volition interferes in thinking as well as behaviour. Patient shows pronounced indecision that impedes the initiation and continuation of social and motor activities, and which also may be evidence in halting speech.

6 **Severe** - Disturbance of volition interferes in the execution of simple automatic motor functions, such as dressing or grooming, and markedly affects speech.

7 **Extreme** – Almost complete failure of volition is manifested by gross inhibition of movement and speech resulting in immobility and/or mutism.

**G14. POOR IMPULSE CONTROL** – Disordered regulation and control of action on inner urges, resulting in sudden, unmodulated, arbitrary or misdirected discharge of tension and emotions without concern about consequences.

**Basis for rating** – Behaviour during the course of interview and reported by primary care workers or family.

1 **Absent** - Definition does not apply

2 **Minimal** - Questionable pathology; may be at the upper extreme of normal limits

3 **Mild** - Patient tends to be easily angered and frustrated when facing stress or denied gratification but rarely acts on impulse.

4 **Moderate** - Patient gets angered and verbally abusive with minimal provocation. May be occasionally threatening, destructive, or have one or two episodes involving physical confrontation or a minor brawl.

5 **Moderate Severe** - Patient exhibits repeated impulsive episodes involving verbal abuse, destruction of property, or physical threats. There may be one or two episodes involving serious assault, for which the patient requires isolation, physical restraint, or p.r.n. sedation.

6 **Severe** - Patient frequently is impulsive aggressive, threatening, demanding, and destructive, without any apparent consideration of consequences. Shows assaultive behaviour and may also be sexually offensive and possibly respond behaviourally to hallucinatory commands.
7 **Extreme** - Patient exhibits homicidal, sexual assaults, repeated brutality, or self-destructive behaviour. Requires constant direct supervision or external constraints because of inability to control dangerous impulses.

**G15. PREOCCUPATION** - Absorption with internally generated thoughts and feelings and with autistic experiences to the detriment of reality orientation and adaptive behaviour.

**Basis for rating** - Interpersonal behaviour observed during the course of interview.

1 **Absent** - Definition does not apply

2 **Minimal** - Questionable pathology; may be at the upper extreme of normal limits

3 **Mild** - Excessive involvement with personal needs or problems, such that conversation veers back to egocentric themes and there is diminished concerned exhibited toward others.

4 **Moderate** - Patient occasionally appears self-absorbed, as if daydreaming or involved with internal experiences, which interferes with communication to a minor extent.

5 **Moderate Severe** - Patient often appears to be engaged in autistic experiences, as evidenced by behaviours that significantly intrude on social and communicational functions, such as the presence of a vacant stare, muttering or talking to oneself, or involvement with stereotyped motor patterns.

6 **Severe** - Marked preoccupation with autistic experiences, which seriously delimits concentration, ability to converse, and orientation to the milieu. The patient frequently may be observed smiling, laughing, muttering, talking, or shouting to himself.

7 **Extreme** - Gross absorption with autistic experiences, which profoundly affects all major realms of behaviour. The patient constantly may be responding verbally or behaviourally to hallucinations and show little awareness of other people or the external milieu.

**G16. ACTIVE SOCIAL AVOIDANCE** - Diminished social involvement associated with unwarranted fear, hostility, or distrust.

**Basis for rating** - Reports of social functioning primary care workers or family.

1 **Absent** - Definition does not apply

2 **Minimal** - Questionable pathology; may be at the upper extreme of normal limits

3 **Mild** - Patient seems ill at ease in the presence of others of others and prefers to spend time alone, although he participates in social functions when required.
4 **Moderate** - Patient begrudgingly attends all or most social activities but may need to be persuaded or may terminate prematurely on account of anxiety, suspiciousness, or hostility.

5 **Moderate Severe** - Patient fearfully or angrily keeps away from many social interactions despite others’ efforts to engage him. Tends to spend unstructured time alone.

6 **Severe** - Patient participates in very few social activities because of fear, hostility, or distrust. When approached, the patient shows a strong tendency to break off interactions, and generally he tends to isolate himself from others.

7 **Extreme** - Patient cannot be engaged in social activities because of pronounced fears, hostility, or persecutory delusions. To the extent possible, he avoids all interactions and remains isolated from others.
Appendix D: Methodology of a Three-Factor Model of Symptomatology in Schizophrenia using the PANSS
Calculation of Three Symptom Dimensions:
Positive, Negative and Disorganised

Positive, negative and disorganised symptom scores were generated using the Positive and Negative Syndrome Scale (PANSS) data. Specifically, certain PANSS items comprised each of the symptom dimensions, with the mean score of these items forming the sub-score. Outlined below are the PANSS items that corresponded to each particular symptom domain.

Positive symptom dimension (reality distortion):

- P1. Delusions
- P3. Hallucinations
- P5. Grandiosity
- P6. Suspiciousness/Persecution
- G9. Unusual Thought Content

Negative symptom dimension (psychomotor poverty):

- N1. Blunted Affect
- N2. Emotional Withdrawal
- N3. Poor Rapport
- N4. Passive Social Withdrawal
- N6. Lack of Spontaneity and Flow of Conversation
- G7. Motor Retardation
- G16. Active Social Avoidance
Disorganised symptom dimension (disorganisation):

- P2. Conceptual Disorganisation
- N5. Difficulty in Abstract Thinking
- G10. Disorientation
- G11. Poor Attention

Example:

Consider the symptom domain of disorganisation. If a patient had the individual scores of: ‘conceptual disorganisation’ (3), ‘difficulty in abstract thinking’ (4), ‘disorientation’ (3), and ‘poor attention’ (4), the overall score for disorganised symptom dimension would be 3.5. That is, the mean of the individual scores.
Appendix E: Global Functioning Scales: Role and Social
GLOBAL FUNCTIONING: ROLE SCALE

Superior role functioning

10 Independently maintains superior functioning in demanding roles. Obtains only superior performance evaluations at competitive work placement. Obtains all A’s in mainstream school. Generates, organizes, and completes all homemaking tasks with ease.

Above average role functioning

9 Independently maintains very good functioning in demanding roles. Rarely absent or unable to perform. Obtains good to superior performance evaluations at competitive work placement. Obtains grades in A and B range in all courses in mainstream school. Generates, organizes, and completes all homemaking tasks.

Good role functioning

8 Independently maintains good role functioning in demanding roles. Occasionally falls behind on tasks but always catches up; obtains satisfactory performance evaluations at competitive work placement; obtains grades of C and above in mainstream school; occasional difficulty generating or organizing homemaking tasks; or maintains above average performance with minimal support (e.g., tutoring, reduced academic course load at 4-year university, attends community college, may receive additional guidance at work less than 1-2 times a week). Receives As and Bs, good work/school evaluations, and completes all tasks with this level of support.

Mild impairment in role functioning

7 Mildly impaired functioning in demanding roles independently. Frequently behind on tasks or unable to perform; frequently obtains poor performance evaluations at competitive work placement or grades of Ds or better in mainstream school; frequent difficulty generating or organizing homemaking tasks; or maintains good performance with minimal support (e.g., minimal accommodations in general education classroom, receives additional guidance/support at work 1-2 times a week). Receives Cs or higher, satisfactory work/school evaluations, and completes most homemaking tasks with this level of support.

Moderate impairment in role functioning

6 Moderately impaired independently. May receive occasional F in mainstream courses, persistently poor performance evaluations at competitive work placement; may change jobs because of poor performance, persistent difficulty generating, or organizing homemaking tasks; or requires partial support (some resource or special education courses, receives guidance/support at work 2+ times per week). May require less demanding or part-time jobs and/or some supervision in home environment but functions well or adequately given these supports (may fall behind but eventually completes assigned tasks, obtains satisfactory evaluations at work or passing grades in school).

Serious Impairment in Role Functioning

5 Serious impairment independently. Failing multiple courses in mainstream school, may lose job, or unable to complete most homemaking tasks independently; or in entirely special education classes, requires less demanding job/daily support or guidance, may require vocational rehabilitation, and/or some supervision in home environment but maintains “above average” performance—receives As and Bs, good evaluations at work/school, completes all tasks.

Major impairment in role functioning

4 Very serious impairment independently. All Fs in mainstream school or failing out of school; cannot obtain or hold independent job or unable to complete virtually any homemaking tasks independently; or adequate to good functioning with major support. Requires assisted work environment, entirely special education classes, non-public or psychiatric school, home schooling for the purpose of a supportive school environment, and/or supported home environment but functions adequately given these supports (may fall behind but completes assigned tasks, obtains satisfactory performance evaluations at work or passing grades).
Marginal ability to function

3 Impaired functioning with major support. Requires supported work environment, entirely special education classes, non-public or psychiatric school, home schooling for the purpose of a supportive school environment, and/or supported home environment but functions poorly despite these supports (persistently behind on tasks, frequently unable to perform, obtains poor performance evaluations at work or fails courses at school).

Inability to function

2 Disabled but participates in structured activities. On disability or equivalent non-independent status. Not working for pay, attending classes for grades, or living independently. Spends 5 or more hours a week in structured role-related activities (e.g., residential treatment, volunteering, tutoring, sheltered work programs).

Extreme role dysfunction

1 Severely disabled. On disability or equivalent non-independent status. Not working for pay, attending classes for grades, or living independently. Spends fewer than 5 hours a week in structured role-related activities.
GLOBAL FUNCTIONING: SOCIAL SCALE

Superior social/interpersonal functioning

10 Superior functioning in a wide range of social and interpersonal activities. Frequently seeks out others and has multiple satisfying interpersonal relationships, including multiple close and casual friends. Is sought out by others because of his or her many positive qualities. Age-appropriate involvement in intimate relationships.

Above average social/interpersonal functioning

9 Good functioning in all social areas, and interpersonally effective. Interested and involved in a wide range of social and interpersonal activities, including both close and casual friends. Age-appropriate involvement in intimate relationships. No more than everyday interpersonal problems or concerns (e.g., an occasional argument with spouse, girlfriend/boyfriend, friends, co-workers, or classmates). Able to resolve such conflicts appropriately.

Good social/interpersonal functioning

8 Some transient mild impairment in social functioning. Mild social impairment is present, but transient and expectable reactions to psychosocial stressors (e.g., after minor arguments with spouse, girlfriend/boyfriend, friends, coworkers, or classmates). Has some meaningful interpersonal relationships with peers (casual and close friends), and/or age-appropriate intimate relationships. Infrequent interpersonal conflict with peers.

Mild problems in social/interpersonal functioning

7 Some persistent mild difficulty in social functioning. Mild impairment present that is NOT just expectable reaction to psychosocial stressors (e.g., mild conflicts with peers, coworkers or classmates; difficulty resolving conflicts appropriately). Has some meaningful interpersonal relationships with peers (casual and/or close friends). Some difficulty developing or maintaining age-appropriate intimate relationships (e.g., multiple short-term relationships).

Moderate impairment in social/interpersonal functioning

6 Moderate impairment in social functioning. Present (e.g., few close friends; significant but intermittent conflicts with peers, coworkers, or classmates). Moderate difficulty developing age-appropriate intimate relationships (e.g., infrequent dating). Occasionally seeks out others but will respond if invited by others to participate in an activity.

Serious impairment in social/interpersonal functioning

5 Serious impairment in social functioning. No close friends or intimate partner but has some casual social contacts (e.g., acquaintances, school/work friends only). Rarely seeks out others. Occasional combative or verbally argumentative behavior with peers. Beginning to withdraw from family members (e.g., does not initiate conversation with family, but will respond if addressed).

Major impairment in social and interpersonal functioning

4 Major impairment in social functioning. Serious impairment in relationships with friends or peers (e.g., very few or no friends, frequent conflicts with friends, or frequently avoids friends). Frequent combative or verbally argumentative behavior with peers. Infrequent contact with family members (e.g., sometimes does not respond to family or avoids family members).

Marginal ability to function socially

3 Marginal ability to function socially or maintain interpersonal relationships. Frequently alone and socially isolated. Serious impairment in relationships with all peers, including acquaintances. Few interactions with family members (e.g., often alone in room). Serious impairment in communication with others (e.g., avoids participating in most social activities).
Inability to function socially

2 Unable to function socially or to maintain any interpersonal relationships. Typically alone and socially isolated. Rarely leaves home. Rarely answers the phone or the door. Rarely participates in interactions with others at home or in other settings (e.g., work, school).

Extreme social isolation

1 Extreme social isolation. No social or family member contact at all. Does not leave home. Refuses to answer the phone or door.
Appendix F: Multidimensional Adolescent Functioning Scale (MAFS)
MAFS Questionnaire

Below are a number of statements about people’s lives. For each statement, please rate in the first column how much it describes your situation. In the second column rate how important this statement is to you (even if it is currently not applicable).

<table>
<thead>
<tr>
<th>Describes my situation?</th>
<th>Important to me?</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. My parents’ rules are reasonable.</td>
<td>1. Not at all or rarely</td>
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<tr>
<td></td>
<td>2. Sometimes</td>
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<td></td>
<td>3. Often</td>
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<td></td>
<td>4. Always or almost always</td>
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<tr>
<td></td>
<td>0. Not applicable</td>
</tr>
</tbody>
</table>

| 2. I feel like I’m working towards a goal (related to work or study). | 1. Not at all or rarely | 1. Very important |
|                                                                      | 2. Sometimes           | 2. Quite important |
|                                                                      | 3. Often               | 3. Neutral        |
|                                                                      | 4. Always or almost always | 4. Only slightly important |
|                                                                      | 0. Not applicable      | 5. Not at all important |

<p>| 3. My friends are encouraging. | 1. Not at all or rarely | 1. Very important |
|                               | 2. Sometimes           | 2. Quite important |
|                               | 3. Often               | 3. Neutral        |
|                               | 4. Always or almost always | 4. Only slightly important |
|                               | 0. Not applicable      | 5. Not at all important |</p>
<table>
<thead>
<tr>
<th></th>
<th>I get on well with my parents.</th>
<th></th>
<th>I am pleased with how my life is going.</th>
<th></th>
<th>I look after my health.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>□ 1 Not at all or rarely</td>
<td></td>
<td>□ 1 Not at all or rarely</td>
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<td>□ 1 Not at all or rarely</td>
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<td>□ 5 Not at all important</td>
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</tbody>
</table>
7. I have plenty to do most of the time. | □ 1  Not at all or rarely | □ 1  Very important |
| □ 2  Sometimes | □ 2  Quite important |
| □ 3  Often | □ 3  Neutral |
| □ 4  Always or almost always | □ 4  Only slightly important |
| □ 0  Not applicable | □ 5  Not at all important |

8. I often feel bored. | □ 1  Not at all or rarely | □ 1  Very important |
| □ 2  Sometimes | □ 2  Quite important |
| □ 3  Often | □ 3  Neutral |
| □ 4  Always or almost always | □ 4  Only slightly important |
| □ 0  Not applicable | □ 5  Not at all important |

9. My parents disapprove of my friends, lifestyle or appearance. | □ 1  Not at all or rarely | □ 1  Very important |
| □ 2  Sometimes | □ 2  Quite important |
| □ 3  Often | □ 3  Neutral |
| □ 4  Always or almost always | □ 4  Only slightly important |
| □ 0  Not applicable | □ 5  Not at all important |

10. I feel close to my friends. | □ 1  Not at all or rarely | □ 1  Very important |
<p>| □ 2  Sometimes | □ 2  Quite important |
| □ 3  Often | □ 3  Neutral |
| □ 4  Always or almost always | □ 4  Only slightly important |
| □ 0  Not applicable | □ 5  Not at all important |</p>
<table>
<thead>
<tr>
<th></th>
<th>Question</th>
<th>Rating Options</th>
<th>Importance Options</th>
</tr>
</thead>
<tbody>
<tr>
<td>11</td>
<td>My living arrangements are stable.</td>
<td>□ 1 Not at all or rarely</td>
<td>□ 1 Very important</td>
</tr>
<tr>
<td></td>
<td></td>
<td>□ 2 Sometimes</td>
<td>□ 2 Quite important</td>
</tr>
<tr>
<td></td>
<td></td>
<td>□ 3 Often</td>
<td>□ 3 Neutral</td>
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<td></td>
<td></td>
<td>□ 4 Always or almost always</td>
<td>□ 4 Only slightly important</td>
</tr>
<tr>
<td></td>
<td></td>
<td>□ 0 Not applicable</td>
<td>□ 5 Not at all important</td>
</tr>
<tr>
<td>12</td>
<td>I spend quite a lot of time with my friends.</td>
<td>□ 1 Not at all or rarely</td>
<td>□ 1 Very important</td>
</tr>
<tr>
<td></td>
<td></td>
<td>□ 2 Sometimes</td>
<td>□ 2 Quite important</td>
</tr>
<tr>
<td></td>
<td></td>
<td>□ 3 Often</td>
<td>□ 3 Neutral</td>
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<tr>
<td></td>
<td></td>
<td>□ 4 Always or almost always</td>
<td>□ 4 Only slightly important</td>
</tr>
<tr>
<td></td>
<td></td>
<td>□ 0 Not applicable</td>
<td>□ 5 Not at all important</td>
</tr>
<tr>
<td>13</td>
<td>Members of my family are disappointed in me.</td>
<td>□ 1 Not at all or rarely</td>
<td>□ 1 Very important</td>
</tr>
<tr>
<td></td>
<td></td>
<td>□ 2 Sometimes</td>
<td>□ 2 Quite important</td>
</tr>
<tr>
<td></td>
<td></td>
<td>□ 3 Often</td>
<td>□ 3 Neutral</td>
</tr>
<tr>
<td></td>
<td></td>
<td>□ 4 Always or almost always</td>
<td>□ 4 Only slightly important</td>
</tr>
<tr>
<td></td>
<td></td>
<td>□ 0 Not applicable</td>
<td>□ 5 Not at all important</td>
</tr>
<tr>
<td>14</td>
<td>I am pleased with what I’ve achieved in my life so far.</td>
<td>□ 1 Not at all or rarely</td>
<td>□ 1 Very important</td>
</tr>
<tr>
<td></td>
<td></td>
<td>□ 2 Sometimes</td>
<td>□ 2 Quite important</td>
</tr>
<tr>
<td></td>
<td></td>
<td>□ 3 Often</td>
<td>□ 3 Neutral</td>
</tr>
<tr>
<td></td>
<td></td>
<td>□ 4 Always or almost always</td>
<td>□ 4 Only slightly important</td>
</tr>
<tr>
<td></td>
<td></td>
<td>□ 0 Not applicable</td>
<td>□ 5 Not at all important</td>
</tr>
<tr>
<td>15</td>
<td>My friends are often disappointed in me.</td>
<td>□ 1 Not at all or rarely</td>
<td>□ 1 Very important</td>
</tr>
<tr>
<td></td>
<td></td>
<td>□ 2 Sometimes</td>
<td>□ 2 Quite important</td>
</tr>
<tr>
<td></td>
<td></td>
<td>□ 3 Often</td>
<td>□ 3 Neutral</td>
</tr>
<tr>
<td></td>
<td></td>
<td>□ 4 Always or almost always</td>
<td>□ 4 Only slightly important</td>
</tr>
<tr>
<td></td>
<td></td>
<td>□ 0 Not applicable</td>
<td>□ 5 Not at all important</td>
</tr>
<tr>
<td>Question</td>
<td>Rating Options</td>
<td>Importance Options</td>
<td></td>
</tr>
<tr>
<td>-------------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------------------</td>
<td>---------------------</td>
<td></td>
</tr>
<tr>
<td>16. My friends are supportive of me when I need it.</td>
<td>□ 1 Not at all or rarely □ 2 Sometimes □ 3 Often □ 4 Always or almost always □ 0 Not applicable</td>
<td>□ 1 Very important</td>
<td></td>
</tr>
<tr>
<td></td>
<td>□ 2 Quite important □ 3 Neutral □ 4 Only slightly important □ 5 Not at all important</td>
<td></td>
<td></td>
</tr>
<tr>
<td>17. My family are supportive of me when I need it.</td>
<td>□ 1 Not at all or rarely □ 2 Sometimes □ 3 Often □ 4 Always or almost always □ 0 Not applicable</td>
<td>□ 1 Very important</td>
<td></td>
</tr>
<tr>
<td></td>
<td>□ 2 Quite important □ 3 Neutral □ 4 Only slightly important □ 5 Not at all important</td>
<td></td>
<td></td>
</tr>
<tr>
<td>18. My friends disapprove of me in some way (e.g., lifestyle, appearance, etc.).</td>
<td>□ 1 Not at all or rarely □ 2 Sometimes □ 3 Often □ 4 Always or almost always □ 0 Not applicable</td>
<td>□ 1 Very important</td>
<td></td>
</tr>
<tr>
<td></td>
<td>□ 2 Quite important □ 3 Neutral □ 4 Only slightly important □ 5 Not at all important</td>
<td></td>
<td></td>
</tr>
<tr>
<td>19. I am in good physical health.</td>
<td>□ 1 Not at all or rarely □ 2 Sometimes □ 3 Often □ 4 Always or almost always □ 0 Not applicable</td>
<td>□ 1 Very important</td>
<td></td>
</tr>
<tr>
<td></td>
<td>□ 2 Quite important □ 3 Neutral □ 4 Only slightly important □ 5 Not at all important</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
20. My parents are encouraging.

<table>
<thead>
<tr>
<th>Frequency</th>
<th>Importance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not at all or rarely</td>
<td>Not at all or rarely</td>
</tr>
<tr>
<td>Sometimes</td>
<td>Quite important</td>
</tr>
<tr>
<td>Often</td>
<td>Neutral</td>
</tr>
<tr>
<td>Always or almost always</td>
<td>Only slightly important</td>
</tr>
<tr>
<td>Not applicable</td>
<td>Not at all important</td>
</tr>
</tbody>
</table>

21. I attend school/TAFE/university or work regularly.

<table>
<thead>
<tr>
<th>Frequency</th>
<th>Importance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not at all or rarely</td>
<td>Not at all or rarely</td>
</tr>
<tr>
<td>Sometimes</td>
<td>Quite important</td>
</tr>
<tr>
<td>Often</td>
<td>Neutral</td>
</tr>
<tr>
<td>Always or almost always</td>
<td>Only slightly important</td>
</tr>
<tr>
<td>Not applicable</td>
<td>Not at all important</td>
</tr>
</tbody>
</table>

22. I get along well with my teachers or supervisor/boss.

<table>
<thead>
<tr>
<th>Frequency</th>
<th>Importance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not at all or rarely</td>
<td>Not at all or rarely</td>
</tr>
<tr>
<td>Sometimes</td>
<td>Quite important</td>
</tr>
<tr>
<td>Often</td>
<td>Neutral</td>
</tr>
<tr>
<td>Always or almost always</td>
<td>Only slightly important</td>
</tr>
<tr>
<td>Not applicable</td>
<td>Not at all important</td>
</tr>
</tbody>
</table>

23. I think that going to school/TAFE/university or work is important for my future.

<table>
<thead>
<tr>
<th>Frequency</th>
<th>Importance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not at all or rarely</td>
<td>Not at all or rarely</td>
</tr>
<tr>
<td>Sometimes</td>
<td>Quite important</td>
</tr>
<tr>
<td>Often</td>
<td>Neutral</td>
</tr>
<tr>
<td>Always or almost always</td>
<td>Only slightly important</td>
</tr>
<tr>
<td>Not applicable</td>
<td>Not at all important</td>
</tr>
</tbody>
</table>
Appendix G: Plain Language Statement and Consent Form (patient version)
TO: Participants

Plain Language Statement

Date: 30th June, 2008

Full Project Title: Social cognition in early psychosis and its relationship with neurocognition, symptomatology and social functioning

Principal Researcher: Dr. Linda Byrne

Student Researcher: Alicia Papas

This Plain Language Statement and Consent Form is 7 pages long. Please make sure you have all the pages.

1. Your Consent

You are invited to take part in this research project. This Plain Language Statement contains detailed information about the research project. Its purpose is to explain to you as openly and clearly as possible all the procedures involved in this project so that you can make a fully informed decision whether you are going to participate.

Please read this Plain Language Statement carefully. Feel free to ask questions about any information in the document. You may also wish to discuss the project with a relative or friend or your local health worker. Feel free to do this.

Once you understand what the project is about and if you agree to take part in it, you will be asked to sign the Consent Form. By signing the Consent Form, you indicate that you understand the information and that you give your consent to participate in the research project.

You will be given a copy of the Plain Language Statement and Consent Form to keep as a record.

2. Purpose and Background

The main purpose of this project is to examine how well people who have experienced symptoms of psychosis process social and emotion information, focus their attention, remember things, and learn things. I am a student researcher undertaking my Doctorate in Clinical Psychology and as part of this degree I will be investigating whether people who are receiving treatment for psychosis perform more poorly on such tasks. Previous research has shown that people who have experienced psychosis find it harder to do these things than people who have not had problems with their mental health.

A total of 100 people will participate in this project; 50 people currently being treated for psychosis, and 50 people without a history of mental illness who will serve as a control group.

You are invited to participate in this research project because you have are being treated for psychosis. The results of this research may be used to help student researcher, Alicia Papas, obtain a Doctor of Clinical Psychology degree.
3. **Funding**

This research is made possible by funding provided by Deakin University, and the services and functions of ORYGEN Youth Health and ORYGEN Research Centre.

4. **Procedures**

Participation in this project will involve completion of a number of card-sorting, paper and pencil, and touch-computer tasks that assess things like learning, memory, and information processing ability. The card-sorting tasks take approximately 20 minutes, paper and pencil tests about 60 minutes, and the computer assessment is 40 minutes in duration. You will also be asked to complete a couple of short questionnaires about your work/school and social life, which will take no more than 10 minutes. The researcher will also ask you some general demographic questions, questions about your daily activities, and any symptoms you may be experiencing. This interview part will take about 45 minutes. The whole assessment is about 3 hours long. If necessary, the assessment can be scheduled over 2 sessions within a 2 week period. As the assessment is quite lengthy, breaks for refreshments will be given regularly (after the first hour and the second hour). You are free to take as many breaks as you need, please let the researcher know if you require a break at any time.

5. **Possible Benefits**

There will be no direct benefit to you, however your participation will improve our understanding about the cognitive ability (things like processing information, learning, memory, and recognising emotions) of people who have experienced psychosis, and whether certain symptoms make these tasks harder for people. The information derived from the research may help to develop better interventions and therapies in the future.

6. **Possible Risks**

There are no anticipated risks, side effects and discomforts as you are simply required to take part in an interview and complete some tasks. However, the assessment time (approximately 3 hours) is quite long so you may feel a little tired afterward.

Sometimes people find that talking about their illness is upsetting. If at any time during the assessment you feel distressed, please let the researcher know immediately, or contact your case manager at ORYGEN Youth Health on 9342 2800 to discuss how you feel.

If you feel you no longer want to participate in the assessment once you start, for example, if you are too tired or can no longer concentrate, you are not obliged to keep going. You can either ask the researcher to make a second appointment to finish off the assessment, or end your participation altogether.

7. **Privacy, Confidentiality and Disclosure of Information**

Any information obtained in connection with this research project that can identify you will remain confidential and will only be used for the purpose of this research project. It will only be disclosed with your permission, except as required by law. By signing the attached Consent Form, you authorise release of, or access to, this confidential information to the relevant study personnel. You also authorise the Ethics Committee to have direct access to your information purely for the purpose of verification of the study procedures and/or data to ensure the study is being carried out correctly.

The information collected during the study will be stored in research and computer files but your name would not be; instead your information would be coded by a number. The information will be stored at ORYGEN Research Centre until the research project is finished, then transported to Deakin University where it will be locked in a filing cabinet for a minimum of 6 years. Only the research staff will know that the information is related to you. The results of the study may be published in medical...
literature, but your identity will not be revealed. All records identifying you will be kept confidential and, to the extent permitted by applicable laws and/or regulations, will not be made publicly available.

8. Results of Project

Feedback of test results is not a routine part of this project, however please contact the student researcher, Alicia Papas, via email (apap@deakin.edu.au) or phone 9342 2993 if you would like to access your test results or information. Alternatively you may call the principal researcher on 9244 6424. A meeting can be organised to discuss the results.

The results of this research project will be published in a thesis and submitted for grading. Publications in medical journals are also anticipated.

9. Participation is Voluntary

Participation in any research project is voluntary. If you do not wish to take part you are not obliged to. If you decide to take part and later change your mind, you are free to withdraw from the project at any stage, that is, up until the data is processed and your identifying details are removed. Any information obtained from you up until the time you withdraw will not be used and will be destroyed.

Your decision whether to take part or not to take part, or to take part and then withdraw, will not affect your relationship or level of care from ORYGEN Youth Health.

Before you make your decision, a member of the research team will be available to answer any questions you have about the research project. You can ask for any information you want. Sign the Consent Form only after you have had a chance to ask your questions and have received satisfactory answers.

If you decide to withdraw from this project, please notify a member of the research team or complete and return the Revocation of Consent Form attached.

10. Ethical Guidelines

This project will be carried out according to the National Statement on Ethical Conduct in Human Research (2007) produced by the National Health and Medical Research Council of Australia. This statement has been developed to protect the interests of people who agree to participate in human research studies.

The ethics aspects of this research project have been approved by the Human Research Ethics Committee of Deakin University.

Approval has also been provided by the Melbourne Health Research Ethics Committee to carry out the research at ORYGEN Youth Health.

11. Complaints

If you have any complaints about any aspect of the project, the way it is being conducted or any questions about your rights as a research participant, then you may contact:

The Executive Officer, Human Research Ethics Committee, Deakin University, 221 Burwood Highway, Burwood Victoria 3125, Telephone: 9251 7123, Facsimile: 9244 6581; research-ethics@deakin.edu.au.

Please quote project number: EC65 - 2008.
12. **Reimbursement for your costs**  

It is acknowledged that the assessment is quite lengthy therefore you will be given $20 in appreciation of your time once you complete the assessment. This reimbursement covers any travel expenses you incur to attend the assessment (e.g. petrol or public transport costs).

13. **Further Information, Queries or Any Problems**  

If you require further information, wish to withdraw your participation or if you have any problems concerning this project, you can contact the principal researcher Dr. Linda Byrne at Deakin University on 9244 6424, or if you are in distress after hours you can call the ORYGEN YAT duty worker via the paging service (03) 9483 4556 or a toll-free number 1800 888 320.

The researchers responsible for this project are:

* **Alicia Papas (student researcher), Deakin University, Faculty of Health, Medicine, Nursing and Behavioural Sciences, School of Psychology, 221 Burwood Hwy, Burwood, 3125.**  
  Phone: 9342 2993 (at ORYGEN Research Centre).

* **Dr Linda Byrne (principal researcher), Deakin University, Faculty of Health, Medicine, Nursing and Behavioural Sciences, School of Psychology, 221 Burwood Hwy, Burwood, 3125.**  
  Phone: 9244 6424.

* **After hours contact: Dr. Linda Byrne 9244 6424 – please leave a message if this number is unattended as it will be monitored after hours during the course of this project. If the matter is urgent and your mental health is concerned, please call YAT on 1800 888 320.**
TO: Participants

Consent Form

Date: 30th June, 2008

Full Project Title: Social cognition in early psychosis and its relationship with neurocognition, symptomatology and social functioning

I have read, or have had read to me, and I understand the attached Plain Language Statement.

I freely agree to participate in this project according to the conditions in the Plain Language Statement.

I have been given a copy of the Plain Language Statement and Consent Form to keep.

The researcher has agreed not to reveal my identity and personal details, including where information about this project is published, or presented in any public form.

Participant's Name (printed) .................................................................
Signature .............................................................. Date .........................
TO: Parents or Carers

Third Party Consent Form

(To be used by parents/guardians of minor children, or carers/guardians consenting on behalf of adult participants who do not have the capacity to give informed consent)

Date: 30th June, 2008

Full Project Title: Social cognition in early psychosis and its relationship with neurocognition, symptomatology and social functioning

I have read, or have had read to me, and I understand the attached Plain Language Statement.

I give my permission for ................................................................. (name of participant) to participate in this project according to the conditions in the Plain Language Statement.

I have been given a copy of Plain Language Statement and Consent Form to keep.

The researcher has agreed not to reveal my identity and personal details, including where information about this project is published, or presented in any public form.

Participant’s Name (printed) .............................................................

Name of Person giving Consent (printed) ............................................

Relationship to Participant: .............................................................

Signature ................................................................. Date ..........................
TO: Participants

Revocation of Consent Form

(To be used for participants who wish to withdraw from the project)

Date: 30th June, 2008

Full Project Title: Social cognition in early psychosis and its relationship with neurocognition, symptomatology and social functioning

I hereby wish to WITHDRAW my consent to participate in the above research project and understand that such withdrawal WILL NOT jeopardise my relationship with Deakin University or ORYGEN Youth Health.

Participant's Name (printed) .................................................................

Signature .................................................................................................. Date ................................

Please mail or fax this form to:

Alicia Papas
DPsych Clinical student
School of Psychology
221 Burwood Hwy
Burwood Vic 3125
Ph: 9342 2993 (at ORYGEN)
Fax: 9244 6581 (at Deakin University)
Appendix H: Plain Language Statement and Consent Form (controls version)
DEAKIN UNIVERSITY
PLAIN LANGUAGE STATEMENT AND CONSENT FORM

TO: Participants

Plain Language Statement

Date: 30th June, 2008
Full Project Title: Social cognition in early psychosis and its relationship with neurocognition, symptomatology and social functioning

Principal Researcher: Dr. Linda Byrne
Student Researcher: Alicia Papas

This Plain Language Statement and Consent Form is 7 pages long. Please make sure you have all the pages.

14. Your Consent
You are invited to take part in this research project.

This Plain Language Statement contains detailed information about the research project. Its purpose is to explain to you as openly and clearly as possible all the procedures involved in this project so that you can make a fully informed decision whether you are going to participate.

Please read this Plain Language Statement carefully. Feel free to ask questions about any information in the document. You may also wish to discuss the project with a relative or friend. Feel free to do this.

Once you understand what the project is about and if you agree to take part in it, you will be asked to sign the Consent Form. By signing the Consent Form, you indicate that you understand the information and that you give your consent to participate in the research project.

You will be given a copy of the Plain Language Statement and Consent Form to keep as a record.

15. Purpose and Background
The main purpose of this project is to examine how well people who have experienced a psychotic episode process social and emotion information, focus their attention, remember things and learn things. I am a student researcher undertaking my Doctorate in Clinical Psychology and as part of this degree I will be investigating whether people who are receiving treatment for psychosis perform more poorly on such tasks. Previous research has shown that people who have had a psychotic episode or schizophrenia find it harder to do these things than people who have not experienced psychosis. In order to assess this, people from the community who do not have a psychotic illness are also required to complete some testing as a comparison, or “control” group.

A total of 50 patients attending a mental health service, and 50 controls from the community will participate in this project.
You are invited to participate in this research project because you have not experienced an episode of psychosis and would form part of a control group. The results of this research may be used to help student researcher, Alicia Papas, obtain a Doctor of Clinical Psychology degree.

16. Funding
This research is made possible by funding provided by Deakin University, and the services and functions of ORYGEN Youth Health and ORYGEN Research Centre.

17. Procedures
This project will involve a short interview-based assessment (approximately 15 minutes) during which you will be screened for any mental health problems to ensure you are eligible to take part in the research as a control participant. You will also be asked some demographic questions (e.g. your age, level of education etc) and some questions about your daily life and activities. Completion of a number card-sorting, paper and pencil, and touch-computer tasks is also required. These will assess things like learning, memory, and information processing ability. The card-sorting tasks take approximately 20 minutes, paper and pencil tests about 60 minutes, and the computer assessment is 40 minutes in duration. You will also be asked to complete a couple of short questionnaires about your work/school and social life, which will take no more than 10 minutes. The entire assessment will take about 2.5 hours and will be conducted at ORYGEN Youth Health. As the assessment is quite lengthy, breaks for refreshments will be given. Please feel free to tell the researcher if you need additional breaks at any stage.

18. Possible Benefits
There will be no direct benefit to you, however your participation will help us to learn more about the cognitive ability (things like processing information, learning, memory, and recognising emotions) of people who have experienced psychosis, and whether certain symptoms make these tasks harder for people. The information derived from the research may help to develop better interventions and therapies for people with psychotic disorders in the future.

19. Possible Risks
There are no anticipated risks, side effects and discomforts as you are simply required to take part in an interview and complete some tasks. If you feel you no longer want to participate in the assessment once you start, you are not obliged to keep going. Just let the researcher know and you will be free to leave.

20. Privacy, Confidentiality and Disclosure of Information
Any information obtained in connection with this research project that can identify you will remain confidential and will only be used for the purpose of this research project. It will only be disclosed with your permission, except as required by law. By signing the attached Consent Form, you authorise release of, or access to, this confidential information to the relevant study personnel. You also authorise the Ethics Committee to have direct access to your information purely for the purpose of verification of the study procedures and/or data to ensure the study is being carried out correctly.

The information collected during the study will be stored in research and computer files but your name would not be; instead your information would be coded by a number. The information will be stored at ORYGEN Research Centre until the research project is finished, then transported to Deakin University where it will be locked in a filing cabinet for a minimum of 6 years. Only the research staff will know that the information is related to you. The results of the study may be published in medical literature, but your identity will not be revealed. All records identifying you will be kept confidential and, to the extent permitted by applicable laws and/or regulations, will not be made publicly available.
21. Results of Project

Feedback of test results is not a routine part of this project, however please contact the student researcher, Alicia Papas, via email (apap@deakin.edu.au) or phone 9342 2993 if you would like to access your test results or information. Alternatively you may call the principal researcher on 9244 6424. A meeting can be organised to discuss the results.

The results of this research project will be published in a thesis and submitted for grading. Publications in medical journals are also anticipated.

22. Participation is Voluntary

Participation in any research project is voluntary. If you do not wish to take part you are not obliged to. If you decide to take part and later change your mind, you are free to withdraw from the project at any stage, that is, up until the data is processed and your identifying details are removed. Any information obtained from you up until the time you withdraw will not be used and will be destroyed.

Your decision whether to take part or not to take part, or to take part and then withdraw, will not affect your relationship or level of care from ORYGEN Youth Health.

Before you make your decision, a member of the research team will be available to answer any questions you have about the research project. You can ask for any information you want. Sign the Consent Form only after you have had a chance to ask your questions and have received satisfactory answers.

If you decide to withdraw from this project, please notify a member of the research team or complete and return the Revocation of Consent Form attached. This notice will allow the research team to inform you if there are any health risks or special requirements linked to withdrawing.

23. Ethical Guidelines

This project will be carried out according to the National Statement on Ethical Conduct in Human Research (2007) produced by the National Health and Medical Research Council of Australia. This statement has been developed to protect the interests of people who agree to participate in human research studies.

The ethics aspects of this research project have been approved by the Human Research Ethics Committee of Deakin University.

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24. Complaints

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The Executive Officer, Human Research Ethics Committee, Deakin University, 221 Burwood Highway, Burwood Victoria 3125, Telephone: 9251 7123, Facsimile: 9244 6581; research-ethics@deakin.edu.au.

Please quote project number: EC65 - 2008.
25. Reimbursement for your costs

It is acknowledged that the assessment is quite lengthy, therefore you will be given $20 in appreciation of your time once you complete the assessment. This reimbursement covers any travel expenses you incur to attend the assessment (e.g. petrol or public transport costs).

26. Further Information, Queries or Any Problems

If you require further information, wish to withdraw your participation or if you have any problems concerning this project, you can contact the principal researcher Dr. Linda Byrne at Deakin University on 9244 6424.

The researchers responsible for this project are:

*Alicia Papas (student researcher), Deakin University, Faculty of Health, Medicine, Nursing and Behavioural Sciences, School of Psychology, 221 Burwood Hwy, Burwood, 3125. Phone: 9342 2993.*

*Dr Linda Byrne (principal researcher), Deakin University, Faculty of Health, Medicine, Nursing and Behavioural Sciences, School of Psychology, 221 Burwood Hwy, Burwood, 3125. Phone: 9244 6424.*

*After hours contact: Dr. Linda Byrne on 9244 6424 – please leave a message if this number is unattended as it will be monitored after hours during the course of this project.*
TO: Participants

Consent Form

Date: 30th June, 2008

Full Project Title: Social cognition in early psychosis and its relationship with neurocognition, symptomatology and social functioning

I have read, or have had read to me, and I understand the attached Plain Language Statement.

I freely agree to participate in this project according to the conditions in the Plain Language Statement.

I have been given a copy of the Plain Language Statement and Consent Form to keep.

The researcher has agreed not to reveal my identity and personal details, including where information about this project is published, or presented in any public form.

Participant's Name (printed) ...........................................................................................................

Signature .................................................................................................................. Date .........................
TO: Parents or Carers

Third Party Consent Form

(To be used by parents/guardians of minor children, or carers/guardians consenting on behalf of adult participants who do not have the capacity to give informed consent)

Date: 30th June, 2008

Full Project Title: Social cognition in early psychosis and its relationship with neurocognition, symptomatology and social functioning

I have read, or have had read to me, and I understand the attached Plain Language Statement.

I give my permission for ...........................................................(name of participant)
to participate in this project according to the conditions in the Plain Language Statement.

I have been given a copy of Plain Language Statement and Consent Form to keep.

The researcher has agreed not to reveal my identity and personal details, including where information about this project is published, or presented in any public form.

Participant's Name (printed) .................................................................

Name of Person giving Consent (printed) ..................................................

Relationship to Participant: .................................................................

Signature ................................................................. Date ..................
DEAKIN UNIVERSITY
PLAIN LANGUAGE STATEMENT AND CONSENT FORM

TO: Participants

Revocation of Consent Form

(To be used for participants who wish to withdraw from the project)

Date: 30th June, 2008

Full Project Title: Social cognition in early psychosis and its relationship with neurocognition, symptomatology and social functioning

I hereby wish to WITHDRAW my consent to participate in the above research project and understand that such withdrawal WILL NOT jeopardise my relationship with Deakin University.

Participant’s Name (printed) ……………………………………………………………

Signature ……………………………………………………………………… Date …………………

Please mail or fax this form to:

Alicia Papas
DPsych Clinical student
School of Psychology
221 Burwood Hwy
Burwood Vic 3125
Ph: 9342 2993
Fax: 9244 6581
Appendix I: Schema Component Sequencing Task – Revised (SCST-R):

Original and Modified Versions
Social Component Sequencing Task – Revised (SCST-R):

List of scenarios and component actions

Below is a list of the social scenarios that comprise the SCST-R (Corrigan & Addis, 1995), with component actions in their correct sequence.

Scenarios marked with (*) denote those that include modified actions from the original version of the SCST-R. In such instances, the updated action is listed directly to the right of the original action. If the order of actions was affected due to any modified actions, the updated sequence of actions is stipulated.

Scenarios marked with (^) were replaced altogether with other age and/or culturally appropriate situations. The updated scenario is listed alongside the original scenario (on the right hand side of the page).

Example scenario:

Eating at a Restaurant

Give reservation name
Be seated
Order drinks
Look at menu
Order meal
Eat food
Pay bill
Leave tip
The 12 SCST-R Scenarios

**Get Groceries***

Go to store  
Go to supermarket  
Get cart  
Get trolley  
Get items from shelves  
Stand in line at cashier  
Groceries rung up  
Groceries scanned  
Pay for groceries

**Go to Movie***

Pick move out of newspapers  
Find theatre  
Find cinema  
Wait in line  
Buy ticket  
Give tickets to usher  
Find seat  
Watch previews  
Watch main feature  
Leave theatre  
Leave cinema
**Prepare a Meal***

*Note: The action of “greet friends” was replaced by “eat food”, which changed the order of the actions as indicated.*

- Plan menu
- Buy food
- Cook food
- Greet friends
- Serve food
- Eat food
- Clear dishes off table

**Take a Bath***

- Close bathroom door
- Enter bathroom
- Turn on water
- Check water temperature
- Get in shower
- Soap body
- Rinse body
- Dry body
- Spray deodorant
- Put on clean clothes
Find a Job*

Search want ads
Identify interesting jobs
Call for job interview
Fill out job application
Go to job site for interview
Get job

Buy Gifts*

Make list
Browse catalogue
Drive to store
Ask for item location
Find gifts in store
Pay for items
Wrap in pretty paper
Put on bow
Give gifts

Buy Christmas Gifts

Search job ads
Apply for jobs
Get interview
Go to store
Gift wrap items
Put Christmas name tags on gifts
Play Football*
Go to park
Pick teams
Kick off football
Play first half
Play second half
Buy winners a beer
Buy winners a drink

Giving a Speech*
Asked to give speech
Find out topic
Outline speech
Practice presentation
Go to place for speech
Wait to be introduced
Give speech
Answer questions
Enjoy applause

Giving a Presentation
Asked to give presentation
Prepare presentation
Go to place for presentation
Give presentation
**Going Camping**

Ask friends to go
Plan trip together
Drive to site
Set up camp
Cook dinner
Sing around the campfire
Light campfire

**Getting a Raise**

Get up courage
Go to work
Ask boss for time to talk
Go to boss’ office
Sit in front of boss’ desk
Ask for a raise
Listen to boss’ response
Thank boss
Go celebrate

**Assignment Extension**

Go to teacher’s office
Be greeted by teacher
Sit down in front of teacher
Ask teacher for an extension
Give reasons for extension
Listen to teacher’s response
Discuss new due date
Thank teacher
Leave teacher’s office
**Getting Eye Glasses**

Go to eye doctor

Read eye chart

Get glasses prescription

Wait for glasses to be made

Pay for glasses

Notice good vision

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**Going to Church**

Get dressed up

Drive to church

Greet neighbours

Sit in pew

Listen to sermon

Pass the plate

Thank pastor

Drive home

Hang up clothes

---

**Washing Clothes**

Put clothes in washing machine

Put in washing powder

Select appropriate wash setting

Press start

Wait for wash cycle to finish

Take clothes out of washing machine

Hang up clothes to dry

Bring in clothes from washing line

Put clothes away