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Social Cognitive Performance and the Psychosis-Spectrum Prodrome

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Abstract

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By Sandra M. Goulding

Social cognitive (SC) impairment is pervasive in chronic and first-episode psychosis (FEP) samples, and an emerging body of research suggests individuals at clinical high risk (CHR) for psychosis exhibit deficits intermediate to those found in healthy control (HC) and FEP groups. However, given the limited body of literature to date, it is unclear how SC deficits are linked with illness progression. This current investigation examines multiple SC domains (i.e., emotional processing/recognition, theory of mind, and relationship/social perception) in a sample large enough to compare SC performance between HC and CHR groups, examine the influence of sex on SC performance in the CHR group, and determine the relatedness of SC performance to clinical symptoms and functional status in CHR subjects at both baseline and 12-month follow-up. Results indicated SC deficits in the CHR group relative to the HC group. The influence of sex on SC performance was mixed depending on the SC domain measured. Similarly, at baseline, support for the relatedness of SC deficits and clinical symptoms/functioning varied as a function of the measure. Finally, there was no support for the predictive utility of baseline SC deficits for clinical symptoms and functioning at 12-month follow-up. Overall, this study contributes to efforts purposed to gain a more thorough understanding of the role of SC impairment in the psychosis-spectrum of disorders. Although prior investigations suggest the predictive utility of SC for detecting those at CHR for psychosis conversion, results from this study indicate that SC is likely best understood as a clinical outcome domain that prompts help-seeking behaviors in individuals who present at CHR clinics.

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Individuals with schizophrenia spectrum disorders (SSDs), including schizophrenia and other psychotic disorders, manifest a constellation of symptomatology (American Psychological Association, 2000) that includes positive psychotic symptoms (e.g., delusions and hallucinations) and may also include negative (e.g., avolition and social withdrawal) and/or disorganized (e.g., disorganized thinking or behavior) symptoms. Deficits in cognitive and social functioning are also associated with SSDs. Among the most severe and debilitating mental illnesses (Murray & Lopez, 1996), SSDs typically begin in late adolescence/young adulthood, often have a chronic and relapsing course, and can result in devastating functional outcomes. Prior to the clinical onset of psychosis, the “prodrome” is a period of time that is characterized by increasing symptoms and gradual functional decline that can span months to years before clinical threshold is met (Cornblatt et al., 2003). Current consensus is that research on the prodrome will allow for greater understanding of the mechanisms of onset and progression of SSDs, as well as provide opportunities for preventive intervention efforts that can target those most likely to benefit (Addington & Heinssen, 2012).

SSDs are also characterized by marked deficits in social skills, which contribute to impairments in social competencies that are essential for initiating and maintaining meaningful relationships and obtaining employment (Bellack, Morrison, Wixted, & Muser, 1990). Deficits in social functioning are present in the first episode of psychosis (FEP), detectable in the prodromal phase, and increase as a function of illness chronicity (Addington, et al., 2008; Drake et al., 2007; Gørna, et al., 2008; Grant, et al., 2001; MacDonald et al., 1998; Voges & Addington, 2005). Therefore, in addition to being a

core characteristic of SSDs, social functioning deficits are among the most debilitating aspects of these illnesses.

It has been proposed that the social skills deficits observed in psychotic disorders are a consequence of impairments in the cognitive abilities required for effective social functioning. However, while nonsocial cognitive (NSC) abilities (e.g., processing speed, working memory, and verbal learning) are related to social functioning (Green, 1996; Green, et al., 2000; Green, Kern, &Heaton, 2004), performance on these tasks account for only 20-40 % of the variance in functional outcome (Green et al., 2000; 2008a). In addition, as with management of symptoms alone (Heydebrand et al., 2004), attempts at remediating NSC deficits have limited impact on functional (i.e., social and role) improvement (Kurtz et al., 2001). Resultantly, increasing efforts seek to identify other causes of social functioning impairment and develop treatment approaches for the remediation of the domains that contribute to them.

As evidenced by the measures included in current prediction algorithms intended to detect individuals in the putatively prodromal phase of a SSD, social functioning represents a global construct that implies performance across many domains. Encompassed within are the concepts of social skills (e.g., overt behaviors involved in successful social functioning) and social cognition (e.g., processes underlying social behavior), which are increasingly molecular in nature (Yager & Ehmann, 2006). More recently, research has begun to focus on the multiple social cognitive (SC) processes believed to underlie social behavior.

As described below, SC abilities involve multiple functions that are required for the perception and processing of social information. Furthermore, an extensive body of

literature indicates SC impairments in patients suffering from SSDs. More recently, an emerging body of research also provides evidence of SC deficits in the prodromal phase. To date, however, there has been little research aimed at determining how impairments in SC might contribute to the development of prodromal symptoms. The purpose of the present study is to examine SC in individuals in the putatively prodromal phase for a SSD, with the primary goal of elucidating the relation between SC deficits and the subsequent progression of illness onset. This approach has the potential to shed light on the role of SC deficits in contributing to the gradual increase in subclinical psychotic symptoms and functional impairment that characterizes the prodrome. As described below, by focusing on individuals with putatively prodromal syndromes, as opposed to a psychotic disorder, we maximize opportunities for identifying antecedents of SSDs and minimize problems due to confounding factors (e.g., psychotropic medication).

Social Cognition

Broadly, SC refers to the mental processes involved in social interactions, including perception, interpretation, and the ability to generate appropriate social responses to others (Adolphs, 2001; Brothers, 1990; Dunbar, 2009; Fiske & Taylor, 1991; Kunda, 1999). Recent reviews indicate that SC involves multiple cognitive processes and, as summarized below, included among these are the four SC processes most commonly studied in SSDs. More specifically, attributional bias, emotion processing/recognition, social perception/knowledge, and theory of mind (ToM).

Attributional bias refers to an individual's characteristic tendencies to explain the causes of the events that they experience. In general, attributions are causal statements that include or imply the word "because" and frequently occur in verbal expressions or

explanations of what they are inferred to be the causes of positive and negative events (Zullow, Oettingen, Peterson, & Seligman, 1988). Measurement of attributional biases is obtained through questionnaires and ratings from transcripts of interactions (Kinderman & Bentall, 1996; Lee, Randall, Beattie, & Bentall, 2004), with key distinctions made between external personal attributions (i.e., causes attributed to others), external situational attributions (i.e., causes attributed to situational factors), and internal attributions (i.e., causes attributed to one's self).

Emotion processing/recognition (also termed affect recognition/perception) broadly refers to the accurate perception and adaptive utilization of emotional stimuli. One of the more influential models for emotional processing puts forth a definition of emotional intelligence that includes the four components of identifying, facilitating, understanding, and managing emotions (Mayer, Saovey, Caruso, & Sitarenios, 2001). Included in this model is the domain of affect perception that is often measured in research involving individuals with schizophrenia (Edwards, Jackson, & Pattison, 2002; Kohler, Bilker, Hagendoorn, Gur, & Gur, 2000; Salem, Kring, & Kerr, 1999). Measurement includes a variety of ratings that vary broadly in approach, such as ratings of emotions displayed in faces or voices, and ratings of how individuals manage, regulate, or facilitate emotion that are derived from how they respond to written or videotaped vignettes of interpersonal interactions.

Social perception generally refers to the ability to ascertain social cues from behavior provided in a social context, and is closely tied to social knowledge and conventions. Therefore, measures of social perception assess the ability to identify social roles, societal rules, and social context (Penn et al., 2002; Sergi & Green, 2003; Toomey

et al., 2002). This is accomplished through tasks that require processing of nonverbal, paraverbal, or verbal cues in order to make inferences about complex or ambiguous social situations. While tasks include the observation of individuals acting on their own, they also can involve assessing the nature of a relationship between people who are interacting with each other.

Theory of mind (ToM; also known as mental-state attribution or mentalizing) refers to the ability to infer the intentions, dispositions, and beliefs of others (Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001; Frith, 1992). In other words, the ability to understand that others' mental states differ from one's own, and the capability to make inferences about the content of those mental states. Measurement can involve responding to questions about brief social vignettes or arranging cartoon panels in a sensible order in an effort to demonstrate their understanding of more complex and abstract concepts; such as perspective taking, nonliteral language use (e.g., sarcasm), and deception.

Neurobiological Underpinnings

As summarized below, there has been a plethora of research and theorizing on the brain regions and neural systems sub-serving SC. While there may be considerable functional overlap among regions (Abe et al., 2007; Platek et al., 2009), evolutionarily more recent neocortical regions have been implicated as being involved in SC processes (Adolphs, 2001; Dunbar 2009). More specifically, in 1990, Brothers wrote a seminal article proposing a neural system of SC composed of the orbitofrontal cortex, the superior temporal sulcus (STS), and the amygdala. Numerous subsequent investigations have confirmed these neuronal structures, in addition to others that may play secondary roles in the processing of social information. With regard to affect perception, emotion

regulation, and social cooperation, several investigations have suggested the involvement of the amygdala, together with the medial prefrontal cortex, orbitofrontal cortex, and rostral anterior cingulate (Adolphs, 2001; Hall et al., 2004; Meltzoff & Decety, 2003; Rilling et al., 2002). For empathy, ToM, self-reflection, and processing of social cues/gestures, numerous studies have implicated the inferior frontal gyrus, temporo-parietal junction, frontal and temporal poles, and medial prefrontal cortices (Adolph, 2001; Brüne et al., 2008; Calarge, Andreasen, & O’Leary, 2003; Cheng et al., 2009; D’Argembeau et al., 2007; Farrow, Whitford, Williams, Gomes, & Harris, 2001; Hall et al., 2004; Meltzoff & Decety, 2003; Montgomery & Haxby, 2008; Ortigue, Thompson, Parasuraman, & Grafton, 2009; Platek et al., 2006; Rilling et al., 2002; Saxe, 2006; Uddin, Kaplan, Molnar-Szackas, Zaidel, & Iacoboni, 2005; van Overwalle, 2009; Yamada et al., 2010).

Research on the neurobiology of SC (Baron-Cohen, Ring, Bullmore, Wheelwright, Ashwin, & Williams, 2000; Drevets & Raichle, 1998) has also distinguished between “hot” (i.e., affect-laden/personally relevant processes) and “cold” (i.e., information-driven/personally irrelevant processes) SC skills. This distinction is considered by some to be important because affect-laden information processing may differ from that of information-driven information processing. In fact, hot SC skills (e.g., emotion recognition) appear to be dependent on the ventral mPFC and the connections it has with the limbic system, while cold SC skills (e.g., ToM) seem to be dependent on the prefrontal cortex (PFC) and a network of other brain regions (e.g., inferior parietal cortex, occipitotemporal junction). According to a recent review of the literature on the

developing adolescent brain (Blakemore & Chodhury, 2006), these brain regions, in particular the PFC, go through significant changes during puberty.

Furthermore, within the last decade, research demonstrating that neocortical maturation continues beyond childhood, throughout adolescence, and into early adulthood has led to investigators to posit that increasing regulation of the subcortical regions during those later periods results in the emergence of SC capacities (e.g., Choudhury, Blakemore, & Charman, 2006; Paus, 2005; Rubia et al., 2006; Yurgelun-Todd, 2007). This is especially important when considering deficits in SC processes. In fact, the neuromaturation that occurs during this critical period may also involve aberrant synaptic and neuronal pruning hypothesized in SSDs (Keshavan, Anderson, & Pettegrew, 1994; Pantelis et al., 2003). Subsequently, research on SC, while still in the early stages, has become an area of increasing empirical inquiry in the early course and prodromal stage of psychotic disorders.

Differentiation from Nonsocial Cognition

There are several ways in which SC differs from NSC (e.g., learning and memory, processing speed, vigilance or attention, working memory, and reasoning/problem solving about non-emotional stimuli/events), including the type of stimuli processed, the relationship of the perceiver to the stimulus, and how performance in the presence of such stimuli is evaluated within research investigations (Penn, Corrigan, Bentall, Racenstein, & Newman, 1997). For NSC tasks, stimuli are affectively neutral and static, the relationship between perceiver and stimuli is unidirectional, and the research focuses on assessing for deficits in the ability to attend to that stimuli. With regard to SC tasks, stimuli are personally relevant and can range from the perception of single features of

individuals (e.g., eye gaze) to complex social interactions. Furthermore, the relationship between perceiver and stimuli is interactive and research examines biases (i.e., response style independent of task performance) in addition to assessing for deficits.

As summarized by Pinkham and colleagues (2003), studies involving non-psychiatric samples suggest that SC and NSC are partially independent constructs. For example, individuals with frontal or prefrontal cortex damage have impaired social behavior and functioning despite having intact NSC (e.g., memory and language) skills. In addition, prosopagnosia (i.e., face blindness) impairs ability to recognize faces despite other aspects of visual processing (e.g., nonsocial object recognition) and NSC capacities remaining intact. Finally, while individuals with Williams' syndrome tend to have relatively intact SC skills (i.e., facial affect/emotion recognition and first-order ToM skills) despite deficits in NSC (i.e., visual-spatial processes and general cognitive functioning/IQ), those with Asperger's syndrome tend to show SC impairments despite having intact NSC abilities. Taken together, these studies were suggested to indicate that there may be unique neural circuits that underlying SC relative to NSC.

Such summaries of the literature have prompted increased efforts to examine the neural mechanisms involved in NSC and SC. In brief, fMRI findings have consistently demonstrated similar yet different patterns of neuronal activation for individuals engaged in NSC versus SC tasks. In fact, results from a recent meta-analysis (Van Overwalle, 2009) revealed that SC tasks measuring the ability to infer temporary states (e.g., goals, intentions, and desires) of others and enduring dispositions (e.g., interpersonal norms and scripts) of self and others consistently engaged the TPJ and mPFC regions, respectively. However, the NSC processes (e.g., action monitoring, attention, dual task monitoring,

and episodic memory retrieval) believed to assist SC processes did not contribute uniquely to activation in the TPJ and mPFC. In sum, while NSC tasks influenced complex social cognitive processes to some degree, they did not play a critical role in them. In other words, there seems to be partially overlapping and partially distinct patterns of neural activation between NSC and SC tasks.

Social Cognition in SSDs

A growing body of literature has demonstrated consistent patterns of association between SC and a variety of aspects involved in social functioning (Couture et al., 2006). Even after controlling for NSC, SC has been shown to be related to social impairments in SSDs (Penn et al., 2001). Further, SC has been found to be more closely related to social functioning and a stronger predictor of community functioning and social behavioral problems in SSDs (Brüne, Abdel-Hamid, Lehmkämer, & Sonntag, 2007; Pjnenborg et al., 2009; Roncone et al., 2002). In fact, this is consistent with the findings that SC contributes has incremental predictive validity for functional outcome beyond that which is provided by NSC (Brekke, Kay, Lee, & Green, 2005; Poole, Tobias, & Vinogradov, 2000; Vauth, Rüscher, Wirtz, & Corrigan, 2004). As a result, SC has been indicated as a mediator to explain the association between NSC and social functioning (Brekke et al., 2005; Sergi et al 2006; Vauth et al., 2004). In sum, taken together these findings indicate SC has the potential to allow for greater understanding of outcome above and beyond what can currently be explained NSC alone.

As briefly mentioned above, historically, research on SC in SSDs falls into one of four overlapping areas of inquiry: attributional bias, emotion processing, social perception, and ToM (Green, Olivier, Crawley, Penn, & Silverstein, 2005; Green et al.,

2008b; Penn, Addington, & Pinkham, 2006). Results from a recent National Institute of Mental Health workshop (Green et al., 2008b) delineated SC as a high priority research topic in SSDs. In fact, more work on attributional bias, emotion processing, social perception, and ToM was specified. Since then, considerable research has demonstrated pervasive SC impairment in chronic (i.e., attributional bias, emotion perception, social perception, and ToM) and first-episode (i.e., attributional bias, emotion recognition and ToM) patient samples (Bentall et al., 2001; Bigelow et al., 2006; Bora et al., 2009; Garety & Freeman, 1999; Green et al., 2012; Horan et al., 2012; Kohler et al., 2009; Krstev, Jackson, & Maude, 1999; Sprong et al., 2007).

While these findings do suggest that impairments are not merely associated with illness chronicity, they are generally limited by statistical comparison of group differences within cross-sectional study designs. More specifically, while cross-sectional studies can compare SC performance between groups at a single point in time, longitudinal studies allow researchers to detect developments or changes in SC over time. However, taken together, two more recent companion studies have begun to address the question of whether SC deficit are stable over time.

First, a cross-sectional study by Green and colleagues (2012) compared chronic and first-episode patient samples to age-matched healthy control (HC) groups. Results from that study indicated a similar magnitude of impairment in SC (i.e., emotion recognition, social perception, and ToM) across patient groups, and that age had limited impact on performance for both. In addition, the companion study by Horan and colleagues (2012) revealed longitudinal stability of SC impairment (i.e., ToM, relationship/social perception, and emotional intelligence) across a 12-month follow-up

period in the FEP group. Together these two studies provide evidence that SC impairments are relatively stable over time, suggesting that deficits are trait-like and not simply state-dependent.

Relatedness to Nonsocial Cognition

Mancuso and colleagues (2011) recently examined the factor structure of SC in outpatients with SSDs. In brief, results provided evidence for SC as a multidimensional construct with hierarchically distinct lower- and higher-level abilities. In fact, the three factors of hostile attributional style, lower-level social cue detection (i.e., facial emotion identification, social perception, lie detection/first-order ToM), and higher-level inferential regulatory processes (i.e., sarcasm detection/second-order ToM, and emotional intelligence/regulation) provided the best fit to the data.

In light of these findings, a recent meta-analysis of research on patients with SSD by Ventura and colleagues (2011) sought to examine the relatedness of the six NSC domains included in the Measurement and Treatment Research to Improve Cognition in Schizophrenia (MATRICS; Nuechterlein et al., 2008) battery to four SC domains (i.e. emotion perception and processing, social perception and knowledge, attributional bias, and ToM) similar to those examined by Mancuso and colleagues (2011). Findings did support prior results in that the relationship between each NSC domain and each remaining SC domain were moderate in magnitude ($r = -0.22$ to -0.33). Thus, because NSC only explained approximately 5-11% of the variance in SC, these findings support prior assertions that SC and NSC are related but separate constructs. In addition, because specific NSC abilities were not selectively associated with specific SC processes, they also reveal that no one NSC ability is a prominent component of SC performance.

However, while such findings indicate that NSC abilities are not the primary determinant of SC performance, the question of whether a hierarchical relationship exists between NSC and SC (i.e., possible to have impaired NSC but normal-range SC performance) has largely gone unaddressed.

Because of that, Fanning and colleagues (2012) sought to examine this in a sample of 119 out patients with schizophrenia. Findings revealed that NSC generally explained 10-20% of the variance in SC (with an average shared variance of 10%), leaving a significant portion of SC performance unexplained by NSC ability. Given that 25% of the participants had impaired SC despite having intact NSC, such findings do provide evidence that intact NSC is not sufficient for good SC performance.

Nonetheless, because good SC performance was extremely rare (<1%) in the presence of poor NC, findings also suggest that adequate NSC performance is a necessary basis of SC. Therefore, although strong claims cannot be made based on one study alone, findings do provide preliminary evidence for a building block model of SC which suggests that intact NSC may be necessary but not sufficient for good SC performance (Ostrom, 1984; Penn et al., 1997). As a result, they suggest it is necessary to consider NSC (e.g., general cognitive functioning/IQ) when investigating the relatedness of SC abilities to other key domains of interest (e.g., clinical symptoms and functional outcomes).

Relatedness to Clinical Symptoms and Functional Outcome

In light of the well-established literature on SC deficits in patients with SSDs, researchers have more recently focused on the question of how these deficits are linked with illness progression. First, what is the relatedness of SC deficits to functional

impairment? Second what is the nature of the relationship between SC deficits and clinical symptom domains? Third do demographic or disease burden variables explain variability within each SC domain to the degree that they should be considered when investigating the relatedness of SC domains to functional impairments or clinical symptoms?

In an attempt to begin addressing these questions, after determining the factor structure of SC in outpatients with SSD, the aforementioned study by Mancuso and colleagues (2011) also investigated the relatedness of the three factors to clinical symptoms and functional outcome. While hostile attribution was positively correlated with clinical symptoms (i.e., positive, depression-anxiety, agitation), lower-level (i.e., facial emotion identification, social perception, lie detection/first-order ToM) and higher-level (i.e., sarcasm detection/second-order ToM, and emotional intelligence/regulation) cue detection were positively associated with functional outcome. Also, lower-level cue detection accounted for unique incremental variance in functional capacity over and above both negative symptoms and NSC. Such findings suggest that there also may be distinct patterns of relatedness to clinical symptoms and functional deficits. Nevertheless, although a good initial step, studies with larger samples and greater representation across sex and age are required to parse out the independent relatedness SC domains with clinical symptoms and functional impairment.

More specific to the first question concerning functional impairment, a recent meta-analysis by Fett and colleagues (2011a) sought to examine the associations between NSC and SC and different types of functional outcome (e.g., community functioning, social behavior in the milieu, social problem solving, and social skills). Results from that

study indicated that community functioning (i.e., independent living skills and social/work functioning that are direct indicators of everyday functioning), was more strongly related to SC (in particular, ToM) than NSC. As the authors also point out, such findings are consistent with prior research discussed above that suggests SC explains unique variance in outcome, despite the likelihood of requisite underlying NSC abilities (Fanning et al., 2012; Ostrom, 1984; Penn et al., 1997).

In fact, they are also consistent with findings from a recent meta-analysis of 15 studies that investigated SC as a potential mediator of the relationship between NSC performance and functional outcome in SSDs (Schmidt, Mueller, & Roder, 2011). Results from that study indicated that the relatedness of NSC and functional outcome is at least partially mediated by SC domains (ES = 0.20 for the mean standardized indirect effect). In addition, the authors further analyzed their mediation hypothesis through the use of structural equation modeling in a sample of 148 patients with SSDs. Findings from those analyses were consistent with those from the meta-analysis, suggesting SC as a mediator between NSC and functional outcome in SSDs.

With regard to second question pertaining to clinical symptom domains, Fett and colleagues (2013) recently sought to address the relatedness of SC domains to clinical symptoms in a study that compared patients with SSDs to their unaffected siblings and a HC group. In brief, they focused on the two most studied domains of SC in SSDs (i.e., ToM and facial affect/ emotion processing) that have also been suggested to play a role in the development of psychotic/positive symptoms. Within the patient group, ToM performance was inversely related to positive, negative, and disorganized symptoms, with the magnitude of relation increasing in that order. Although to a lesser degree than

ToM, performance on the emotion processing task was also inversely related with both disorganized and negative symptoms.

Such findings are also consistent with those from the aforementioned meta-analysis by Ventura and colleagues (2011). More specifically, the relatedness of the SC domains and reality distortion were nearly zero to moderate, with emotion perception/processing and social perception/knowledge having moderate relationships ($r=-0.22$ and -0.21 , respectively) and attributional bias and ToM having minimal relationships ($r=-0.07$ and -0.08 , respectively). In addition, magnitude of relatedness was significantly different across SC domains (e.g., emotion perception and ToM). In contrast, relatedness to disorganized ($r=-0.22$ to -0.32) and negative symptoms were ($r=-0.20$ to -0.26) were consistently moderate across all SC domains.

Finally, in addition to cross-sectional investigations, one prospective longitudinal study (Hamm et al., 2012) has provided additional evidence that SC deficits (i.e., ToM/metacognition capacity and emotion recognition) are significantly correlated with each other and stable over time in individuals with SSDs. In this study, SC (i.e., ToM/metacognition) was also inversely related to both negative and disorganized symptom domains at baseline. Furthermore, after controlling for baseline negative symptoms and NSC (i.e., executive functioning), SC deficits (i.e., Tom/metacognition) were uniquely related to negative symptoms at 6-month follow-up. Thus, results from this study suggest that SC (i.e., ToM/metacognition) contributes to the development of negative symptoms.

Pertaining to the third question on demographic and disease burden variables, Fiszdon and colleagues (2013) recently sought to further characterize the nature of SC

deficits in SSDs by determining demographic, symptom and functional correlates, while providing more evidence of the types of SC deficits that are found in SSDs. In fact, in a sample of 119 stable outpatients, relatedness of SC to clinical symptoms were similar to those found by Fett and colleagues (2013) and Ventura and colleagues (2011).

Furthermore, consistent with results from the meta-analysis by Fett and colleagues (2011), there also were small to moderate correlations with functioning measures.

Finally, within analyses of demographic characteristics (i.e., age, years of education, rate of hospitalization, and age at onset of psychosis) age was associated with a measure stated to be closely related to ToM and attributional bias.

Such results are fairly consistent with a recent meta-analysis by Savla and colleagues (2012) that sought to determine the average magnitude of differences between patient samples and HCs, and the extent to which demographic or disease burden variables can explain the variability within each domain. As with the aforementioned meta-analytic results from studies that addressed ToM and emotion recognition, results indicated that patients performed worse than HCs across SC domains, with large effects (Cohen's $d = 0.89-1.04$) in decreasing order of social perception, ToM, emotion perception, and emotion processing. Furthermore, regression analyses revealed that variability within domains was not explained by age, education, or gender. However, greater deficits in social and emotion perception were associated with inpatient status, and deficits in emotion processing were positively correlated with illness duration/chronicity.

Summary

Based on the studies available to date, it is apparent that SC is partially independent of NSC, indicating that it provides unique information about functional outcome. In addition, SC has consistent relationships with functional outcome, with meta-analytic results suggesting that these relationships are stronger than those between NSC and functional outcome. Furthermore, SC has been shown to act as a mediator between NSC and outcome, and in the models available to date, it is more proximal to functional outcome than NSC.

When considering clinical symptom domains, the most consistent trend in cross-sectional research is that SC deficits (i.e. emotion perception/processing, social perception/knowledge, and ToM) are moderately related to disorganized and negative symptoms. In addition, for the specific positive symptom of reality distortion, magnitude of relatedness appears to vary from minimal (i.e., attributional bias and ToM) to moderate (i.e., emotion perception and social perception). Similarly, one additional cross-sectional study has revealed that the two most commonly studied SC deficits (i.e., ToM and emotion processing) are inversely related to all three broad clinical symptom clusters, in increasing magnitude from positive, negative, and disorganized symptom domains. Finally, one longitudinal study provides preliminary evidence that SC (i.e., ToM/metacognition) may be a predictive determinant for negative symptoms.

While current findings indicate significant relations of SC with both clinical symptoms and functional outcomes, the relative contribution of each SC domain to clinical symptoms and functional decline (i.e. role and social) remains unclear given the limited number of prospective investigations available to date. In addition, although emerging evidence suggests demographic and disease burden variables can explain

variability within SC domains, the impact of these characteristics on the relationships between SC and functional impairments and/or clinical symptomatology is not yet known. As a result, further research is needed to clarify relatedness of SC to clinical symptoms and functional outcomes, while accounting for potentially confounding covariates (e.g., age, sex, education or general cognitive functioning/IQ, and illness duration/chronicity) in samples of individuals with SSDs.

Social Cognition in At-Risk Groups

Because the ‘prodrome’ is a retrospective construct that suggests inevitability of illness onset, contemporary prospective research standardized measures to index subclinical symptoms and those that meet a specified severity threshold designate “clinical high risk” (CHR) status (Klosterkötter, et al., 2001; Miller et al., 2003; Riecher-Rössler et al., 2008; Yung et al., 2005). An emerging body of evidence indicates that CHR samples have SC deficits (i.e., emotion recognition and ToM) intermediate to HCs and first-episode patients (Green et al., 2012; Horan et al., 2012; Kim et al., 2011; Thompson, Bartholomeusz, & Yung, 2011). While findings are not consistent across studies (Couture et al., 2008; Pinkham et al., 2007), limited sample sizes have constrained the statistical power that can be achieved (e.g., accounting for potential confounders). As a result, further characterization of deficits in CHR samples is required, and these studies should involve larger samples with measures that assess multiple SC processes.

Nonetheless, two studies have examined the relationship of NSC and SC to clinical symptoms and rate of psychosis conversion (e.g., positive symptoms + functional decline) in CHR individuals. For the first, Jalbrzikowski and colleagues (2012) examined SC and NSC in a cross-sectional comparison of HCs ($n=31$) and individuals with

22q11.2 microdeletion syndrome (22qDS; $n=31$); one of the largest known genetic risk factors for psychosis. Groups were matched on key demographic characteristics (e.g., age, sex and education), and relative to controls, the 22qDS group did not show expected age-related improvements in working memory or verbal knowledge. Also, processing speed was the best predictor of negative symptoms, and ToM was the best predictor of positive symptoms. Results were suggested to indicate that SC may be an intermediate vulnerability marker (i.e., relate more directly to underlying genes and neurocircuitry than current symptom criteria) with predictive utility in detecting individuals at genetic high risk (GHR) for psychosis.

In the second study, Kim and colleagues (2011) conducted a longitudinal examination assessing for the utility of SC tasks to predict conversion rate over and above NSC tasks in a CHR sample. While groups did not differ with regard to clinical symptoms, a global functioning measure, age, gender, or parental socioeconomic status, HCs were more educated than the CHR group and converters had a lower IQ than both nonconverters and HCs. At baseline, while covarying for education, CHR converters ($n=13$) performed significantly worse than CHR nonconverters ($n=26$) and HCs ($n=45$) on tasks assessing ToM and tasks assessing executive function (i.e., processing speed/set-shifting), working memory, visual memory, and verbal memory. Finally, psychosis conversion was significantly predicted by a model combining ToM and select NSC tasks (i.e., working memory, visual memory, and executive function). However, due to the small sample size, independent contributions of SC and NSC measures could not be established. Nonetheless, results were interpreted to suggest the predictive utility of ToM for detecting individuals at CHR for SSDs.

Although these two studies indicate that SC (i.e., ToM) may have predictive utility in CHR samples, very little investigation to date has sought to determine if relations between SC and symptom domains or functional outcome vary by subtype of SC (e.g., ToM and emotion recognition). In addition, given that certain thresholds for both clinical symptom and functional decline are used designate CHR and conversion status, those studies only indirectly assess causal relationships between SC deficits and clinical symptoms and/or functional outcome. In fact, they only provide evidence about the predictive utility of these deficits with regard to the time to transition for psychosis. Finally, these studies are limited with regard to their assessment of the relatedness of key sample characteristics of interest (e.g., age, sex, and educational attainment or general cognitive functioning/IQ) to SC performance, as well as their impacts on the relatedness of SC to clinical symptoms and/or functional outcome.

Summary and Conclusions

In sum, a plethora of research has demonstrated pervasive SC impairment in chronic and FEP patient samples. Emerging evidence also suggests that CHR groups exhibit performance on SC tasks (e.g., attribution, emotion recognition, and ToM) that is intermediate to HC and FEP groups, and that SC deficits (i.e., emotion recognition and ToM) are more pronounced in converters than nonconverters. In addition, while two studies of CHR samples suggested the potential predictive utility of SC (i.e., ToM) for positive symptoms at baseline, one has also provided preliminary evidence that SC measures (i.e., ToM) predict faster conversion rates even when controlling for NSC. Finally, although not yet examined in CHR samples, evidence for temporal stability of impairment in those with recent onset of a SSD suggests that deficits are trait-like rather

than transient. However, given the infancy of this area of empirical inquiry, there is much we do not yet know about the earlier phases of SSDs and SC deficits.

Clearly more research is required before substantive claims can be made about the degree to which impairments in SC are present in CHR groups. In addition, given the relative dearth of research available to date, future studies are needed in order to make firm conclusions about the relatedness of SC deficits to key domains of interest (e.g., NSC, symptoms, and functional impairment) in CHR samples. Nonetheless, at the present time, studies involving patients with SSDs do provide some evidence indicating that SC deficits are associated with more severe symptoms, especially negative symptoms, even when NSC is controlled for. Furthermore, research indicates that SC deficits are stable and predictive of functional outcome.

As discussed above, one question that has yet to be answered concerns the longitudinal relation of SC deficits with both clinical symptoms and functional outcome. In particular to what extent is SC contributing to future symptom severity or functional deficit? While no single study can answer these questions, longitudinal research on the relation of SC performance with symptom progression or functional deterioration over time can shed light on this question. Further, addressing these issues in a CHR sample, as opposed to diagnosed patients, reduces the challenges posed by treatment effects. Such investigations should involve samples large enough to provide sufficient power for multiple comparisons and use of the more advanced statistical modeling techniques. Doing so would also allow for control of potentially confounding variables (e.g., age, sex, education, and general cognitive functioning/IQ) in the examination of the relatedness

between key domains of interest (e.g., symptoms and functional outcome) in CHR groups.

Goals of the Current Study

In the present study, the goal is to expand on previous research by characterizing SC deficits in a CHR sample large enough to examine relatedness of SC performance (i.e., emotion processing/recognition, ToM, and relationship/social perception) to clinical symptom domains (i.e., positive, negative and disorganized) and functioning (i.e., social and role), while controlling for potentially confounding variables gleaned from the body of literature on SC in psychosis and prodromal groups (i.e., age, sex, and general cognitive functioning/IQ). In addition, while accounting for potential confounders, analyses will investigate whether baseline SC deficits have predictive utility for later symptom levels and functional outcomes at 12-month follow-up.

Based on past research, the present study will test the following hypotheses. First, greater severity of social cognitive deficits is predicted in the CHR group than in the HC group. In addition, within the CHR group, measures of social cognition are expected to be significantly correlated with each other and with general cognitive functioning/IQ. Furthermore, given evidence of sex differences in social cognitive abilities in SSDs, greater SC deficits are predicted in CHR males than CHR females. While controlling for potential confounds (i.e., age, sex, years of education, and general cognitive functioning/IQ), it is also predicted that baseline SC deficits will be associated with baseline severity of all three clinical symptom domains in the CHR group, and those deficits will predict the worsening of symptoms over the course of 12 months. Finally, while controlling for potential confounds (i.e., age, sex, years of education, and general

cognitive functioning/IQ), SC deficits are predicted to be associated with baseline functional impairment in the CHR group, and that those deficits will predict the increasing levels of functional impairment over the course of 12 months.

Method

Participants

This current study included the subsample of participants ($n = 462$) who completed the entire social cognitive battery for the baseline assessment and 12-month follow-up clinical assessment of the ongoing North American Prodrome Longitudinal Study (NAPLS) as of October 2011. Funded by the National Institute of Mental Health for the purposes of determining correlates and predictors of psychosis onset, as described previously (Addington et al., 2012), NAPLS is a consortium comprised of eight research sites located at the University of Calgary, the University of California at San Diego, the University of California at Los Angeles, Zucker Hillside Hospital, the University of North Carolina, Harvard University, Yale University, and Emory University.

Procedures

CHR participants were largely obtained through two sources. First, healthcare providers, educators, or social service agencies referred potential participants from their communities. Others self-referred themselves in response to community announcements (e.g., grand rounds in academic settings, psychoeducational presentations, mailings, postings, websites, public service announcements, and public advertisements). The HC group was primarily recruited through three sources, including a combination of community outreach and advertisements.

All potential participants were first screened over the telephone in order ensure that they were between 12 and 35 years of age, and as an initial attempt to exclude those with possible Axis I psychotic disorders, mental retardation, or current substance abuse or dependence. Those deemed likely to meet criteria for a CHR syndrome were invited to participate in an in-person screening assessment.

During the screening appointment, potential participants were informed about study procedures and provided informed consent. As approved by the Emory University Institutional Review Board, in the case of those below 18 years of age, assent was obtained from the minor and written informed consent was obtained from a parent or legal guardian. After consent, using measures described below, clinical assessments determined if potential participants met criteria for a CHR syndrome and whether or not they also met criteria for any Axis I or Axis II cluster A personality disorder. Additional measures gathered information on demographic characteristics, global social and role functioning, family history of mental illness, and history of treatment utilization, head injury, and substance use.

For both groups (i.e., CHR and HC) participants were excluded if they had current or lifetime history of an Axis I psychotic disorder, an intellectual quotient (IQ) below 70, current or lifetime history of a clinically significant central nervous system disorder that may contribute to prodromal symptoms or confound their assessment, current or lifetime history of a closed head injury, or substance dependence within the 6 months immediately prior to screening assessment. Unless better accounted for by prodromal symptoms, non-psychotic Axis I disorders were also exclusionary criteria. Furthermore,

participants were also excluded if they had a lifetime history of treating psychotic symptoms with antipsychotic medication for a period four or more weeks.

In addition to the inclusion and exclusion criteria for both groups, participants were excluded from the HC group if they met criteria for any CHR syndrome, or any current or past Axis I disorder/Axis II Cluster A personality disorder diagnosis. Exclusionary criteria also included a family history (i.e., first- or second-degree relative) of SSDs (e.g., schizophrenia, schizoaffective disorder, schizotypal personality disorder) or any other disorder involving psychotic symptoms. After screening for inclusion and exclusion criteria, potential participants were discussed on a weekly clinical conference call with investigators from each site of the NAPLS consortium. Participants were admitted to the study if consensus was achieved.

Measures

CHR Designation

The *Structured Interview for Prodromal Syndromes* (SIPS; Miller et al., 1999) is a reliable and valid (Miller et al., 2002), semi-structured diagnostic interview purposed to determine if individuals met the Criteria of Prodromal Syndromes (COPS; McGlashan) used to identify CHR status. Symptoms are rated on a seven-point scale reflecting severity, frequency, duration, and intensity/degree of conviction. Scores range from zero to six, with zero to two reflecting what is considered to be normal/subprodromal symptomatology, three to five indicative of a prodromal level of symptomatology/CHR status, and scores of six suggesting the possibility of a psychotic state. Items allow for assessment of five positive symptoms, six negative symptoms, four disorganized symptoms, and four general symptoms, in addition to recording data on criteria for

Schizotypal Personality Disorder, family history of mental illnesses, and current- and past-year global functioning. Ratings are averaged to derive a score for each symptom dimension.

The *Structured Interview for DSM-IV Personality Disorders* (SIDP-IV; Pfohl, Blu, & Zimmerman, 1997) is a semi-structured interview designed to rate personality disorder criteria based on DSM-IV Axis II criteria. Items are rated on a scale from zero (not present) to three (strongly present), with average ratings of symptom criteria resulting in a total symptom score.

The *Structured Clinical Interview for DSM-IV Axis I Disorders* (SCID-IV; First, Spitzer, Gibbon, & Williams, 2002) is a semi-structured interview designed to verify and categorize the presence of Axis I disorders according to DSM-IV criteria.

Assessment of Functional Outcome

The *Global Functioning: Social and Role Scales* (GF:S and GF:R; Cornblatt et al., 2007) are clinician-rated scales that are similar in scope and design to the Global Assessment of Functioning Scale (GAF; Skodall et al., 1988) and the Social and Occupational Functioning Assessment Scale (SOFAS; Goldman, Skodol & Lave, 1992). These well-anchored scales take age and phase of illness into account, allowing for social and role functioning to be examined independent of the potential confound of clinical symptoms. More specifically, the GF:S assesses quantity and quality of peer relationships, level of peer conflict, age-appropriate intimate relationships, and involvement with family members. For the GF:R, level of performance is evaluated based on the primary role of the participant (i.e., student, employee, or homemaker). For

both, total scores can range from 1 to 10, with the former indicating extreme dysfunction and the latter indicating superior functioning.

Assessment of Nonsocial Cognition/General Cognitive Functioning/IQ

The *Wechsler Abbreviated Scale of Intelligence* (WASI; PsychCorp, 1999) was designed to provide a short and reliable estimate of overall intellectual abilities/IQ. Participants in the current study completed the Vocabulary (WASI-V) and Block Design (WASI-BD) subtests of the WASI. For the WASI-V, purposed to assess for expressive vocabulary, verbal knowledge, and fund of information, participants were asked to provide definitions for up to a maximum of 42 orally and visually presented words. With regard to the WASI-BD task, which taps visual-spatial skills, visual-motor coordination, and abstract conceptualization skills, participants were asked to replicate 13 visually presented patterns using two-color (i.e., red and white) cubes. Both of tasks yield raw scores and age- and sex-normed standard scores, and when summed together, provide an estimate of general cognitive functioning/IQ.

Assessment of Social Cognitive Domains

The *Penn Emotion Recognition Test* (ER40; Kohler et al., 2000) is a computerized emotion identification task in which 40 photographs of adult faces vary by race and gender. Participants identified the emotion (i.e., happy, sad, anger, fear, or no emotion) shown on each face. Each emotion type was randomly presented eight times. Responses sum to an overall score that can range from 0 to 40. The ER40 task has adequate test-retest reliability (Carter et al., 2009; Rojahn et al., 2000), has been widely used in studies addressing SSDs (e.g., Butler et al., 2009), and has begun to be used in at-risk samples (e.g., Jalbrzikowski et al., 2012).

The Awareness of Social Inference Test (TASIT; McDonald, Flanagan, & Rollins, 2002) Part III: This social inference-Enriched task is an untimed computerized task that is intended to assess an individual's ability to comprehend the intentions of others. Comprised of 16 video vignettes of 15 to 60 seconds in length, the approximate total length of this task is 15 minutes. Administered in a fixed random order, eight of these vignettes involve one person telling another person a lie, while the other eight include an interaction in which someone makes use of sarcasm. Between each vignette, the task was paused so that participants could respond to a set of four forced-choice (yes/no) questions that addressed: what one person was trying to make another think or feel (i.e., what he/she was doing to another person); the message one person was trying to get across to another person (i.e., what he/she was trying to say to another person); the underlying belief (i.e., what the he/she was thinking); and what emotion a person was feeling or how a given person felt about another person/toward a particular situation (i.e., what he/she was feeling). Responses sum to an overall total score that can range from 0 to 64. Shown to have adequate reliability and validity in samples of brain injured patients (McDonald et al., 2006), the TASIT has also been used in CHR and FEP groups (Green et al., 2012; Jalbrzikowski et al., 2012).

The *Relationships Across Domains* (RAD; Sergi et al., 2009) task is a paper and pencil measure of competence in relationship perception. Comprised of 25 brief written vignettes (two-to-four sentences) involving male-female dyads, each is consistent with one of four relational models (i.e., communal sharing, authority ranking, equality matching, and market pricing) believed to govern social behaviors across social interaction domains. Based on relational models theory (Fisk, 1992), it is believed that

individuals make use of these models in order to both understand social relationships and make inferences about the behavior of social partners in different domains. Each vignette is followed by three statements describing that dyad's interpersonal behavior in different social domains (each consistent with one of the relational models). Participants decide if the three statements are likely (yes/no) and responses sum to an overall total score that can range from 0 to 75. Validated for use in studies involving individuals with SSDs (Sergi et al., 2009), the RAD has also been used in studies including both patient (i.e., FEP and chronic) and CHR groups (e.g., Green et al., 2012; Horan et al., 2012).

Data Analysis

All analyses were performed using PASW version 22.0. First, basic descriptive statistics were calculated for the overall sample. Second, independent samples t-tests and chi-square tests were utilized to compare the CHR and HC groups on baseline demographic and clinical variables. Third, correlation coefficients were computed to determine the relationships between proposed predictor and dependent variables in the CHR group. Fourth, independent samples t-tests were utilized to compare SC performance by sex within the CHR group. Fifth, a series of linear regressions were used to determine the variance explained by baseline SC performance for each clinical symptoms/functioning variable of interest (i.e., SIPS Positive, SIPS Negative, SIPS Disorganized, Role Functioning, and Social Functioning scales) at baseline and 12-month follow-up in the CHR group.

Results

Demographic and clinical characteristics for the overall sample are reported by group in Table 1. There were no significant differences in age, race, or ethnicity between

the CHR and HC groups. However, as expected, the HC group had a significantly higher general cognitive functioning/IQ than CHR group. In addition, as expected, the CHR group had significantly higher symptom severity (i.e., positive, negative, and disorganized), significantly lower social and role functioning, and significantly worse performance on all three SC tasks.

Results from regression analyses used to determine whether diagnostic group moderated the relationship between age and SC performance are presented in Table 2. For all three models, age and diagnostic group were included on the first block and the interaction term (i.e., age x diagnostic group) was entered on the second block. Findings indicate that diagnostic group moderates the relationship between age and performance on both the TASIT and ER40, but not the RAD. Examination of the interaction plots revealed that, as age increases, performance on the TASIT and ER40 tasks increases for both HCs and CHRs. However, the increase is greater for HCs than CHRs for both tasks.

Results from correlational analyses for proposed predictor variables in the CHR group are displayed in Table 3. As expected, the three SC measures were significantly correlated with each other within the CHR group. However, age, education, and the general cognitive functioning/IQ variables were only significantly correlated with the RAD and the TASIT.

Sex differences in SC performance within the CHR group are reported in Table 4. As expected, males had lower scores on all three social cognitive tasks than females in the CHR group. However, males only performed significantly below females on the RAD and TASIT tasks.

Results from regression analyses determining variance explained by SC performance for baseline clinical symptoms, when controlling for age, sex, years of education, and general cognitive functioning/IQ are displayed in Table 5. The control variables (i.e., age, years of education, and general cognitive functioning/IQ) were entered on the first block, the SC variable of interest (i.e., ER40, RAD, TASIT) was entered on the second block, and the interaction term (SC variable of interest x Sex) investigating the influence of sex on the relationship between the SC variables and the dependent variable of interest was entered on the third block. None of the SC variables were significantly associated with positive or negative symptom severity in the CHR group. However, while results only indicate a trend toward a significant main effect for the RAD, the TASIT and ER40 tasks were significantly inversely associated with disorganized symptom severity. In addition, while results only indicate a trend toward a significant main effect for the RAD and TASIT, the ER40 task was significantly positively associated with baseline global role functioning. Finally, only the ER40 task was significantly positively associated with baseline global social functioning. Sex did not moderate any of the relationships between SC variables and baseline clinical symptoms or functioning.

Results from regression analyses determining variance explained by baseline SC performance for 12-month clinical symptoms/functioning variables of interest, when controlling for age, sex, years of education, general cognitive functioning/IQ and baseline clinical symptom severity or functioning are displayed in Table 6. The control variables (i.e., age, years of education, general cognitive functioning/IQ, and the baseline clinical symptom/functioning variable) were entered on the first block, the SC variable of interest

(i.e., ER40, RAD, TASIT) was entered on the second block, and the interaction term (i.e., SC variable of interest x Sex) examining the influence of sex on the relationship between the SC variable of interest and clinical symptom or functioning variables of interest was entered on the third block. Not one of the SC variables was significantly predictive of clinical symptoms or functioning at 12-month follow-up. Thus, there was no evidence of a predictive relationship between baseline SC and follow-up symptoms and functioning when baseline levels were controlled for.

Discussion

As described above, a plethora of evidence indicates pervasive SC impairment in chronic and first-episode patients, and emerging evidence suggests that CHR groups exhibit SC deficits intermediate to those found in HC and FEP groups. However, given mixed results from only a handful of studies comprised of a variety of study designs, firm conclusions about the degree of SC impairments in CHR groups, the influence of sex on SC performance in CHR groups, and predictive utility of SC for clinical symptoms and functioning in CHR groups are not yet plausible. In addition to comparing SC performance between CHR and HC groups, the present study compared SC deficits in a CHR sample large enough to examine the influence of sex on SC performance and relatedness of SC performance to clinical symptoms and functioning while examining and controlling for confounding variables of interest at baseline and 12-month follow-up.

In brief, findings provide support the presence of SC deficits in the CHR group relative to the HC group. With regard to sex differences in SC performance within the CHR group, as discussed below, results varied depending on the SC domain measured. Furthermore, results provide mixed support for relatedness of SC deficits and clinical

domains of interest at baseline and no support for predictive utility of baseline SC deficits for clinical symptoms and functioning at 12-month follow-up.

Diagnostic Group Differences: CHRs vs. HCs

As expected, CHRs had significantly lower general cognitive functioning/IQ than HCs. However the values still indicate intact NSC, considered as necessary but not sufficient for good SC performance (Ostrom, 1984; Penn et al., 1997). The CHR group also had significantly worse social and role functioning, significantly higher symptom severity (i.e., positive, negative, and disorganized symptoms), and significantly worse SC performance than HCs. Furthermore, regression analyses indicated that the relationship between age and social cognitive performance is moderated by diagnostic group for both the TASIT and ER40 tasks, with HCs appearing to have a stronger positive relationship between age and performance than CHRs. Such results are consistent with a recent review suggesting that CHR groups are associated with milder versions of psychological deficits (including SC performance) and biological abnormalities that are intermediate to HCs and patients diagnosed with psychotic disorders (Goulding et al., 2013).

SC, General Cognitive Functioning/IQ and Sociodemographics in the CHR Group

Before turning to a discussion of findings relevant to study hypotheses, it is of interest to mention the relations of the SC tasks with general cognitive functioning/IQ and education, as well as the relations among the SC tasks themselves. First, as shown in Table 3, the modest positive correlations among SC measures indicate they are tapping into the general construct of SC. Yet, for the RAD and TASIT, the correlations with IQ and education are positive and larger in magnitude, indicating general intellectual factors

play a substantial role in determining SC abilities in these domains. This is generally consistent with previous studies on the role of general cognitive ability in SC.

While performance on the RAD was significantly positively associated with age, education and general cognitive functioning/IQ, performance on the TASIT was only significantly positively associated with education and general cognitive functioning/IQ, and performance on the ER40 task was not significantly associated with age, education, or general cognitive functioning/IQ. While it may seem counterintuitive that the lower-level/first-order cue detection task (ER40) was not associated with any of these variables, it is important to remember that emotion recognition begins at birth and an elaborate system is already in place by 3 years of age (Tonks, Williams, Frampton, Yates, & Slater, 2007), with dramatic improvements in facial expression recognition (Kolb & Taylor, 1992) and ability to read emotions from eyes (Baron-Cohen, Wheelwright, Scahill, Lawson, & Spong, 2001) occurring the ages of 8 and 10 years.

In contrast, higher-level/second-order cue detection tasks (i.e., TASIT and RAD) begin later on in early childhood and then improve gradually through experiences with interpersonal interactions in both adolescence and early adulthood. For example, while ToM is believed to begin with pretend play and joint attention as a toddler (Onishi, Baillargeon, & Leslie, 2007), the ability to infer emotion from speech does not occur until between 4 and 10 years of age, followed by a gradual shift from a tendency to judge emotion from literal language to reliance on affective cues throughout adolescence that is fully present in adulthood (Morton & Trehub, 2001). In fact the two requisite skills for ability to detect faux pas (recognizing that the person who stated the faux pas did not understand it's inappropriateness and appreciating the feelings of the recipient), the

benchmark for ToM typical of adulthood, does not occur until between 9 and 11 years of age (Onishi, Baillargeon, & Leslie, 2007).

As a result of these differing developmental trajectories, it is not surprising that age and years of education are not significantly positively associated with the ER40 tasks as they are with the TASIT and RAD tasks in this sample of participants ranging from 14 to 25 years of age. In addition, given the complex nature of higher-level/second-order cue detection tasks relative to lower-level/first-order cue detection tasks, it also is not surprising that the general cognitive functioning/IQ variable is not significantly positively associated with the ER40 task as it is with the TASIT and RAD tasks. The general cognitive functioning/IQ variable assesses expressive vocabulary, verbal knowledge, and fund of information through a straightforward vocabulary test. It also taps visual-spatial skills, visual-motor coordination, and abstract conceptualization skills using static nonsocial stimuli. While the RAD and TASIT tasks require more knowledge of *and* experience with expressive vocabulary and visual-spatial skills in to detect sarcasm/lies and understand the relationship between two people and their actions across situations, the ER40 tasks requires only basic recognition of straightforward, static depictions of facial affect.

Influence of Sex on SC Performance within the CHR Group

Similar to the results for age, years of education, and general cognitive functioning/IQ, while CHR males tended to perform less well on SC tasks than CHR females, males were only significantly worse on the RAD and TASIT tasks. Furthermore, sex was not found to moderate the relationships between SC tasks and clinical symptoms or functioning variables at either baseline or 12-month follow-up. Such results may

indicate that the ER40 task, a lower-level cue detection task (i.e., facial affect recognition), is sensitive to SC deficits that are independent of sex in clinically high risk populations. They also may indicate that performance on higher-level cue detection tasks (i.e., sarcasm and lie detection for the TASIT, and emotional intelligence/relationship perception for the RAD) are influenced by sex.

However, at this point in time, a paucity of studies investigating these relationships precludes strong conclusions. As a result, it is important to continue to attempt to account for the potential influence of sex on SC performance in CHR groups. In particular, since sex differences in social functioning/SC impairment have been detected in both psychosis and general population samples (Hass & Garratt, 1998).

Relatedness of Baseline SC to Baseline Symptoms/Functioning in CHRs

When controlling for sociodemographic variables of interest (i.e., age, sex, race, and ethnicity), none of the SC variables were associated with positive or negative symptoms at baseline. Also, the RAD was not positively associated with disorganized symptoms, global role functioning or global social functioning at baseline. However, baseline performance on the TASIT and ER40 tasks were significantly inversely associated with disorganized symptoms at baseline. In other words, worse baseline performance on the TASIT and ER40 tasks was associated with higher levels of baseline disorganized for CHR participants. Furthermore, baseline performance on the ER40 tasks was also significantly positively associated with global role functioning and global social functioning at baseline. More specifically, worse baseline performance on the ER40 task was associated with worse overall baseline social and role functioning for those in the CHR group.

While the lack of associations between SC tasks and positive and negative symptoms is somewhat surprising given the literature on SSDs summarized earlier (e.g., Fett et al., 2013), findings indicating that performance on the TASIT and ER40 tasks were positively associated with higher baseline levels of disorganized for CHR are consistent with studies linking SC to disorganized symptoms (e.g., Fett et al., 2013) in SSDs. The SIPS disorganized symptoms domain is assumed to tap obvious and/or bizarre behaviors (e.g., odd behavior or appearance, trouble with focus and attention, hygiene, bizarre thinking) rather than merely detecting normative behavioral oddities. It is possible that failure to detect emotions accurately (e.g., facial affect) and a compromised ability to detect sarcasm and/or lies could result in erroneous beliefs about the intentions of others in social situations. Such misinterpretations may result in awkward social interactions due to behavioral responses that others may determine to be non-normative or inappropriate. Within a workplace, this can result in an inability to work with others or serve others well. In close social relationships, misguided interactions can also cause problems over time. Such awkward/inappropriate interactions have the potential to cause a great deal of interpersonal strife and compromise one's ability to fulfill major life roles.

Although not all of life's roles require accurate detection of sarcasm and lies to successfully negotiate social interactions, most require at least somewhat accurate perception of expressed emotional cues (e.g., facial affect). Therefore, even though a previous study revealed that both first-order and second-order cue detection were positively associated with functional outcome in SSDs (Mancuso et al., 2011), it is not completely surprising that the ER40 task is the only SC task to be positively associated with the broad measures of global role and global social functioning used in this current

investigation. In fact, in that same study (Mancuso et al., 2011), only lower-level cue detection accounted for incremental variance in functional capacity over and above NSC in SSDs.

Predictive Utility of Baseline SC for 12-month Symptoms/Functioning in CHRs

When controlling for sociodemographic variables of interest (i.e., age, sex, race, and ethnicity) and baseline clinical symptoms/functioning, none of the SC variables were predictive of clinical symptoms (i.e., positive, negative, and disorganized) or functioning variables (i.e., social and role) at 12-month follow-up. While the lack of predictive utility of SC for predicting the subsequent course of symptoms or functioning, beyond baseline levels, is somewhat surprising, it is also important to keep in mind that the risk for transition to psychosis in individuals who meet CHR criteria extends for years after baseline assessment. Also, the body of literature investigating the relatedness of SC deficits and clinical symptoms/functional outcomes in CHR groups is limited to date. In fact, prior to this study, only two studies had proposed the potential predictive utility of SC in CHR groups (Jalbrzikowski et al., 2012; Kim et al., 2011). Also, of the two, only one study (Kim et al., 2011) looked at the relationship between SC and rate of psychosis conversion (as measured by increasing positive symptoms and functional decline). In that study, only indirect evidence for the predictive utility of SC impairments on clinical symptoms and functional outcome was provided because it assessed time to transition rather than relatedness to the symptoms or functional declines themselves.

If findings from the current study had indicated that SC predicted 12-month clinical symptoms and social/role functioning, when controlling for sociodemographics and baseline clinical symptoms/functioning, results would have provided direct evidence

for the potential⁰ for SC to predict prognosis in CHR subjects. Furthermore, they would have provided support for including SC measures in baseline assessment for future CHR studies attempting to enhance prediction of course and, ultimately, conversion. However, the results from this current study indicate that the suggested predictive utility of SC for subsequent clinical and functional outcomes is likely a byproduct of the threshold criteria for confirmation CHR syndromes and psychosis diagnoses. More specifically, results do not provide support for the use of SC to enhance prediction of psychosis in CHR groups.

Strengths and Limitations

There are several notable strengths of the present study. First, this is the largest CHR sample to date. As a result, there was sufficient statistical power to detect small effects. The literature on SC in SSDs, let alone CHR groups, has been limited by small sample sizes, which have likely contributed to the inconsistency of the findings. However, the present study provides ample power to reliably detect group differences and examine the relatedness of multiple SC domains to both clinical symptoms and functional outcomes. In addition, this investigation has enough power to detect the influence of several key sample characteristics (e.g., age, sex, education, general cognitive functioning/IQ) on those relationships.

Furthermore, the current sample only includes the first half of the 24-month time period measured in the larger NAPLS II study. As a result, findings can be replicated with 24-month clinical and functioning outcomes once the data becomes available. In doing so, future research can build upon the results of this study by determining if disease burden impacts SC performance and the relatedness of performance to clinical symptoms, functional outcomes and, ultimately conversion to psychosis. Such analyses can help

clarify whether SC deficits have predictive utility for future clinical symptoms or functional decline, or whether they might be yet another symptom of the underlying disease progression that prompts help-seeking behaviors in individuals who are deemed at CHR for psychosis.

Nonetheless, because significant findings were only obtained in the cross-sectional baseline portion of the current study, the present study provides no support for the predictive utility of SC performance for clinical and/or functional outcomes of interest. Thus, there is no evidence that the inclusion of SC measures in the assessment or designation of CHR status would enhance the prediction of subsequent symptom severity or functional course. Once the study is completed and conversion data becomes available, findings from the present study can be expanded upon through comparisons of the relations of SC performance with clinical symptom domains and functional outcomes across converters and nonconverters. Results from such analyses would have the potential to clarify whether/not SC performance has any predictive utility for psychosis conversion in CHR groups.

In fact, given that SC deficits (i.e., TASIT and ER40 tasks) were associated with disorganized symptoms at baseline, it is possible that SC may be a predictor of the generalized psychopathology observed in individuals who present for help at CHR clinics. As predictive algorithms are fine-tuned to enhance prediction of conversion to psychosis, it is still of interest to study SC in CHR samples. More specifically, to help differentiate those CHR subjects who remit or remain stable from those who actually are in the prodromal phase of a psychotic disorder. In particular, in studies involving longer

follow-up periods and analyses that involved comparisons of the relationship between SC and symptoms/functioning outcomes according to conversion status.

Conclusions

The present study expanded on previous research by characterizing SC deficits in a CHR sample large enough in size to have the statistical power to examine relatedness of SC (i.e., emotional processing/recognition, ToM, and relationship/social perception) to clinical symptoms (i.e., positive, negative, and disorganized) and functional outcomes (i.e., social and role) at baseline and 12-month follow-up. While previous studies have suggested that SC may be an intermediate vulnerability marker with predictive utility for detecting individuals at GHR for psychosis, the results of this current study indicate that SC is more likely to be considered yet another clinical/outcome domain that prompts help-seeking behaviors in individuals who present at CHR clinics. Future investigations should seek to replicate and extend these findings over the full 24-month follow-up period included in many CHR studies and by comparing the relation of SC performance with clinical symptoms/functioning outcomes and conversion status in large CHR samples.

Table 1

Baseline Demographic and Clinical Characteristics by Diagnostic Group (n=462)

Variable	CHR (n = 294)	Controls (n = 168)
<i>Age, years (mean ± SD)</i>	19.2 ± 4.2	19.7 ± 4.9
<i>Sex, n (%)</i> *		
Males	174 (59.2%)	83 (49.4%)
Females	120 (40.8%)	85 (50.6%)
<i>Race, n (%)</i> ^a		
First Nations	6 (2.0%)	3 (1.8%)
East Asian	6 (2.0%)	8 (4.8%)
Southeast Asian	7 (2.5%)	4 (2.4%)
South Asian	9 (3.1%)	3 (1.8%)
Black	38 (12.9%)	24 (14.3%)
Central/South American	14 (4.8%)	7 (4.2%)
West/Central Asia & Middle East	2 (0.7%)	2 (1.2%)
White	165 (56.1%)	98 (58.3%)
Native Hawaiian/Pacific Islander	3 (1.0%)	0 (0.0%)
Interracial	44 (15.0%)	19 (11.3%)
<i>Ethnicity, n (%)</i>		
Latino/a	60 (20.4%)	31 (18.5%)
Non-Latino/a	234 (79.6%)	137 (81.5%)
<i>Education, years (mean ± SD)</i> **	11.7 ± 2.6	12.6 ± 3.7
<i>Baseline Symptom Severity (mean ± SD)</i>		
SIPS Positive Subscale***	2.4 ± 0.8	0.2 ± 0.3
SIPS Negative Subscale***	2.0 ± 1.1	0.2 ± 0.4
SIPS Disorganized Subscale***	1.3 ± 0.8	0.1 ± 0.3
SIPS General Subscale***	2.3 ± 1.1	0.3 ± 0.6
<i>Baseline Global Functioning (mean ± SD)</i>		
Global Functioning Scale: Role***	6.0 ± 2.2	8.7 ± 1.2
Global Functioning Scale: Social***	6.3 ± 1.7	8.9 ± 0.9
<i>Baseline General Cognition/IQ (mean ± SD)</i> ***	104.3 ± 15.6	112.2 ± 13.8
<i>Baseline Social Cognition (mean ± SD)</i>		
Relationships Across Domains (RAD)***	31.9 ± 5.0	34.0 ± 4.6
The Awareness of Social Inference Task (TASIT)***	52.6 ± 6.1	54.9 ± 5.0
Penn Emotion Recognition Task (ER40)**	32.7 ± 3.5	33.6 ± 2.6

^a Likelihood ratio was used because seven cells have an observed count of less than five.

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

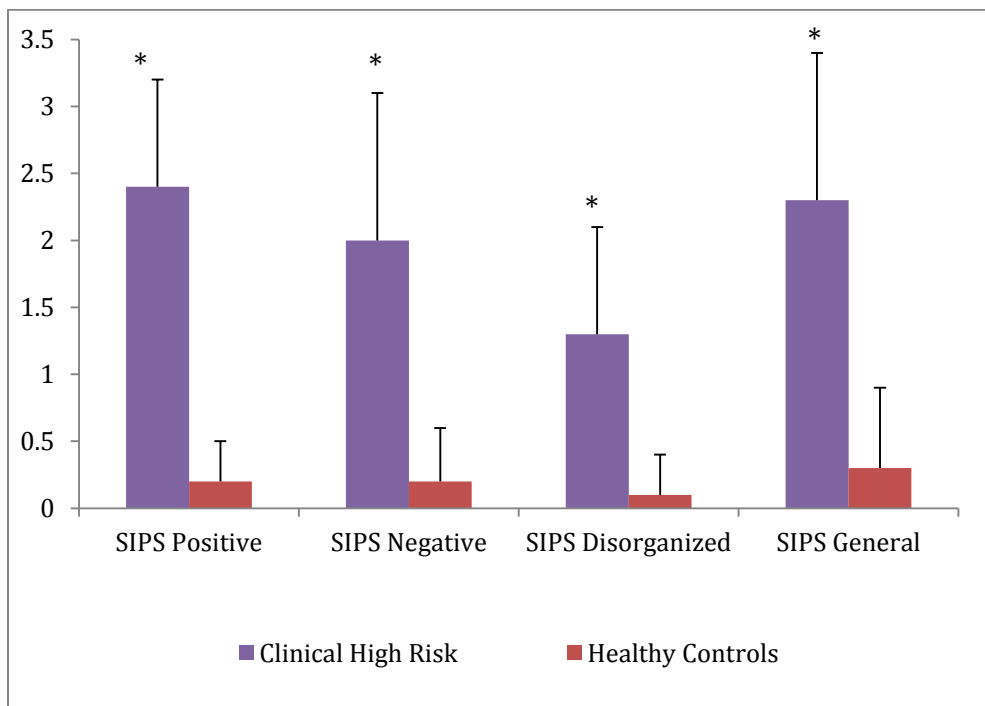


Figure 1. Mean Clinical Symptom Scores by Diagnostic Group

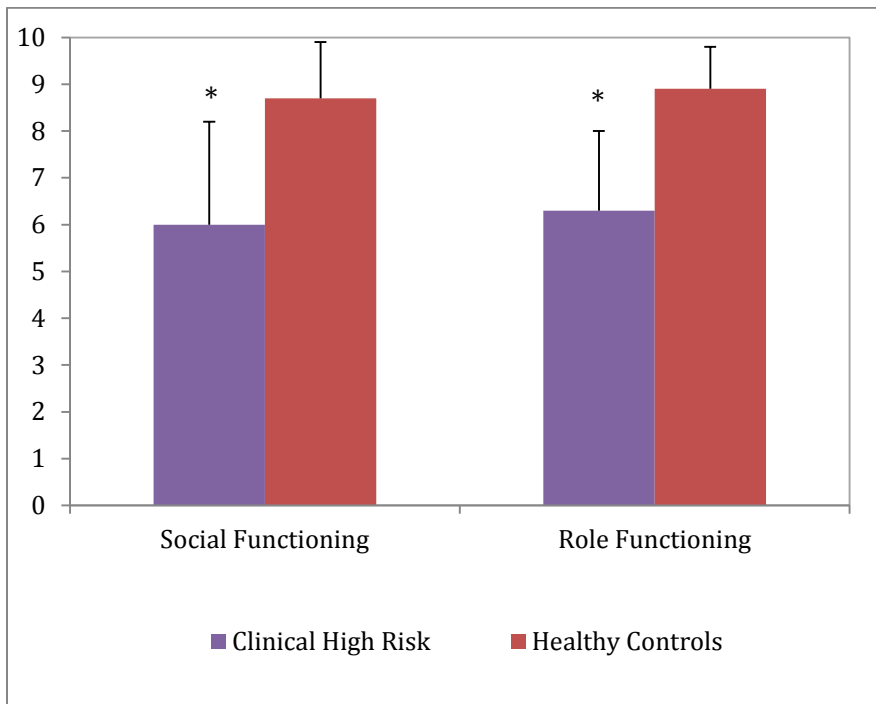


Figure 2. Mean Global and Social Role Functioning Scores by Diagnostic Group

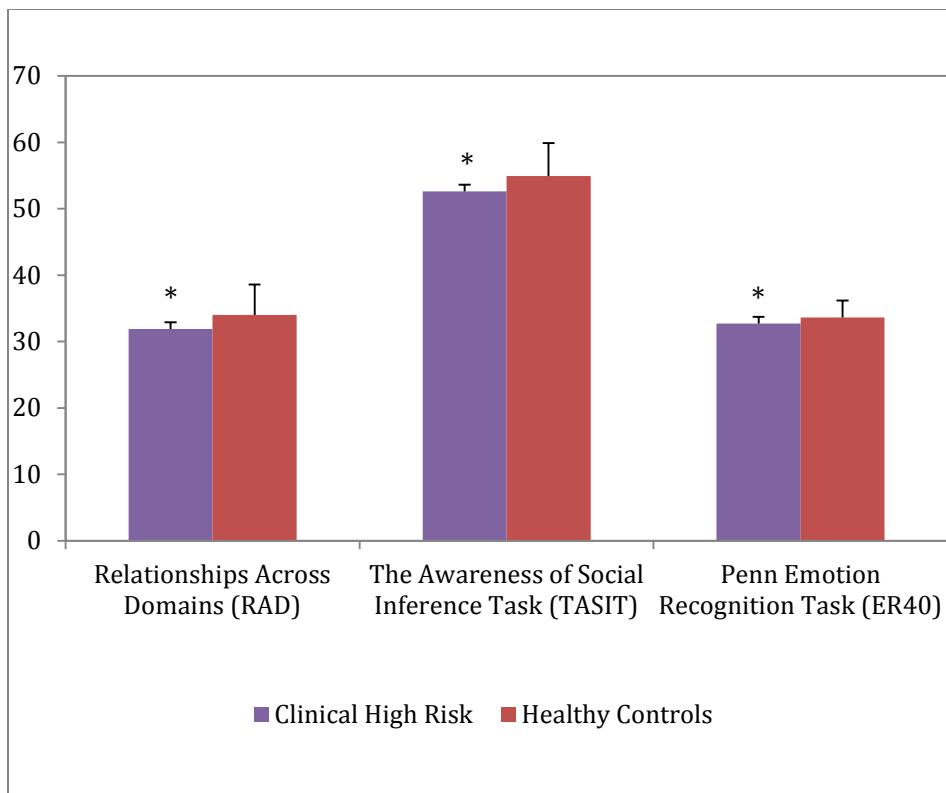


Figure 3. Mean Social Cognitive Domain Scores by Diagnostic Group

Table 2

Regression Analyses: Influence of Diagnostic Group on the Relatedness of Age to Social Cognitive Performance (n=462)

Dependent Variable	RAD		TASIT		ER40	
	ΔR^2	β	ΔR^2	β	ΔR^2	β
Step 1	0.079***		0.065***		0.017*	
<i>Age and Diagnostic Group</i>						
Step 2	0.000		0.011*		0.022**	
<i>Age x Diagnostic Group</i>		-0.036		-.356*		-.493**
Total R^2	0.079		0.076		0.039	

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 3

Intercorrelations: Predictor and Dependent Variables of Interest in the CHR Group (n=294)

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
<i>Demographics:</i>															
1. Age (years)	-														
2. Education (years)	.87**	-													
3. IQ	.23**	.31**	-												
<i>Baseline Social Cognition:</i>															
4. RAD	.24**	.31**	.56**	-											
5. TASIT	.11	.18**	.40**	.47**	-										
6. ER40	-.10	-.15	-.00	.15*	.22**	-									
<i>Baseline DVs:</i>															
7. SIPS Positive	.05	.07	.05	.04	.06	-.03	-								
8. SIPS Negative	.06	-.04	.00	.00	-.04	-.08	.16**	-							
9. SIPS Disorganized	.05	-.01	.17**	.09	.03	-.19**	.39**	.44**	-						
1. Role Functioning	.02	.13*	.20**	.25**	.24**	.16**	-.12*	-.54**	-.25**	-					
11. Social Functioning	-.13*	-.06	.72	.29	.10	.13*	-.59	-.54**	-.27**	.42**	-				
<i>12-month DVs:</i>															
12. SIPS Positive	.10	.04	.06	.01	-.01	-.08	.48**	.20**	.29**	-.13	-.13	-			
13. SIPS Negative	.09	.04	-.02	-.09	-.12	-.05	.21**	.46**	.20**	-.32**	-.39**	.40**	-		
14. SIPS Disorganized	-.04	-.08	.14*	.06	.04	-.13	.29**	.28**	.51**	-.18**	-.27**	.46**	.48**	-	
15. Role Functioning	.03	.13	.13	.23**	.20**	.19**	-.16*	-.30**	-.11	.56**	.36**	-.21**	-.52**	-.24*	-
16. Social Functioning	-.14*	-.11	.02	.01	.14	.15*	-.19**	-.44**	-.21**	.30**	.59**	-.25**	-.64**	-.29**	.42**

* Correlations significant at the $p < 0.05$ level

** Correlations significant at the $p < 0.01$ level

Table 4

Baseline Social Cognitive Performance by Sex within the CHR Group (n=294)

Variable	Mean \pm SD	Gender	<i>n</i>	<i>t</i>	<i>p</i>	<i>d</i>
Relationships Across Domains*	31.3 \pm 5.3	Male	174	2.64	0.009	0.31
	32.9 \pm 4.6	Female	120			
The Awareness of Social Inference Task*	51.6 \pm 6.2	Male	174	3.43	0.001	0.40
	54.0 \pm 5.7	Female	120			
Penn Emotion Recognition Task	32.6 \pm 3.4	Male	174	0.92	0.359	0.11
	32.9 \pm 3.7	Female	120			

Table 5

Regression Analyses: Relatedness of Baseline Social Cognitive Tasks to Baseline Clinical Symptoms and Functioning in the CHR Group (n=294)

Dependent Variable	SIPS Positive		SIPS Negative		SIPS Disorganized		Role Functioning		Social Functioning	
	ΔR^2	β	ΔR^2	β	ΔR^2	β	ΔR^2	β	ΔR^2	β
Relationships Across Domains (RAD)										
Step 1 <i>CVs^a</i>	0.005		0.017		0.030		0.063**		0.034*	
Step 2 <i>RAD</i>	0.000		0.001		0.001		0.011		0.002	
		0.018		0.036		-0.042		0.131		-0.061
Step 3 <i>RAD x Sex</i>	0.005		0.000		0.000		0.001		0.001	
		-0.119		0.022		0.016		-0.042		-0.052
Total R^2	0.010		0.018		0.031		0.074		0.037	
The Awareness of Social Inference Task (TASIT)										
Step 1 <i>CVs^a</i>	0.005		0.017		0.030		0.063**		0.034*	
Step 2 <i>TASIT</i>	0.000		0.004		0.017*		0.009		0.005	
		-0.012		-0.068		-0.151*		0.111		0.083
Step 3 <i>TASIT x Sex</i>	0.009		0.002		0.006		0.000		0.004	
		-0.157		0.072		0.131		0.001		-0.106
Total R^2	0.014		0.023		0.053		0.073		0.043	
Penn Emotion Recognition Task (ER40)										
Step 1 <i>CVs^a</i>	0.005		0.017		0.030 [†]		0.063**		0.034*	
Step 2 <i>ER40</i>	0.012 [†]		0.003		0.036**		0.013*		0.016*	
		-0.109		-0.057		-0.193**		0.117*		0.127*
Step 3 <i>ER40 x Sex</i>	0.001		0.000		0.001		0.000		0.000	
		-0.043		-0.019		0.034		0.011		0.028
Total R^2	0.018		0.020		0.066		0.077		0.050	

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

^a CVs include: age, years of education, and general cognitive functioning/IQ

Table 6

Regression Analyses: Predictive Utility of Baseline Social Cognitive Tasks for 12-month Clinical Symptoms and Functioning in the CHR Group (n=294)

Dependent Variable	SIPS Positive		SIPS Negative		SIPS Disorganized		Role Functioning		Social Functioning	
	ΔR^2	β	ΔR^2	β	ΔR^2	β	ΔR^2	β	ΔR^2	β
Relationships Across Domains (RAD)										
Step 1 <i>CVs^a</i>	0.269***		0.265***		0.301***		0.299***		0.344***	
Step 2 <i>RAD</i>	0.001	-0.036	0.003	-0.070	0.004	-0.081	0.008	0.115	0.001	0.038
Step 3 <i>RAD x Sex</i>	0.000	0.022	0.000	-0.002	0.005	0.119	0.000	-0.018	0.007	0.144
Total R^2	0.270		0.268		0.311		0.307		0.352	
The Awareness of Social Inference Task (TASIT)										
Step 1 <i>CVs^a</i>	0.269***		0.265***		0.301***		0.299***		0.344***	
Step 2 <i>TASIT</i>	0.002	-0.047	0.004	-0.075	0.001	-0.037	0.000	0.007	0.006	0.092
Step 3 <i>TASIT x Sex</i>	0.000	0.005	0.003	-0.081	0.000	0.028	0.002	0.066	0.010	0.157
Total R^2	0.271		0.272		0.303		0.301		0.360	
Penn Emotion Recognition Task (ER40)										
Step 1 <i>CVs^a</i>	0.269***		0.265***		0.301***		0.299***		0.344***	
Step 2 <i>ER40</i>	0.003	-0.057	0.001	-0.037	0.002	-0.050	0.015	0.126	0.001	0.023
Step 3 <i>ER40 x Sex</i>	0.004	-0.085	0.001	-0.039	0.000	0.011	0.003	-0.076	0.004	-0.087
Total R^2	0.276		0.267		0.304		0.317		0.348	

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

^a CVs: age, years of education, general cognitive functioning/IQ, and baseline clinical symptoms/functioning variables (i.e., SIPS Positive, SIPS Negative, SIPS Disorganized, Role Functioning, and Social Functioning scales).

References

- Abe, N., Suzuki, M., Mori, E., Itoh, M., & Fujii, T. (2007). Deceiving others: distinct neural responses of the prefrontal cortex and amygdala in simple fabrication and deception with social interactions. *Journal of Cognitive Neuroscience, 19*, 287–295. doi:10.1162/jocn.2007.19.2.287.
- Adolphs, R., (2001). The neurobiology of social cognition. *Current Opinion in Neurobiology, 11*, 231–239 doi: S0959-4388(00)00202-6
- Addington, J., Cadenhead, K.S., Cornblatt, B.A., Mathalon, D.H., McGlashan, T.H., ... & Cannon, T.D. (2012). North American Prodrome Longitudinal Study (NAPLS 2): overview and recruitment. *Schizophrenia Research, 142*, 77-82. doi: 10.1016/j.schres. 2012.09.012
- Addington, J., & Heinssen, R. (2012). Prediction and prevention of psychosis in youth at clinical high risk. *Annual Reviews in Clinical Psychology, 8*, 269-89. doi:10.1146/annurev-clinpsy-032511-143146
- Addington, J., Penn, D., Woods, S.W., Addington, D., & Perkins, D.O. (2008). Social functioning in individuals at clinical high risk for psychosis. *Schizophrenia Research, 99*, 119–124. doi:10.1016/j.schres.2007.10.001
- American Psychiatric Association. (2000). *Diagnostic and Statistical Manual of Psychiatric Disorders*. 4th ed. Washington, DC: American Psychiatric Press.
- Baron-Cohen, S., Ring, H.A., Bullmore, E.T., Wheelwright, S., Ashwin, C., & Williams, S.C.R. (2000). The amygdala theory of autism. *Neuroscience and Behavioral Reviews, 24*, 355–364. doi:10.1016/S0149-7634(00)00011-7

- Baron-Cohen, S., Wheelwright, S., Hill, J., Raste, Y., & Plumb, I. (2001). The “Reading the mind in the eyes” test revised version: a study with normal adults , and adults with Asperger syndrome or high functioning autism. *Journal of child Psychology and Psychiatry*, *42*, 241–251. doi: 10.1111/1469-7610.00715
- Bellack, A.S., Morrison, R.L., Wixted, J.T., & Mueser, K.T. (1990). An analysis of social competence in schizophrenia. *British Journal of Psychiatry*, *156*, 809–818. doi:10.1192/bjp.156.6.809
- Bentall, R.P. (2001). Social cognition and delusional believes. In P.W. Corrigan & D.L. Penn (Eds.), *Social Cognition in Schizophrenia* (pp.123–148). Washington, D.C.: American Psychological Association.
- Bigelow, N.O., Paradiso, S., Adolphs, R., Moser, D.J., Arndt, S., ... & Andreasen, N.C. (2006). Perception of socially relevant information in schizophrenia. *Schizophrenia Research*, *83*, 257-267. doi:10.1016/ j.schres.2005.12.856
- Blakemore, S.J., & Choudhury, S. (2006). Development of the adolescent brain: implications for executive function and social cognition. *Journal of Child Psychology and Psychiatry*, *47*, 296–312. doi:10.1111/j.1469-7610.2006.01611.x
- Bora, E., Yucel, M. & Pantelis, C. (2009). Theory of mind impairment in schizophrenia: meta-analysis. *Schizophrenia Research*, *109*, 1–9. doi:10.1016/j.schres.2008.12.020
- Brothers L. (1990). The neural basis of primate social communication. *Motivation and Emotion*, *14*, 81–91. doi:10.1007/BF00991637
- Butler, P.D., Abeles, I.Y., Weiskopf, N.G., Tambini, A., Jalbrzikowski, M., ... & Javitt, D.C. (2009). Sensory contributions to impaired emotion processing in

schizophrenia. *Schizophrenia Bulletin*, 35, 1095–1107. doi:
10.1093/schbul/sbp109

Carter, C.S., Barch, D.M., Gur, R., Pinkham, A., & Ochsner, K. (2009). CNTRICS final task selection: social cognitive and affective neuroscience-based measures. *Schizophrenia Bulletin*, 35, 153-162. doi: 10.1093/schbul/sbn157

Cornblatt, B.A., Auther, A.M., Niendam, T., Smith, C.W. Zinberg, J., ... & Cannon, T.D. (2007). Preliminary findings for two new measures of social and role functioning in the prodromal phase of schizophrenia. *Schizophrenia Bulletin*, 33, 688-702. doi:10.1093/ schbul/sbm029

Cornblatt, B.A., Lencz, T., Smith, C.W., Correll, C.U., Auther, A.M., & Nakayama, E. (2003). The Schizophrenia prodrome revisited: A neurodevelopmental perspective. *Schizophrenia Bulletin*, 29 (4), 633–651.

Couture, S.M., Penn, D.L., Woods, S.W., Addington, J., & Perkins, D.O. (2008). Assessment of social judgments and complex mental states in the early phases of psychosis. *Schizophrenia Research*, 100, 237–241.
doi:10.1016/j.schres.2007.12.484

Drake, R.J., Dunn, G., Tarrier, N., Bentall, R.P., Haddock, G., & Lewis, S.W. (2007). Insight as a predictor of the outcome of first-episode nonaffective psychosis in a prospective cohort study in England. *Journal of Clinical Psychiatry*, 68 (1), 81–86.

Drevits, W.C., & Raichle, M.E. (1998). Reciprocal suppression of regional cerebral blood flow during emotional versus higher cognitive processes: implications for

- interactions between emotion and cognition. *Cognition and Emotion*, *12*, 353–385. doi:10.1080/026999398379646
- Dunbar, R.I. (2009). The social brain hypothesis and its implications for social evolution. *Annals of Human Biology*, *36*, 562–572. doi:10.1080/03014460902960289
- Edwards, J., Jackson, H.J., & Pattison, P.E. (2002). Emotion recognition via facial expression and affective prosody in schizophrenia: a methodological review. *Clinical Psychology Review*, *22*, 789–832. doi:10.1016/S0272-7358(02)00130-7
- Erwin, R.J., Gur, R.C., Gur, R.E., et al. (1992). Facial emotion discrimination I. Task construction and behavioral findings in normal subjects. *Psychiatry Research*, *42*, 231-40. doi:10.1016/0165-1781(92)90115-J
- Fanning, J.R., Bell, M.D., & Fiszdon, J.M. (2012). Is it possible to have impaired neurocognition but good social cognition in schizophrenia. *Schizophrenia Research*, *135*, 68-71. doi: 10.1016/j.schres.2011.12.009.
- Fett, A.K., Viechtbauer, W., Dominguez, M.D., Penn, D.L., van Os, J., & Krabbendam, L. (2011a). The relationship between neurocognition and social cognition with functional outcomes in schizophrenia: A metaanalysis. *Neuroscience and Biobehavior Reviews*, *35*, 573-588. doi: 10.1093/schbul/sbr058.
- Fett, A.K., Maat, A., & GROUP Investigators. Social cognitive impairments and psychotic symptoms: what is the nature of their association? *Schizophrenia Bulletin*, *39*, 77-85. doi: 10.1093/schbul/sbr058
- First, M., Spitzer, R.L., Gibbon, M., & Williams, J.B. (2002). *Structured Clinical Interview for DSM-IV Axis I Disorders, REsearch Version, Patient Edition*

(*SCID-I/P*). Biometrics Research. New York: New York State Psychiatric Institute.

Fiske, S.T., & Taylor, S.E. (1991). *Social Cognition* (2nd ed.). New York, NY: McGraw-Hill Book Company.

Fiszdon, J.M., Fanning, J.R., Johannesen, J.K., & Bell, M.D. (2013). Social cognitive deficits in schizophrenia and their relationship to clinical and functional status. *Psychiatry Research*, *205*, 25-29. doi: 10.1016/j.psychres.2012.08.041

Garety, P.A., & Freeman, D. (1999). Cognitive approaches to delusions: a critical review of theories and evidence. *British Journal of Psychology*, *38*, 113–154. doi: 10.1348/014466599162700

Goldman, H.H., Skodak, A.E., & Lave, T.R. (1992). Revising axis V for DSM-IV: a review of measures of social functioning. *American Journal of Psychiatry*, *149* (9), 1148–1156

Goulding, S.M., Holtzman, C.W., Trotman, H.T., Ryan, A.T., MacDonald, A.N., ... & Walker, E.F. (2013). The Prodrome and Clinical Risk for Psychotic Disorders. *Child and Adolescent Psychiatric Clinics of North America*, *22*, 557–567. doi:10.1016/j.chc.2013.04.002

Gòrna, K., Jaracz, K., Rybakowski, F., & Rybakowski, J. (2008). Determinants of objective and subjective quality of life in first-time-admission schizophrenic patients in Poland: a longitudinal study. *Quality of Life Research*, *17*, 237–247. doi:10.1007/s11136-007-9296-z

- Grant, C., Addington, J., Addington, D., & Konnert, C. (2001). Social functioning in first- and multiepisode schizophrenia. *Canadian Journal of Psychiatry, 46*(8), 746–749.
- Green, M.F. (1996). What are the functional consequences of neurocognitive deficits in schizophrenia? *American Journal of Psychiatry, 153* (3), 321–330.
- Green, M.F., Kern, R.S., Braff, D.L., & Mintz, J. (2000). Neurocognitive deficits and functional outcome in schizophrenia: are we measuring the “right stuff”? *Schizophrenia Bulletin, 26* (1), 119–136.
- Green M.F., Kern, R.S., & Heaton, R.K. (2004). Longitudinal studies of cognition and functional outcome in schizophrenia: implications for MATRICS. *Schizophrenia Research, 72*, 41–51. doi: 10.1016/j.schres.2004.09.009
- Green, M.F., Nuechterlein, K.H., Kern, R.S., Baade, L.E., Fenton, W.S., ... & Marder, S.R. (2008b). Functional co-primary measures for clinical trials in schizophrenia: results from the MATRICS psychometric and standardization study. *American Journal of Psychiatry, 165*, 221–228. doi: 10.1176/appi.ajp.2007.07010089
- Green, M.F., Olivier, B., Crawley, J.N., Penn, D.L., & Silverstein, S. (2005). Social Cognition in schizophrenia: Recommendations from the MATRICS New Approaches Conference. *Schizophrenia, Bulletin, 31*, 882–887. doi: 10.1093/schbul/sbi049
- Green, M.F., Penn, D.L., Bentall, R., Carpenter, W.T., Gaebel, W., Gur, R.C., ... Heinsen, R. (2008a). Social cognition in schizophrenia: an NIMH workshop on

definitions, assessment, and research opportunities. *Schizophrenia Bulletin*, *34*, 1211–1220. doi:10.1093/schbul/sbm145

Green, M.F., Bearden, C.E., Cannon, T.D., Fiske, A.P., Helleman, G.S., Horan, W.P., ... & Nuechterlein, K.H. (2012). Social Cognition in Schizophrenia, Part 1: Performance Across Phase of Illness. *Schizophrenia Bulletin*, *38*, 854-64. doi:10.1093/schbul/sbq171

Hamm, J. A., Renard, S. B., Fogley, R. L., Leonhardt, B. L., Dimaggio, G., ... & Lysaker, P. H. (2012). Metacognition and Social Cognition in Schizophrenia: Stability and Relationship to Concurrent and Prospective Symptom Assessments. *Journal of Clinical Psychology*, *68*, 1303-1312. doi: 10.1002/jclp.21906

Heydebrand, G., Weiser, M., Rabinowitz, J., Hoff, A.L., DeLisi, L.E., & Csernansky, J.G., (2004). Correlates of cognitive deficits in first episode schizophrenia. *Schizophrenia Research*, *162*, 1–9. doi:10.1016/S0920-9964(03)00097-5

Horan, W.P., Green, M.F., de Groot, M., Fiske, A., Helleman, G., Kee, K., ... & Nuechterlein, K.H. (2012). Social cognition in schizophrenia, Part 2: 12 month prediction of outcome in first-episode patients. *Schizophrenia Bulletin*, *38*, 865-72. doi: 10.1093/ schbul/sbr001

Jalbrzikowski, M., Carter, C., Senturk, D., Chow, C., Hopkins, J. M., Green, M. F., ... & Bearden, C. E. (2012). Social cognition in 22q11.2 microdeletion syndrome: relevance to psychosis? *Schizophrenia Research*, *142*, 99-107. doi: 10.1016/j.schres.2012.10.007.

- Kim, H.S., Shin, N.Y., Jang, J.H., Kim, E., Shim, G., & Park, H.Y. (2011). Social cognition and neurocognition as predictors to psychosis in individuals at ultra-high risk. *Schizophrenia Research, 130*, 170-175. doi: 10.1016/j.schres.2011.04.023.
- Kinderman, P., & Bentall, R.P. (1996). A new measure of causal locus: the internal, personal, and situational attributions questionnaire. *Personality and Individual Differences, 20*, 261–264. doi: 10.1016/0191-8869(95)00186-7
- Klosterkötter, J., Hellmich, M., Steinmeyer, E.M., & Schultze-Lutter, F. (2001). Diagnosing schizophrenia in the initial prodromal phase. *Archives of General Psychiatry, 58*, 158-64.
- Kohler, C.G., Bilker, W., Hagendoorn, M., et al. (2000). Emotion recognition deficit in schizophrenia: association with symptomatology and cognition and cognition. *Biological Psychiatry, 48*, 127-136. doi: 10.1016/S0006-3223(00)00847-7
- Kolb, B., Wilson, B., & Taylor, L. (1992). Developmental changes in the recognition and comprehension of facial expression: implications for frontal lobe function. *Brain and Cognition, 20*, 74-84. doi: 10.1016/0278-2626(92)90062-Q,
- Kohler, C.G., Bilker, W., Hagendoorn, M., Gur, R.E., & Gur, R.C. (2000). Emotion recognition deficit in schizophrenia: association with symptomatology and cognition. *Biological Psychiatry, 48*, 127–136. doi: 10.1016/S0006-3223(00)00847-7
- Kohler, C.G., Walker, J.B., Martin, E.A., Healey, K.M., & Moberg, P.J. (2010). Facial emotion perception in schizophrenia: a meta-analytic review. *Schizophrenia Bulletin, 36*, 1009–1019. doi: 10.1093/schbul/sbn192

- Krstev, H., Jackson, H., & Maude, D. (1999). An investigation of attributional style in first-episode psychosis. *British Journal of Clinical Psychology, 38*, 181-194. doi: 10.1348/ 014466599162737
- Kurtz, MM., Moberg, P.J., Gur, R.C., & Gur, R.E., (2001). Approaches to cognitive remediation of neuropsychological deficits in schizophrenia: a review and meta-analysis. *Neuropsychology Review 11*, (4) 197–210.
- MacDonald, E.M., Jackson, H.J., Hayes, R.L., Baglioni, A.J., & Madden, C. (1998). Social skills as a determinant of social networks and perceived social support in schizophrenia. *Schizophrenia Research, 1998*, 29:275–286. doi:10.1016/S0920-9964(97)00096-0
- Mancuso, F., Horan, H.P., Kerns, R.S., & Green, M.F. (2011). Social cognition in psychosis: Multidimensional structure, clinical correlates, and relationship with functional outcome. *Schizophrenia Research, 125*, 143-51. doi: 10.1016/j.schres.2010.11.007
- Mayer, J.D., Saovey, P., Caruso, D.R., & Sitarenios, G. (2001). Emotional intelligence as a standard intelligence. *Emotion, 1*, 232–242. doi: 10.1037/1528-3542.1.3.232
- McDonald, S., Flanagan, S., & Rollins, J. (2002). *The Awareness of Social Inference Test*. Suffolk, U.K.: Thames Valley Test Company.
- McDonald, S., Bornhofen, C., Shum, D., Long, E., Saunders, C., & Neulinger, K. (2006). Reliability and validity of The Awareness of Social Inference Test (TASIT): a clinical test of social perception. *Disability and Rehabilitation, 28*, 1529-1542. doi:10.1080/ 09638280600646185

- McGlashan, T., Walsh, B.C. & Woods, S.W. (2010). *The Psychosis Risk Syndrome: Handbook for Diagnosis and Follow-up*. Oxford University Press, New York: New York.
- Miller, T.J., McGlashan, T.H., Woods, S.W., Stein, K., Driesen, N., ... & Davidson, L. (1999). Symptoms assessment in schizophrenic prodromal states. *Psychiatric Quarterly*, 70 (4), 273–287.
- Miller, T.J, McGlashan, T.H., Rosen, J.L., Cadenhead, K., Cannon T., ...& Woods, S.W. (2003). Prodromal assessment with the structured interview for prodromal syndromes and the scale of prodromal symptoms: predictive validity, interrater reliability, and training to reliability. *Schizophrenia Bulletin*, 29 (4), 703-715.
- Miller, T.J., McGlashan, T.H., Rosen, J.L., Somjee, L., Markovich, P.J., ... & Woods, S.W. (2002). Prospective diagnosis of the initial prodrome for schizophrenia based on the Structured Interview for Prodromal Syndromes: Preliminary evidence of interrater reliability and predictive validity. *American Journal of Psychiatry*, 159, 863–865. doi: 10.1176/appi.ajp.159.5.863
- Morton, J.B., & Trehub, S.E. (2001). Children’s understanding of emotion in speech. *Child Development*, 72, 834–843. doi: 10.1111/1467-8624.00318
- Murray, C.J.L., & Lopez, A.D. (1996). *The global burden of disease: A comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020*. Boston: Harvard University Press.
- Neuchterlein, K.H., Green, M.F., Kern, R.S., et al. (2008). The MATRICS Consensus Cognitive Battery, Part 1: Test Selection, Reliability, and Validity. *American Journal of Psychiatry*, 165, 203-13. doi: 10.1176/appi.ajp.2007.07010042

- Onishi, K.H., Baillargeon, R., & Leslie, A.M. (2007). 15-month-old infants detect violations in pretend scenarios. *Acta Psychologica (Amsterdam)*, *124*, 106–128. doi: 10.1016/j.actpsy. 2006.09.009
- Ostrom, T.M. (1984). The sovereignty of social cognition. In: Wyer, R.S. & Srull, T.K. (Eds.), *Handbook of social cognition, Volume 1*. Lawrence Erlbaum Associates, Hillsdale, N.J., pp. 1-38.
- Penn, D.L., Addington, J., & Pinkham, A. (2006). Social cognitive impairments. In J.A. Lieberman, T.S. Stroup, & D.O. Perkins (Eds.) *American psychiatric association textbook of schizophrenia* (pp. 261–274). Arlington, VA: American Psychiatric Publishing Press, Inc.
- Penn, D.L., Corrigan, P.W., Bentall, R.P., Racenstein, J.M., & Newman, L. (1997) Social cognition in schizophrenia. *Psychological Bulletin*, *121*, 114–132. doi: 10.1037/0033-2909.121.1.114
- Penn, D.L., Ritchie, M., Francis, J., Combs, D., & Martin, J. (2002). Social perception in schizophrenia: the role of context. *Psychiatry Research*, *109*, 149–159. doi: 10.1016/S0165-1781(02)00004-5
- Pfohl, B., Blum, N., Zimmerman, M. (1995). *Structured Interview for DSM-IV Personality (SID-PV)*. Washington, D.C: American Psychiatric Press.
- Pinkham, A.E., Penn, D.L., Perkins, D.O., Lieberman, J. (2003). Implications for the neural basis of social cognition for the study of schizophrenia. *American Journal of Psychiatry*, *160*, 815-824. doi: 10.1176/appi.ajp.160.5.815
- Pinkham, A.E., Penn, D.L., Perkins, D.O., Graham, K.A., & Siegel, M. (2007). Emotion perception and social skill over the course of psychosis: a comparison of

individuals

“at-risk” for psychosis and individuals with early and chronic schizophrenia spectrum illnesses. *Cognitive Neuropsychiatry*, *12*, 198–212. doi:

10.1080/13546800600985557

Platek, S.M., Loughhead, J.W., Gur, R.C., Busch, S., Ruparel, K., ... & Langleben, D.D.

(2006). Neural substrates for functionally discriminating self-face from personally familiar faces. *Human Brain Mapping*, *27*, 91–98. doi:10.1002/hbm.20168.

PsychCorp (1999). Wechsler Abbreviated Scale of Intelligence. San Antonio: Harcourt Assessment, Inc.

Riecher-Rössler, A., Aston, J., Ventura, J., Merlo, M., Borgwardt, S., ... & Stieglitz, R.D.

(2008). The Basel Screening Instrument for Psychosis: development, structure, reliability and validity. *Fortschritte der Neurologie Psychiatrie*, *76*, 207-16. doi:

10.1055/s-2008-1038155.

Rojahn, J., Gerhards, F., Matlock, S.T., & Kroeger, T.L. (2000). Reliability and validity

studies of the Facial Discrimination Task for emotion research. *Psychiatry Research*, *95* (2), 169-181.

Salem, J.E., Kring, A.M., & Kerr, S.L. (1996). More evidence for generalized poor

performance in facial emotion perception in schizophrenia. *Journal of Abnormal Psychology*, *105*, 480–483. 10.1037/0021-843X.105.3.480

Savla, G. N., Vella, L., Armstrong, C. C., Penn, D. L., & Twamley, E. W. (2012).

Deficits in Domains of Social Cognition in Schizophrenia: A Meta-Analysis of the Empirical Evidence. *Schizophrenia Bulletin* (epub ahead of print). doi:

10.1093/schbul/sbs080

- Schmidt, S. J., Mueller, D. R., & Roder, V. (2011). Social cognition as a mediator variable between neurocognition and functional outcome in schizophrenia: Empirical review and new results by structural equation modeling. *Schizophrenia Bulletin*, *37*, 41-54. doi: 10.1093/schbul/sbr079.
- Sergi, M.J., Fiske, A.P., Horan, W.P., Kern, R.S., Kee, K.S., ... & Green MF. (2009). Development of a measure of relationship perception in schizophrenia. *Psychiatry Research*, *166*, 54-62. doi: 10.1016/j.psychres.2008.03.010
- Sergi, M.J., & Green, M.F. (2003). Social perception and early visual processing in schizophrenia. *Schizophrenia Research*, *59*, 233–241. doi: 10.1016/S0920-9964(01)00405-4
- Skodol, A.E., Link, B.G., Shrout, P.E., & Horwath, E. (1988). Toward construct validity for DSM-III Axis V. *Psychiatry Research*, *24*, 13-23. doi: 10.1016/0165-1781(88)90135-7
- Sprong, M., Schothorst, P., Vos, E., Hox, J., & van Engeland, H. (2007). Theory of mind in schizophrenia: meta-analysis. *British Journal of Psychiatry*, *191*, 5-13. doi: 10.1192/bjp.bp.107.035899
- Suri, L., Caldi, S., & Sperber, D. (2007). Attribution of beliefs by 13-month-old infants. *Psychological Science*, *18*, 580–586. doi: 10.1.1.173.8130
- Thompson, A.D., Bartholomeusz, C., & Yung, R. (2011). Social cognition deficits in the ‘ultra high risk’ for psychosis population: a review of the literature. *Early Intervention in Psychiatry*, *5*, 192-202. doi:10.1111/j.1751-7893.2011.00275.x

- Tonks, L., Williams, W.H., Frampton, I.J., Yates, P.J., & Slater, A.M. (2007). The neurological bases of emotional dys-regulation arising from brain injury in childhood. *Brain Impairment*, 8, 143–153. doi: 10.1375/brim.8.2.143
- Toomey, R., Schuldberg, D., Corrigan, P.W., & Green, M.F. (2002). Nonverbal social perception and symptomatology in schizophrenia. *Schizophrenia Research*, 53, 83–91. doi: 10.1016/S0920-9964(01)00177-3
- Van Overwalle, F. (2009). Social cognition and the brain: a meta-analysis. *Human Brain Mapping*, 30, 829–858. doi:10.1002/hbm.20547.
- Ventura, J, Wood, R.C., & Helleman, G.S. (2011). Symptom domains and neurocognitive functioning can help differentiate social cognitive processes in schizophrenia: a meta-analysis. *Schizophrenia Bulletin*, 39, 102-11. doi: 10.1093/schbul/sbr067
- Voges, M., & Addington, J. (2005). The association between social anxiety and social functioning in first episode psychosis. *Schizophrenia Research*, 76, 287–292. doi:10.1016/j.schres.2005.01.001
- Yager, J.A., & Ehmann, T.S. (2006). Untangling social function and social cognition: a review of concepts and measurement. *Psychiatry*, 69, 47-67. doi: 10.1521/psyc.2006.69.1.47
- Yung, A.R., Yuen, H.P., McGorry, P.D., Phillips, L.J., Kelly, D., ... & Buckby, J. (2005). Mapping the onset of psychosis: the Comprehensive Assessment of At-Risk Mental States. *Australian New Zealand Journal of Psychiatry*, 39, 964-971. doi: 10.1080/j.1440-1614.2005.01714.x

Zullo, H.M., Oettingen, G., Peterson, C., & Seligman, M.E.P. (1988). Pessimistic explanatory style in the historical record: CA Ving. LBJ, presidential candidates, and East versus West Berlin. *American Psychologist*, *43*, 673-682. doi: 10.1037/0003-066X.43.9.673