Facial Affect Recognition and Social Functioning in Individuals at Risk for Schizophrenia Spectrum Disorders

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Facial Affect Recognition and Social Functioning in Individuals at Risk for
Schizophrenia Spectrum Disorders
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Abstract

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by

Marta Statucka

Advisor: Deborah J. Walder, Ph.D.

Facial affect recognition (FAR) is impaired in schizophrenia patients and to a lesser extent in individuals at familial/genetic, clinical, and psychometric risk for psychosis. Reduced FAR reaction time and negative bias are present in patients; however, their role is less clear in at-risk samples. Impaired social functioning, a hallmark of schizophrenia, is also impaired in at-risk individuals and is associated with FAR impairment. Given FAR deficits in schizophrenia, the current study aimed to elucidate the nature of FAR and social functioning impairments among individuals at high psychometric risk for psychosis and to examine whether FAR acts as a mediator in the relationship between schizotypal traits and social functioning. Nine-hundred and sixty-five (653 Female/312 Male) young adults were recruited from across CUNY campuses and asked to complete self-report measures assessing schizotypal traits (to determine psychometric risk status) and social functioning. Participants were also administered a computerized measure of FAR remotely via the Internet. Individuals at high psychometric risk performed significantly worse on total accuracy and neutral accuracy and were significantly more likely to misattribute negative emotions and sadness to neutral faces when compared to low-risk individuals. However, when adjusting for depressive symptoms, schizotypal traits were no longer associated with negative bias. Aspects of FAR performance were
differentially associated with schizotypal traits in the total, high-risk, and low-risk samples. High-risk individuals reported significantly worse social functioning than individuals at low risk, and FAR accuracy was a partial mediator of the relationship between schizotypal traits and social functioning in the total sample. Results demonstrating attenuated FAR deficits in psychometrically at-risk individuals suggest that FAR may be an important endophenotype of schizophrenia spectrum disorders and has implications for understanding etiology of these disorders. Furthermore, results demonstrating that FAR deficits were related to poor social functioning in at-risk individuals have important clinical implications for improving ways of identifying those at risk and potential treatment strategies.
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LIST OF ABBREVIATIONS

CES-D: Center for Epidemiologic Studies Depression scale
CIS: Chapman Infrequency Scale
CUNY: City University of New York
DSM: Diagnostic and Statistical Manual of Mental Disorders
FAR: facial affect recognition
GAF: Global Assessment of Functioning
KDEF: Karolinska Directed Emotional Faces
MDD: major depressive disorder
OCD: obsessive-compulsive disorder
PANSS: Positive and Negative Syndrome Scale
SANS: Scale for the Assessment of Negative Symptoms
SAPS: Scale for the Assessment of Positive Symptoms
SFS: Social Functioning Scale
SPD: schizotypal personality disorder
SPQ: Schizotypal Personality Questionnaire
SPQ-CP: Schizotypal Personality Questionnaire Cognitive-Perceptual subscale
SPQ-D: Schizotypal Personality Questionnaire Disorganized subscale
SPQ-I: Schizotypal Personality Questionnaire Interpersonal subscale
SSD: schizophrenia spectrum disorder
UHR: ultra-high risk
CHAPTER I

INTRODUCTION

Impairment in expression of emotion through facial expressions and gestures is well documented in the research literature (Borod et al., 1989; Martin, Borod, Alpert, Brozgold, & Welkowitz, 1990; Kring, Kerr, Smith, & Neale, 1993; Earnst et al., 1996; Kring & Neale, 1996; Troisi, Spalletta, & Pasini, 1998; Yecker et al., 1999; Tremeau et al., 2005; Mittal et al., 2006) and is one of the key diagnostic criteria of schizophrenia (American Psychiatry Association, 2000; World Health Organization, 1994). Research conducted in the last three decades demonstrates that schizophrenia patients’ ability to recognize the affective states of others is also compromised (for review see Mandal, Pandey & Prasad, 1998; Edwards, Jackson, Pattison, 2002, Kohler, Walker, Martin, Healey, & Moberg, 2010; Chan, Li, Cheuen, & Gong, 2010). Some authors have speculated that difficulties with expressing emotion and perceiving the emotions of others may lead to social anxiety and the social withdrawal and dysfunction (Penn, Corrigan, Bentall, Racenstein, & Newman, 1997; Green & Phillips, 2004; Bell, Tsang, Greig, & Bryson, 2009) typical of schizophrenia patients.

There is some concern that findings of impaired emotion perception (or recognition) in schizophrenia patients may be confounded by a number of factors including chronicity of illness, chronic use of psychotropic medication, and recurrent hospitalization (Mueser, Penn, Blanchard, & Bellack, 1997; Kee, Kern, Marshall, & Green, 1998; Silver, Shlomo, Turner, Gur, 2002; Kohler et al., 2010). In an attempt to overcome these shortcomings, investigators have begun to conduct studies using methods aimed at identifying individuals believed to be at increased risk for developing
schizophrenia, other forms of psychosis, or related personality disorders (i.e., schizotypal, schizoid, paranoid). This approach is advantageous in that these individuals are typically not yet medicated or impaired to a degree that renders them likely to be hospitalized or homeless, nor do they carry a diagnosis burdened with the same degree of social stigma. As one example, researchers have psychometrically identified healthy (non-clinical) individuals from the general population with elevated schizotypal personality traits that are thought to represent a risk (greater than in the normative population) for development of schizophrenia spectrum disorders (SSDs) (see Lenzenweger, 1994 for overview). Researchers have also identified individuals who are genetically at high risk for developing SSDs by virtue of their familial/genetic relationship to individuals with schizophrenia (e.g., first and second degree relatives of patients). More recently, using a sequential screening approach and clear diagnostic criteria researchers have identified individuals who are at ultra-high risk (UHR) for developing psychotic disorders as defined by a recent functional decline or the presence of subthreshold or “prodromal” psychotic symptoms. Research with these at-risk populations represents an increasing interest in understanding schizophrenia and related psychotic disorders as lying on a continuum instead of as distinct categorical end points. Accordingly, individuals with SSDs and those at-risk for these disorders are believed to share some phenotypic traits, deficits, and etiological risk factors. Given the evidence that patients with schizophrenia have significant difficulties labeling the affective states of others based on facial expressions, the current study aimed to elucidate whether similar but attenuated deficits exist in individuals with elevated schizotypal personality traits and to examine whether
these deficits act as a mediator in the relationship between schizotypal traits and social functioning in this population.

The idea of a continuous phenotypic vulnerability for psychotic symptoms was first proposed by Paul Meehl in his seminal paper “Schizotaxia, Schizotypy, Schizophrenia” (1962). Meehl posited that certain individuals may inherit a genetic mutation – which he termed schizotaxia – that predisposes them to schizophrenia. Accordingly, all schizotaxics eventually develop a schizotypic personality organization through social learning; however, only a minority of schizotypic individuals will experience the proper constellation of environmental influences that will cause them to decompensate into schizophrenia. This is also consistent with the diathesis-stress model of psychopathology (Walker & Diforio, 1997). In support of Meehl’s theory, a number of researchers have reported that symptoms that were once regarded as the hallmarks of psychotic disorders – namely delusions and hallucinations – are commonly reported in the general population (Ohayon, 2000; Johns, Nazroo, Bebbington, & Kuipers, 2002; Johns et al., 2004; Cohen, Magai, Yaffee, & Walcott-Brown, 2004) and are identified in more subtle forms (e.g., magical thinking, unusual perceptual experiences) among at-risk populations.

Several schizophrenia symptoms, neurocognitive deficits, physiological and neurobiological findings have already been demonstrated to exist in an attenuated form in at-risk populations and family members of patients, such as attenuated positive and negative symptoms (Yung & McGorry, 1996; Lencz, Smith, Auther, Correll, & Cornblatt, 2004), verbal memory, working memory, and executive functioning deficits (Lencz et al., 2006; Frommann et al., 2011), reduced P300 amplitude (Lee, Namkoong,
Cho, Song, & An, 2010; van Tricht et al., 2010), saccadic eye movement abnormalities (Thaker et al., 2000; Nieman et al., 2007), hippocampal abnormalities (Seidman et al., 2002), and medial temporal lobe dysfunction (Seidman et al., 2003). This evidence of milder phenotypic expression among at-risk populations renders it is reasonable to hypothesize that milder forms of affect recognition deficits and social dysfunction observed among patient samples will also be present in these at-risk groups. While there is growing evidence that individuals at increased psychometric risk perform less accurately on facial affect recognition (FAR) tasks (van’t Wout, Aleman, Kessels, Laroi, & Kahn, 2004; Williams, Henry, & Green, 2007; Aguirre et al., 2008; Brown & Cohen, 2010; Germine & Hooker, 2011; Roddy et al., 2012; Abbott & Green, 2013), to date, only a few studies have examined reaction time (Green et al., 2001; Brown & Cohen, 2010) or bias – that is, erroneously labeling certain emotions or misattributing emotions to neutral faces – (van’t Wout et al., 2004; Williams et al., 2007; Brown & Cohen, 2010) during FAR in this population. Similarly, researchers have begun to address how FAR accuracy is related to schizotypal traits in psychometrically high-risk individuals (Williams et al., 2007; Brown & Cohen, 2010; Germine & Hooker, 2011; Abbott & Green, 2013). Only one study, however, examined the relationship between FAR reaction time and symptoms (Brown & Cohen, 2010), and only one study examined the relationship between FAR bias and symptoms (van’t Wout et al., 2004). Lastly, research on affect recognition ability and social functioning in individuals at increased risk for psychosis is particularly limited (Jahshan & Sergi, 2007; Aguirre, Sergi, & Levi, 2008; Pelletier et al., 2013). Therefore, to our knowledge, this is the first study to examine FAR accuracy, reaction time, and bias in relation to schizotypal traits and social function.
functioning in any SSD population. More research in this area with at-risk populations is warranted as it helps elucidate findings that are otherwise masked by the effects of chronic treatment, hospitalization, and stigma in studies examining patients. Moreover, this line of investigation holds potential for clarifying the extent to which psychopathology lies along a continuum, with implications for revealing important etiological factors and avenues for early identification, prevention, and treatment for schizophrenia.

**Facial Affect Recognition (FAR)**

**FAR Accuracy in SSD Samples.** Numerous studies have demonstrated FAR deficits among 1) patients with schizophrenia (Walker, Marwit, & Emory, 1980; Borod et al., 1989; Borod, Martin, Alpert, Brozgold, & Welkowitz, 1993; Mueser et al., 1996; Addington & Addington, 1998; Mandal, Jain, Hasque-Nizamie, Weiss, & Schneider, 1999; Kohler, Bilker, Hagendoorn, Gur, & Gur, 2000; Penn et al., 2000; Edwards, Pattison, Jackson, & Wales, 2001; Hooker & Park, 2002; Silver et al., 2002; Kohler et al., 2003; Herbener, Hill, Marvin & Sweeney, 2005; Addington, Saeedi, & Addington, 2006; Bediou et al., 2007; Alfimova et al., 2009; Larøi, Fonteneau, Mourad, & Raballo, 2010; Li, Chan, Zhao, Hong, & Gong, 2010b; Malik, Khawar, Chaudhry, & Humphreys, 2010; Behere, Venkatasubramanian, Arasappa, Reddy, & Gangadhar, 2011b; Gilling McIntosh, 2011; Leung, Lee, & Lee, 2011; Surguladze et al., 2012, Goghari & Sponheim, 2012; for review see Mandal et al., 1998; Edwards et al., 2002, Kohler et al., 2010; Chan et al., 2010), 2) first-degree relatives (Kee, Horan, Mintz, & Green, 2004; Bediou et al., 2007; Leppänen et al., 2008; Alfimova et al., 2009; Calkins et al., 2010; Li et al., 2010b; Eack et al., 2010; Surguladze et al., 2012; for review see Lavoie et al., 2013) and second-
degree relatives (Eack et al., 2010) of schizophrenia patients, 3) individuals with schizotypal personality disorder (SPD; Mikhailova, Vladimirova, Iznak, Tsusulkovskaya, & Sushko, 1996; Waldeck & Miller, 2000), and 4) putatively prodromal individuals who are at high risk for developing a SSD (Pinkham, Penn, Perkins, Graham, & Siegel, 2007b; Addington, Penn, Woods, Addington, & Perkins, 2008a; van Rijn et al., 2011; Addington et al., 2012; Amminger et al., 2012a; Amminger et al., 2012b; Thompson et al., 2012; Wölwer et al., 2012; Comparelli et al., 2013). Consistent with these findings, more recent studies using self-report measures to identify psychometrically at-risk individuals within the general population have demonstrated impaired performance on FAR tasks among individuals with high compared to low schizotypal traits (van’t Wout et al., 2004; Williams et al., 2007; Aguirre et al., 2008; Brown & Cohen, 2010; Germine & Hooker, 2011; Roddy et al., 2012; Abbott & Green, 2013).

It is important, however, to note that not all research has supported a FAR deficit in SSDs. For instance, Bölte & Poustka (2003) reported that neither patients with schizophrenia nor their first-degree relatives demonstrated FAR impairments compared to healthy controls. Furthermore, members from multiplex-families (where two or more children in the family carry a diagnosis of schizophrenia) did not differ from members from simplex families where only one child is diagnosed with schizophrenia (Bölte & Poustka, 2003). Similarly, Seiferth et al. (2008) found that UHR individuals were not less accurate on a FAR task compared to healthy controls. Dickey et al. (2011) found that SPD patients performed less accurately on FAR tasks; however, after adjusting for accuracy on a control gender identification task, the difference in FAR performance was no longer significant. The researchers interpreted this as suggesting that individuals with
SPD have a general deficit in face processing as opposed to a specific impairment in FAR. Similarly, some studies have reported no significant differences in the performance of high and low schizotypy subjects (Poreh, Whitman, Weber, & Ross, 1994; Toomey & Schuldberg, 1995; Green, Williams, & Davidson, 2001; Jahshan & Sergi, 2007; Shean, Bell & Cameron, 2007).

Several methodological factors might account for inconsistencies in FAR findings in the at-risk literature. First, a number of the studies reporting null findings (Toomey & Schuldberg, 1995; Jahshan & Sergi, 2007; Shean et al., 2007; Wickline, Nowicki, Bollini, & Walker, 2012) used FAR tasks that were not previously used with schizophrenia spectrum patients and therefore possibly lacked sensitivity to detect (potentially more) subtle deficits among at-risk samples. In support of this possibility, Shean et al. (2007) and Wickline et al. (2012) did not find FAR deficits in a sample of psychometrically at-risk young adults and a sample of SPD adolescents, respectively, using the Diagnostic Analysis of Nonverbal Accuracy Scale-2 (Nowicki, 2010), whereas other investigators have demonstrated FAR deficits in SPD (Mikhailova et al., 1996; Waldeck & Miller, 2000). Wickline et al. (2012) noted that though the group differences for FAR were not significant, the results were in the predicted direction (i.e., SPD less accurate than adolescents with other personality disorders and healthy controls). The authors attributed this null result to limited statistical power resulting from a small sample size and posited that both facial and auditory affect recognition deficits would predict later conversion to schizophrenia, however, their sample size precluded examination of this relationship. One additional possibility is that the measure used by Wickline and colleagues (2012) had not previously been validated in individuals with SSDs and may have lacked the
sensitivity to detect FAR deficits in this sample. It should be noted, however, that previous research supporting a FAR impairment in SPD used samples that differed demographically from Wickline and colleagues’ adolescent sample (i.e., only male subjects in Mikhailova et al., 1996, mean age of 28 in Waldeck & Miller, 2000) and therefore, the potential confounding effects of sex and age cannot be excluded. Selection of an appropriate measure is an especially important consideration in research with psychometrically at-risk populations given that any FAR deficits in this group are likely to be even more subtle than those of SPD patients. The second methodological factor to consider is that some studies reporting null results only assessed positive (as opposed to negative and disorganized) schizotypal traits in their psychometrically at-risk samples (Toomey & Schuldberg, 1995; Green et al., 2001), and positive symptoms alone are less often found to be related to FAR deficits in patient populations. Finally, in all studies we are aware of that demonstrate a significant difference in FAR accuracy between high and low schizotypy groups, the high-schizotypy group was composed of predominantly female participants (range 62-80%) whereas in three of the studies reporting null results the high-schizotypy groups consisted of 46-57% females (Toomey & Schuldberg, 1995; Green et al., 2001; Jahshan & Sergi, 2007), and Poreh et al. (1994) recruited only male participants. This raises the need to consider the potential role of sex differences in FAR performance.

There is clear evidence of impaired accuracy during FAR in patients with schizophrenia, their relatives, and UHR individuals. What remains less clear is whether patients with SPD or individuals at psychometric risk for SSDs demonstrate similar impairments in FAR accuracy. Studies utilizing FAR tasks that were previously used in
studies of schizophrenia patients and screening individuals using the full-range of schizotypy characteristics (i.e., positive, negative, and disorganized symptoms) typically demonstrated reduced accuracy in identifying facial expressions (van’t Wout et al., 2004; Williams, et al., 2007; Aguirre et al., 2008; Brown & Cohen, 2010; Germine & Hooker, 2011; Roddy et al., 2012; Abbott & Green, 2013) in a manner similar to, but less severe than, findings demonstrated in patients with schizophrenia, their relatives, and UHR individuals.

**Speed during FAR in SSD Samples.** To date, the role of reaction time in FAR deficits is less well studied than performance accuracy. Moreover, it remains unclear whether an accuracy-speed tradeoff exists, given limited analyses of reaction time during FAR tasks. Lack of attention in the literature to reaction time and accuracy-speed tradeoff is partially due to the fact that older FAR tasks did not allow for accurate measurement of reaction time, whereas more recently developed computerized FAR task have allowed researchers to examine reaction time during FAR performance more systematically. Examining whether patients and those at elevated risk for SSDs demonstrate slowed reaction time during FAR is of importance as slower ability to identify emotions could have consequences for everyday social interactions when individuals must identify others’ emotions in real time.

Several studies have found that schizophrenia patients are significantly slower than controls when performing FAR tasks (Gur et al., 2002; Machado de Sousa & Hallak, 2008; Habel et al., 2010; Calkins et al., 2010; Li et al., 2010b), though there are some contradictory findings. For example, when examining differences in FAR performance in actively paranoid versus not actively paranoid schizophrenia patients, Pinkham,
Brensinger, Kohler, Gur, & Gur (2011) found no group differences or group by emotion interaction in reaction time; that is, the groups did not differ in how quickly they recognized happy, sad, angry, fearful, or neutral faces. Some authors reported that when performing facial affect matching tasks (Doop & Park, 2009) and FAR tasks (Fakra, Salgado-Pineda, Delaveau, Hariri, & Blin, 2008), patients with schizophrenia did not differ from healthy controls in terms of reaction time. In contrast, however, Seiferth et al. (2009) found that patients with schizophrenia were significantly faster than controls, though only when labeling sad faces, suggesting emotion specificity with respect to reaction time.

In at-risk populations, the findings are also mixed, though – as in patient populations – the majority of findings suggest slower performance among at-risk groups. Eack et al. (2010) reported that first- and second-degree relatives of patients were slower than controls on a FAR task. Furthermore, slower reaction time for the entire task (i.e., labeling happy, sad, angry, fearful, and neutral faces) and for labeling neutral alone faces was associated with more general prodromal symptoms. Similarly, Calkins et al. (2010) reported that individuals at familial high risk for SSDs were significantly slower than controls on emotion processing tasks (i.e., identification and discrimination tasks). In contrast, Li et al. (2010b) found that siblings of patients did not significantly differ from either patients or controls with regard to reaction time. Moreover, parents of patients did not differ in terms of reaction time compared to an age-matched sample of controls after adjusting for education and IQ. In keeping with these findings, other researchers have found that UHR individuals did not differ from controls in terms of speed (Seiferth et al., 2008). As with their findings for accuracy in FAR, Dickey et al. (2011) found that
individuals with SPD were significantly slower than controls; however, after adjusting for speed on a gender identification task, this difference was no longer significant. This finding lends further support to Dickey et al.’s (2011) conclusion that individuals with SPD have a general deficit in face processing.

Two studies reported on reaction time during FAR performance in psychometrically at-risk individuals. Both Brown & Cohen (2010) and Green et al. (2001) found no significant difference in reaction time between at-risk individuals and controls across different emotions. However, Green et al. (2001) reported a significant group by emotion interaction, such that delusion-prone individuals as identified by elevated scores on the Peters Delusions Inventory (Peters, Day, & Garety, 1996) were significantly slower at identifying angry expressions only. The authors posited that slower processing may represent an attentional bias for threat-related stimuli in these individuals that is similar to biases found in paranoid patients. The authors did not measure negative symptoms of schizotypy; thus, symptom domains specificity could not be deduced. It should also be noted that unlike most FAR tasks used in the field, which utilize a forced-choice or multiple-choice format, these investigators required their participants to name the emotions depicted. This methodological issue could present a confound as there is some evidence of reduced verbal fluency and expression in psychometrically at-risk individuals (Barrantes-Vidal et al., 2003; Cohen & Hong, 2011; Cohen, Morrison, Brown, & Minor 2012). Therefore, a forced choice format may be more apt to detect underlying FAR deficits by reducing the confounding cognitive demands placed on participants when they are asked to independently name the facial emotions depicted. In contrast, Brown & Cohen (2010) did not find a significant group
by emotion interaction but reported that higher negative symptoms as measured by the Schizotypal Personality Questionnaire (SPQ; Raine, 1991) were related to quicker reaction times. These results are surprising given that deficits in speed of processing and motor speed have been found in UHR (Niendam et al., 2007) and familial high-risk (Keshavan et al., 2010) groups, and psychomotor slowing is typically associated with negative symptoms in patient with schizophrenia (see Morrens, Hulstijn, & Sabbe, 2007 for review). However, while some researchers have found evidence of psychomotor slowing in psychometrically at-risk individuals (Lenzenweger, 2001; Asai, Sugimori, & Tanno, 2009), others have reported that high psychometric risk individuals did not differ from those at low risk (see Gooding, Matts, Rollmann 2006). Specifically, Brown & Cohen (2010) attributed the fact that individuals from the general population with more negative symptoms had quicker reaction times to a lack of investment in the FAR task given that these individuals were not less accurate than individuals with fewer negative schizotypal traits; suggesting that these psychometrically at-risk individuals were neither more efficient at the FAR task nor that there was a speed-accuracy tradeoff.

The majority of research to date suggests that patients with schizophrenia and their relatives are slower than healthy controls when asked to label emotional faces. To our knowledge, only two studies have examined reaction time in UHR individuals and both reported that UHR individuals did not differ from controls with regard to reaction time. The research on reaction time during FAR tasks in psychometrically at-risk individuals is limited and inconclusive. It is therefore unclear whether these individuals demonstrate slowing during FAR similar to the deficits demonstrated in patient groups.
FAR Performance in Relation to Psychiatric Symptoms. The relationship between specific symptom domains and performance on FAR tasks has been extensively studied in patients with schizophrenia. Several studies have demonstrated a negative correlation between the severity of overall negative symptoms and FAR accuracy (Alfimova et al., 2009) and with specific negative symptoms and FAR accuracy, including alogia (Gaebel & Wölwer, 1992; Silver & Shlomo, 2001; Kohler et al. 2003; Gilling McIntosh, 2011), anergia (Mueser et al., 1996), and affective flattening, avolition, and anhedonia (Kohler et al., 2003). Similarly, Habel et al. (2010) reported that negative symptoms were positively correlated with the tendency of patients with schizophrenia to label neutral faces as emotional (see below section on Bias).

The relationship between symptomatology and FAR performance may also depend on when patients are assessed in terms of hospitalization status, course of illness, and symptom profile. Addington & Addington (1998) reported that when tested as inpatients, there was no relationship between symptoms and FAR. When the same patients were tested 3 months later as outpatients, there was a direct association between severity of negative symptoms and FAR deficits. Similarly, Herbener et al. (2005) reported that only after stabilization with antipsychotics was there a negative correlation between negative symptoms and performance on FAR tasks in first-episode patients with schizophrenia. In both of these studies, the authors used the Positive and Negative Syndrome Scale (PANSS; Kay, Fiszbein, & Opler, 1987) to assess positive and negative symptoms and despite a significant reduction in symptom severity between baseline and follow-up assessments, both studies failed to find an improvement in FAR. Furthermore, positive symptoms were not related to FAR accuracy either at baseline or follow-up.
Herbener et al. (2005) proposed that this finding suggests that FAR is a stable deficit in schizophrenia patients and that a residual core of persistent negative symptoms exists that is difficult to treat and is strongly linked to FAR deficits. The relationship between specific symptoms and FAR may also change over time. Kohler et al. (2000) found that in chronic but clinically stable patients during a baseline evaluation, errors on a FAR task were associated with alogia and positive symptoms (hallucinations and thought disorder). During follow-up assessment 3 to 8 months later, the relationship between FAR and alogia remained significant while FAR performance was no longer related to positive symptoms. The authors suggested that these findings reflect the relative stability of negative symptoms over time, whereas positive symptoms tend to be more variable and therefore less likely to consistently correlate with FAR deficits.

Relationships among FAR performance and symptomatology may also differ as a function of patient population. For instance, two recent studies comparing FAR performance in a forensic schizophrenia patient group and a non-forensic patient group reported different relationships between symptoms and FAR performance in both groups. Wolfkühler et al. (2012) found that in the forensic group, FAR was negatively correlated with both cognitive and positive symptoms, whereas in the non-forensic group, FAR was only correlated with cognitive symptoms. Similarly, Demirbuga et al. (2013) reported that in violent forensic patients general psychopathology was positively correlated with sad and angry accuracy on a FAR task. In non-forensic patients, the relationships were much more complex: positive symptoms were negatively correlated with happy accuracy; negative symptoms were negatively correlated with happy and surprised accuracy; and
general psychopathology was negatively correlated with happy, angry, and surprised accuracy.

While the majority of research suggests a relationship between FAR and negative symptoms in patients, some findings suggest that the relationship between FAR and symptoms may be more complicated. For instance, there is some research to suggest that FAR is negatively correlated not only with negative symptoms but also disorganized and catatonic symptoms in first-episode schizophrenia patients (Edwards et al. 2001).

Mandal et al. (1999) divided a group of patients with chronic schizophrenia into patients with predominantly positive symptoms and patients with predominantly negative symptoms on the basis of the PANSS percentile scores. Accordingly, patients in the predominantly positive symptom group were on average in the 76.33 percentile on the positive symptom scale and in the 44.08 percentile on the negative symptoms scale, while the patients in the predominantly negative symptom group were on average in the 27.66 percentile on the positive symptom scale and in the 87.75 percentile on the negative symptoms scale. The authors found that both patient groups were impaired on FAR compared to controls. However, the patients with predominantly negative symptoms were significantly less accurate than those with predominantly positive symptoms; suggesting that while both domains of symptoms are associated with FAR deficits, negative symptoms are associated with a more generalized emotion-recognition deficit. Furthermore, some researchers have found no relationship between FAR and negative symptoms. Kee, Green, Mintz, & Brekke (2003) reported that negative symptoms were not related to FAR either at baseline or at 12 month follow-up in patients with chronic schizophrenia.
On the other hand, other psychotic (e.g., positive, disorganized, first-rank) and cognitive symptoms have been associated with FAR. Specifically, positive symptoms were negatively correlated with FAR at baseline at a trend level, and conceptual disorganization was negatively correlated with FAR at baseline and follow-up (Kee et al., 2003). Similarly, Larøi et al. (2010) reported that FAR was negatively correlated with positive and cognitive symptoms. Some authors have classified patients by whether or not they experience first-rank symptoms which include positive symptoms such as auditory hallucinations, delusions of control, thought withdrawal, thought insertion, and thought broadcasting. Patients who experienced these first-rank symptoms made more FAR errors than patients who did not experience them and instead, experienced predominately negative symptoms (Beheré et al., 2011b). Interestingly, one recent study reported that after adjusting for positive, negative, cognitive, and Post-Traumatic Stress Disorder symptoms, greater levels of dissociative symptoms predicted poorer ability to recognize negative emotions in patients with schizophrenia (Renard, Pijnenborg, & Lysaker, 2012), suggesting that dimensions of psychopathology beyond schizophrenia symptoms may contribute to FAR deficits in patients.

At least three studies have found no relationship between clinical symptoms and FAR performance in patients with chronic schizophrenia (Li et al. 2010b, Leung et al. 2011; Goghari & Sponheim, 2012) and those experiencing their first episode of psychosis (Leung et al., 2011). It should be noted that in a recent meta-analysis, Chan et al. (2010) reported that negative symptoms only as measured by the PANSS were associated with FAR performance such that more severe symptoms were related to more FAR impairment. Negative symptoms measured by the Scale for the Assessment of Negative
Symptoms (SANS) and positive symptoms measured by the PANSS and the Scale for the Assessment of Positive Symptoms (SAPS) were not associated with FAR. The PANSS and SANS-SAPS scales are moderately correlated (Masiak & Loza, 2004; Rabany, Weiser, Werbeloff, & Levkovitz, 2011); however, principal components analyses demonstrate that while the SANS-SAPS has a 3-factor solution (i.e., negative, positive, disorganized symptoms), the PANSS yields these three factors plus a depression component (Dollfus & Petit, 1995; Masiak & Loza, 2004) and a cognitive component (Masiak & Loza, 2004). Given that Li et al. (2010b) used the PANSS to measure negative symptoms, which has been demonstrated to correlate with FAR performance, the null findings should be interpreted with caution as they may reflect limited power of the study to detect true relationships. Furthermore, Leung et al. (2011) and Goghari & Sponheim (2012) used the SANS-SAPS scales to assess symptoms highlighting the importance of appropriate and evidence-based measurement selection in this area of research. It should be noted that the meta-analysis by Chan et al. (2010) only included four of the studies previously reviewed in this paper. The meta-analysis only included studies published before January 2008, therefore, Alfimova et al. (2009), Habel et al. (2010), and Li et al. (2010b) were not included. Furthermore, studies that did not include a non-emotional facial processing control task and/or lacked sufficient data to calculate effect sizes were excluded (i.e., Kohler et al., 2003, Bediou et al., 2005).

Notwithstanding these issues, the conclusions of Chan et al. (2010) are consistent with the findings of the studies reviewed here; namely, patients with schizophrenia have impaired recognition of facial expressions and in general, patients with severe negative symptoms demonstrate more severe FAR deficits.
To date, few studies have addressed the relationship between FAR deficits and symptomatology in at-risk individuals (e.g., clinical high-risk, familial high-risk, psychometric risk). Two recent studies examining UHR individuals supported the relationship between FAR deficits and negative symptoms (Addington et al. 2012; Wölwer et al., 2012). Some research has supported the relationship between FAR and negative symptoms among familial high-risk samples. For instance, Surguladze et al. (2012) reported that scores on the SPQ interpersonal scale (SPQ-I; negative-like symptoms) were negatively associated with FAR accuracy in first-degree relatives of patients and in healthy controls. On the other hand, Alfimova et al. (2009) reported that unlike their sample of patients with schizophrenia where negative symptoms were negatively correlated with FAR, in the relatives of these patients there was no association between SPQ subscales or total score and FAR performance. Surprisingly, the authors did find evidence of a negative correlation of the SPQ cognitive-perceptual scale (SPQ-CP; positive-like symptoms) and the “unusual perceptual experiences” subscale (also reflecting positive-like symptoms) with FAR in the healthy controls. The discrepancies in findings between Surguladze et al. (2012) and Alfimova et al. (2009) may be accounted for by methodological differences between the studies. Surguladze et al. (2012) only asked subjects to label faces as “emotional” or “neutral” without labeling the specific emotion whereas Alfimova et al. (2009) asked participants to explicitly generate the name of each emotion presented (i.e., happiness, surprise, sadness, anger, disgust, fear, interest/excitement, contempt, shame). Alfimova et al’s (2009) task may be too difficult and introduces a potential language confound. Most FAR measures use a forced-choice or multiple-choice format to avoid confounding emotion recognition
deficits with known language deficits – including reduced verbal fluency – in first-degree relatives of schizophrenia patients (see Keefe et al., 1994; Chen, Chen, & Lieh, 2000; Egan et al., 2001; Bhojraj et al., 2009; Maziade et al., 2009; Keshavan et al., 2010). In addition to not using a forced-choice format, Alfimova et al. (2009) also included more complicated social emotions (i.e., excitement, contempt, shame), which are not usually included in FAR studies. These deviations from the standard methodology used in FAR studies may at least in part account for the reported findings, which are inconsistent with previous research. However, it may also be true that in at-risk populations the relationship between FAR and symptoms does not reflect the pattern established in patient populations. For instance, Eack et al. (2010) also found that first- and second-degree relatives of patients with schizophrenia were significantly worse at identifying neutral faces and that this deficit was related to greater attenuated positive and general psychopathology symptoms but not negative or disorganized symptoms. General psychopathology symptoms were also associated with increased reaction time for identifying neutral faces.

It is also unclear if positive, negative, disorganized, or general schizotypal symptoms are differentially related to FAR performance in psychometrically at-risk samples, as studies to date have reported conflicting results. For example, using the Penn Emotion Recognition Test (Kohler et al. 2003), Brown & Cohen (2010) reported that only general schizotypy was related to accuracy on a FAR task such that individuals with high total SPQ scores performed worse than individuals with low scores but no specific symptom dimensions were related to accuracy. Using both a facial affect discrimination task and a popular FAR task, Williams et al. (2007) reported that general schizotypy was
negatively correlated with ability to discriminate different emotions but only negative symptoms (SPQ-I) were negatively correlated with ability to discriminate and identify emotions. Similarly, Abbott & Green (2013) reported that reduced FAR accuracy was associated only with the SPQ-I factor and more specifically, with the social anxiety subscale. All three of these psychometric at-risk studies used FAR measures that have been previously used with schizophrenia populations. However, studies using idiosyncratic FAR tasks tend not to support the relationship between negative symptoms and FAR deficits commonly reported in patients. Germine & Hooker (2011) measured FAR using computerized neutral faces that slowly morphed into emotional expressions of happiness, disgust, anger, or fear. The authors reported that all three schizotypal symptom dimensions and general schizotypy (as measured by the SPQ) predicted FAR performance. Similarly, van’t Wout et al. (2004) assessed FAR by presenting angry, happy, fearful, and neutral faces in which the visual contrast had been reduced by 30% and they reported that only positive symptoms were related to the number of errors on this task. It should also be noted that the findings of van’t Wout et al. (2004) may be at least partially accounted for by the fact that participants were initially selected on the basis of positive symptomatology alone. Similarly, Roddy et al. (2012) reported that children identified as psychometrically at-risk based on the presence of only sub-clinical positive symptoms performed significantly more poorly on a FAR task especially when labeling sad faces.

In summary, to date, the majority of research in patients with schizophrenia overwhelmingly suggests that FAR deficits are related to severity of negative (but not positive) symptoms. Numerous methodological issues (including variability in the
measures used to assess both symptom severity and FAR performance) across studies of familial and psychometrically at-risk individuals limit the ability to compare directly across studies and draw conclusions regarding the relationship between symptoms domains and FAR performance in at-risk populations. However, in those studies assessing the full spectrum of schizotypal symptoms and using previously validated FAR measures (i.e., Williams et al., 2007; Brown & Cohen, 2010; Abbott & Green, 2013), the relationship between FAR deficits and negative symptomatology tends to be supported in familial and psychometrically at-risk individuals, following the general pattern observed among patient samples.

**Response Bias in FAR.** The majority of research to date supports the existence of a response bias in FAR in patients with schizophrenia. However, due to methodological differences, direct comparison across studies is difficult and the exact nature of this bias remains unclear.

There exists a relatively small number of studies reporting the absence of an emotional response bias. One early study of FAR in patients with schizophrenia, reported that there was no evidence of a response bias (i.e., mislabeling joy, anger, surprise, disgust, shame, fear, sadness, and interest) when compared to healthy controls (Walker et al., 1980). Similarly, Hooker & Park (2002) found no evidence of a bias in the error pattern of patients with schizophrenia. On a simplified FAR task where patients, their siblings, and controls were asked to label faces as either “emotional” or “neutral” without specifically labeling the emotion expressed, neither patients, siblings, nor controls demonstrated a response bias (Surguladze et al., 2012). That is, none of the groups incorrectly labeled neutral faces as “emotional.”
Reports supporting a FAR emotion bias, however, are more common. For instance, using the Penn Facial Discrimination Task (which asks subjects to label happy, sad, and neutral facial expressions), Mandal et al (1999) found that patients with schizophrenia who predominantly experienced positive symptoms were more likely to misattribute happy to faces displaying sad expressions than both controls and patients with negative symptoms. Using the same task, Kohler et al. (2000) found a significant emotion by group interaction such that patients with schizophrenia were significantly worse at labeling happy faces than controls. While patients also performed significantly worse than controls at labeling sad faces, this effect was weaker than the interaction effect when labeling happy faces. The Penn Facial Discrimination Task is relatively simplistic and FAR tasks that require subjects to label a wider array of emotions have typically found that patients with schizophrenia have more difficulty labeling negatively valenced emotions. For example, Edwards et al. (2001) reported that first-episode patients experienced more difficulty labeling fear and sadness compared to controls on a FAR task developed by the first author. Discrepancies in bias findings are likely in large part due to methodological differences resulting from using FAR tasks which vary with regard to face stimuli presented, task demands, task difficulty, and the way bias was conceptualized, calculated, and analyzed.

Other studies have reported a significant group by emotion interaction, such that while patients with schizophrenia may have general deficits in FAR, they are particularly impaired at labeling specific emotions. For example, patients with schizophrenia and their siblings were significantly less accurate at labeling angry faces than at labeling happy faces whereas controls were equally accurate at labeling happy, angry, and neutral
faces (Leppänen et al. 2008). That is, patients and their siblings were particularly impaired at labeling angry faces, thus demonstrating a type of bias. Similarly, Bediou et al. (2007) reported that siblings of patients were significantly worse at labeling only disgust and fear (but not anger and happiness) compared to controls and Machado de Sousa & Hallak (2008) reported that while patients were equally as accurate on a FAR task as controls, they were significantly slower to recognize only disgust and fear, again suggesting that patients demonstrate a response bias during FAR performance. Recently, authors have begun to systematically examine in which specific emotions patients demonstrate deficits, to determine whether deficits in specific emotions drive FAR deficits. Reports vary and suggest that patients are impaired on labeling 1) anger only (Goghari & Sponheim, 2012; Janssens et al., 2012), 2) neutral and fear (Gilling McIntosh, 2011), 3) surprise, fear, and disgust (Leung et al. 2011), and 4) threat emotions such as anger, fear and disgust (Behere et al. 2011b) when compared to healthy controls.

Most studies examining response bias in schizophrenia patients have not examined the relationship between bias and symptom dimensions. One study that has examined this relationship reported that while patients did not demonstrate a negative bias as hypothesized, different types of bias were uniquely related to symptom dimensions (Cohen, Nienow, Dinzeo, & Docherty, 2009). For instance, misattribution of fear to other emotional faces was associated with more positive and depressive/anxious symptoms, misattribution of anger was associated with more disorganized and negative symptoms, and misattribution of shame was associated with less severe positive and disorganized symptoms (Cohen et al., 2009). Palmese (2009) examined bias while viewing neutral scenarios in patients with schizophrenia, and found that patients
misattributed surprise and fear to these scenarios more frequently than controls. Patients with more positive symptoms performed more accurately when assessing these neutral scenarios; the author suggested that patients with increased levels of paranoia may be more suspicious of their surroundings and more vigilant, which may lead to increased attention to detail and greater accuracy (Palmese, 2009).

Studies comparing individuals with SPD to controls with respect to response bias have been somewhat more inconclusive. Individuals with SPD were significantly less accurate at labeling negatively valenced emotions (i.e., anger, disgust, shame, fear, sadness) than positively valenced emotions (i.e., joy, surprise, interest) while controls were equally accurate for all emotions (Waldeck & Miller, 2000). However, when examining individual emotions as opposed to broader categories based on valence, individuals with SPD were only less accurate at labeling joy and surprise. Wickline et al. (2012) reported that SPD adolescents made more errors when labeling angry faces than controls but they also made more errors labeling happy faces when compared to adolescents with other personality disorders. When examining the types of misattribution errors, the errors were similar across the three groups.

Findings are similarly mixed in psychometrically at-risk individuals. For instance, Williams et al. (2007) reported that the SPQ-I scale was associated with reduced accuracy for negatively valenced emotions (disgust, anger, sadness, fear) suggesting that at-risk individuals are particularly poor at identifying negative emotions while they do not differ from individuals with low schizotypal traits in their ability to identify happy faces. Van’t Wout et al. (2004), however, reported that the SPQ-CP scale was associated with erroneously labeling angry faces as happy. Furthermore, the “unusual perceptual
experiences” SPQ subscale was correlated with mislabeling happy faces as angry and happy faces as fearful. Therefore, while both studies seem to support a negative bias in psychometrically at-risk individuals, the symptoms associated with this bias are unclear.

As opposed to the aforementioned studies that defined bias in an idiosyncratic way, numerous researchers have recently begun to look at bias in SSDs more systematically. Specifically, newly developed FAR tasks often include neutral faces which allows investigators to examine whether individuals misattribute emotion to these neutral faces and then to examine the error patterns to see if any particular emotions are more likely to be misattributed in these cases. The Penn Emotion Recognition Test, developed by Kohler and colleagues (2003), is the mostly commonly used FAR task to measure bias in this way. Kohler et al. (2003) reported that patients with schizophrenia and controls only differed in the errors that they made when labeling neutral faces. Examining the error pattern more closely, patients were more likely to attribute disgust to neutral faces. Later studies with patients, their relatives, and psychometrically at-risk individuals using the Penn Emotion Recognition Test have consistently demonstrated that individuals with SSDs are less accurate only when labeling neutral faces and that labeling of all emotional faces is not impaired (Eack, et al., 2010; Brown & Cohen, 2010; Pinkham, et al., 2011). This body of literature suggests that negative bias largely emerges in the context of labeling neutral faces as opposed to the mislabeling of emotionally valanced faces which is interesting given that a number of the studies that reported no evidence of bias or misattribution errors did not present neutral faces (Walker et al., 1980; Waldeck & Miller, 2000; Hooker & Park, 2002; Wickline et al., 2012). For instance, when comparing actively paranoid and not actively paranoid schizophrenia
patients using a task that does not include neutral faces, Silver & Shlomo (2001) found no group differences in performance. However, using Kohler et al.’s (2003) FAR task, Pinkham et al. (2011) demonstrated that actively paranoid patients were only worse at labeling neutral faces as compared to not actively paranoid patients and that they were more likely to misattribute anger to neutral faces. First- and second-degree relatives of patients were also more likely to misattribute negative emotions to neutral faces when compared to controls, most commonly labeling the faces as sad (Eack et al., 2010). Psychometrically at-risk individuals – as determined by the SPQ – were more likely to label neutral faces as disgusted compared to controls (Brown & Cohen, 2010). Using faces that had been passed through a filter in order to reduce their visual contrast, van Rijn et al. (2011) reported that UHR individuals were only more impaired than controls when labeling neutral faces and that they were more likely to misattribute anger to neutral faces. Therefore, while all these studies demonstrated a negative bias in SSD samples, some studies reported more misattribution of disgust (Kohler et al., 2003; Brown & Cohen, 2010), some anger (Pinkham et al., 2011; van Rijn et al., 2011), and one reported sadness was most commonly misattributed emotion to neutral faces (Eack et al., 2010).

As with the findings regarding the relationship between symptomatology and FAR, investigators have used various FAR measures and operationally defined bias in a number of different ways, leading to inconsistent findings and difficulty comparing findings across studies. There is currently evidence to support a negative bias in patients with schizophrenia. By assessing misattribution errors during the labeling of neutral faces, a limited number of studies have also provided evidence of a negative bias in
schizophrenia patients, their relatives and psychometrically at-risk individuals; expansion of this body of literature is warranted.

**Depression and FAR.** Though FAR deficits are consistently reported in patients with schizophrenia, they do not appear to be a feature unique to SSDs. In recent years there has been increased interest in exploring FAR deficits in a number of psychiatric disorders, chief among them depression. Some early reports suggested that FAR deficits in depressed individuals are a result of a generalized visuospatial impairment rather than a specific deficit (Asthana, Mandal, Khurana, & Hasque-Nizamie, 1998). More recent findings, however, consistently show FAR impairments in depressed patients independent of visuospatial impairments. For example, females with major depressive disorder (MDD) were significantly less accurate than healthy female controls on a FAR task despite equivalent performance on memory, visuospatial, motor, and attentional tasks (Langenecker, Bieliauskas, Rapport, Zubieta, Wilde, & Berent, 2005). Though the groups did significantly differ on inhibitory control, this executive functioning deficit was not related to FAR deficits.

The literature vastly demonstrates relatively impaired FAR in depression. Using faces that slowly morphed from neutral to emotional, Harmer et al. (2009) demonstrated that patients with MDD were impaired at recognizing happy and surprise faces relative to controls. However, when treated with a selective norepinephrine reuptake inhibitor, patients showed improved ability to recognize happy faces at lower intensities. Using a similar paradigm, LeMoult, Joormann, Sherdell, Wright, & Gotlib (2009) demonstrated that compared to controls, patients with recurrent MDD required more intense happy faces in order to identify them correctly. Unlike Harmer et al. (2009), however, LeMoult
et al. (2009) reported that the FAR impairment was evident even during remission. In a recent study, Naranjo et al. (2011) reported that inpatients with MDD were less accurate at recognizing emotions than controls not only from facial expressions (fear, anger, and neutral but not joy or sadness) but also from musical excerpts (happy and neutral but not sadness and fear) and vocal prosody (surprise and neutral but not fear, anger, joy, disgust, or sadness).

In contrast, two studies demonstrated that patients with MDD are more accurate at recognizing sad faces at lower intensities (Gollan, McCloskey, Hoxha & Coccaro, 2010; Milders, Bell, Platt, Serrano, & Runcie, 2010). However, while Gollan et al. (2010) found that the ability to identify sad faces was correlated with Hamilton Rating Scale for Depression scores, Milder et al. (2010) reported that this ability was not related to symptom severity and remained stable over six months despite reduction in symptoms, as measured by the Hamilton Rating Scale and Beck Depression Inventory.

Two recent meta-analyses substantiate findings of impaired FAR in depression. In a quantitative review of eight studies, Demenescu, Kortekaas, den Boer, & Aleman (2010) concluded that MDD was associated with FAR impairments ($d = -0.58$). However, it should be noted that though the authors identified 28 studies examining FAR performance in patients with mood and anxiety disorders, 12 studies were excluded because they did not include sufficient data to calculate effect sizes, used non-clinical participants, there was a significant age difference between patient and control groups, and other methodological differences. Importantly, a more recent meta-analysis including 51 studies of FAR in MDD patients also reported a moderate effect size ($d = -0.549$; Kohler, Hoffman, Eastman, Healey, & Moberg, 2011).
With regard to reaction time, Leppänen, Milders, Bell, Terriere, & Hietanen (2004) reported that MDD patients were significantly slower than controls at recognizing neutral faces, while Langenecker et al. (2005) reported no significant difference in reaction time during a FAR task despite the fact that MDD females were somewhat faster than healthy females on a control task. More recent studies have also reported no significant difference in reaction times between MDD and control groups (Harmer et al. 2009; Flanagan, White, & Carter, 2011). This may be due to methodological inconsistencies between the studies as Leppänen et al. (2004) used static photographs of happy, sad, and neutral faces only while Harmer et al (2009) used slowly morphing photographs of faces and included all six basic emotions (happiness, surprise, sadness, fear, anger, and disgust) and Flanagan et al. (2011) used both static and morphing photographs of the six basic emotions.

Based on the literature, it also appears that patients with MDD have a significant negative bias when recognizing neutral expressions. Leppänen et al. (2004) reported that while controls were equally accurate at recognizing happy, sad, and neutral faces, MDD patients were less accurate at recognizing neutral faces than happy and sad faces. Moreover, MDD patients demonstrated a negative bias when recognizing neutral faces (i.e., labeling neutral faces as sad). When tested while in remission, the MDD patients were still impaired when recognizing neutral faces, however, now they demonstrated both a negative and a positive bias (i.e., labeling neutral faces as happy). Importantly, negative bias and positive bias were positively correlated leading the authors to posit that impairment in the processing of emotionally neutral faces may be a trait characteristic of depression. More recent studies have supported Leppänen et al.’s (2004) finding of a
negative bias for neutral faces (Gollan et al., 2010; Naranjo et al. 2011); however, Milders et al. (2010) reported that this bias remained stable over six months despite symptom reduction.

Another methodological consideration is that MDD patients with different types of depression may show differential FAR deficits. To this end, Flanagan et al. (2011) found that women with postpartum depression and women with non-postpartum depression were both significantly worse at recognizing happy and fearful faces compared to controls. However, women with postpartum depression performed worse than women with non-postpartum depression on disgusted and angry faces while women with non-postpartum depression performed worse than women with postpartum depression on happy faces. Therefore, perhaps postpartum depression is a unique type that does not demonstrate the typical pattern of deficits seen in non-postpartum depression suggesting that different types of depression may have unique etiological factors that contribute to different patterns of deficits and may inform diagnosis, treatment, and prognosis. Just as Flanagan et al. (2011) demonstrated impaired recognition of disgusted faces in women with postpartum depression, there is some evidence that patients with obsessive-compulsive disorder (OCD) also have impaired disgust recognition (Sprengelmeyer et al., 1997). Moreover, anxious intrusive thoughts associated with harm avoidant behaviors are present during the postpartum phase and resemble the symptoms of OCD (Leckman et al. 1999). There is also evidence that development of early parental attachment relies on neuroanatomical regions implicated in OCD (Swain et al., 2008). Together these findings suggest that excessive worry and emotional distress during the postpartum phase may be more akin to OCD than MDD.
It appears that FAR deficits are, therefore, not a unique characteristic of SSDs as they are also evident in a range of psychiatric patients, including those with MDD and bipolar disorder (Lembke & Ketter, 2002; Goghari & Sponheim, 2012; see Kohler et al., 2011 for review), anxiety disorders (Sprengelmeyer et al., 1997; Kessler, Roth, von Wietersheim, Deighton, & Traue, 2007; Demenescu et al., 2010), Body Dysmorphic Disorder (Buhlmann, McNally, Etcoff, Tuschen-Caffier, & Wilhelm, 2004), and autism spectrum disorders (Bölte & Poustka, 2003). There are very high rates of comorbidity between schizophrenia and MDD (see Buckley, Miller, Lehrer, & Castle, 2009 for review). Even in the general population, among psychometrically high-risk individuals there is a large positive correlation between schizotypal traits and depressive symptoms (Fonseca-Pedrero, Paino, Lemos-Giráldez, & Muñiz, 2011).

Despite comorbidity, most FAR studies in patients with schizophrenia do not measure or adjust for depressive symptoms and, therefore, it remains unclear to what extent impairments may be due to a common underlying etiology or perhaps distinct neural processes. Of course, the issue of covariance is a complicated one (Miller & Chapman, 2001), and thus simply covarying for depressive symptoms is not a clear approach to reconciling this issue.

Some investigators have begun to investigate the impact of psychotic versus depressive symptoms on FAR ability in patients with schizophrenia and those at risk for SSDs. One early study comparing FAR in newly admitted inpatients with schizophrenia to those with MDD reported that patients with schizophrenia were significantly less accurate than controls, though MDD patients did not demonstrate a recognition deficit compared to controls (Gaebel & Wölwer, 1992). When tested again four weeks later, all
groups improved compared to baseline, however, the schizophrenia patients were still impaired compared to the control and MDD groups. Moreover, at follow-up the patients with schizophrenia were significantly worse than MDD patients at recognizing fearful faces. Gaebel & Wölwer (1992) concluded that patients with schizophrenia demonstrate a trait-like deficit in FAR that is not ameliorated by pharmacological treatment while patients with MDD do not demonstrated an FAR deficit. These results are supported by a more recent study which also demonstrated that patients with schizophrenia are significantly less accurate than patients with MDD and controls, while patients with MDD did not differ from controls (Bediou et al., 2005). Moreover, patients with schizophrenia were significantly less accurate than controls at identifying disgusted faces and (as in Gaebel & Wölwer, 1992) patients with schizophrenia were significantly less accurate than controls and patients with MDD at identifying fearful faces.

Similarly, Weniger, Lange, Ruther, & Irle (2004) reported that patients with disorganized and paranoid type schizophrenia were most impaired on a task requiring sorting emotional faces into groups, while MDD patients had only minor deficits on this task and patients with residual schizophrenia did not differ from controls. While informative, this FAR task is somewhat idiosyncratic and replication studies using reliable and validated measures of FAR are needed.

In a sample of patients with schizotypal personality disorder (SPD) and comorbid depression, patients with MDD, and healthy controls, Mikhailova et al. (1996) reported that both patient groups were significantly impaired in recognition of negative and positive facial expressions compared to controls at baseline. However, though both patient groups were treated with antidepressants for approximately eight weeks and
experienced significant amelioration of depressive symptoms, patients with MDD showed significant recovery of recognition abilities while in patients with SPD recognition did not improve during remission. Mikhailova et al. (1996) concluded that FAR deficits in MDD and SPD are driven by different neurophysiological mechanisms and as a result these deficits are “state” related in MDD while they represent a “trait” deficit in SPD.

Given evidence of FAR impairment in depressed patients when compared to healthy controls and the high comorbidity of depression in patients with schizophrenia, it is possible that at least some of the reported FAR impairments in schizophrenia may actually be due to depressive symptomatology. For example, though patients with panic disorder demonstrate impaired FAR compared to healthy controls, between group FAR accuracy differences disappeared after adjusting for depressive symptoms (Kessler et al., 2007). It remains unclear to what extent this is attributable to independent (versus shared) symptomatology and/or underlying neural mechanisms. It is important to tease apart what (if any) elements (i.e., accuracy, reaction time, negative bias) of the observed FAR impairments may be due to psychotic symptoms as compared to depressive symptoms and furthermore, to examine how the relationship between FAR impairments and symptomatology reflects unique neurobiological mechanisms of schizophrenia versus depression. Few studies examining FAR in SSD samples have measured depressive symptomatology. In patients with schizophrenia, severity of depressive symptoms as measured by the Hamilton Rating Scale for Depression was not associated with FAR performance (Kohler et al., 2000). Similarly, Bediou et al. (2007) reported that depressive symptoms were not related to FAR performance in a control group, patients
with schizophrenia, or their siblings. On the other hand, Hofer et al. (2009) reported that severity of negative symptoms was negatively correlated with ability to recognize surprised faces while severity of depressive and anxiety symptoms was positively correlated with ability to recognize fearful faces in patients with schizophrenia. Therefore, the absence of depressive and/or anxiety symptoms in patients may lead to worse performance on FAR tasks. Given that a number of negative symptoms are very similar to depressive symptoms, and negative symptoms are most commonly found to be related to FAR performance in schizophrenia patients, the results of Hofer et al. (2009) suggest that it is important to distinguish between the two types of symptoms in order to get a clearer understanding of the underlying mechanisms driving FAR deficits in schizophrenia.

The relationship between depressive symptoms versus negative (and other) psychotic symptoms with FAR performance deficits is also mixed in the general population. In a psychometrically at-risk college sample, individuals at high risk reported significantly higher levels of negative affect as measured by the Hospital Anxiety Depression Scale (Williams et al., 2007). Only after adjusting for Hospital Anxiety Depression Scale scores, was negative schizotypy (SPQ-I) negatively correlated with total FAR, recognition of negative faces, and facial affect discrimination. Csukly, Czobor, Simon, & Takacs (2008) examined the relationship between FAR and psychiatric symptoms in a healthy adult sample as measured by the Symptoms Checklist-90. They reported a negative correlation between FAR performance and level of depressive, psychotic, OCD, and phobic-anxiety symptoms. OCD, depressive, and hostility symptoms were also related to negative bias. Csukly et al. (2008) did not adjust
for comorbid psychiatric symptoms in order to determine the unique contribution of psychotic symptoms to FAR performance. Therefore, it appears that reduced FAR accuracy may be related to a host of psychiatric symptoms, while specific features – such as negative bias when recognizing neutral faces – may result from a more circumscribed set of symptoms.

It is important to examine FAR deficits in various patient populations and in those at risk for psychopathology because these deficits may contribute to the social functioning impairments that have been demonstrated in patients with schizophrenia and individuals at clinical and psychometric risk for SSDs (Couture, Penn, & Roberts, 2006), with implications for understanding etiology and potential treatment strategies.

Social Functioning

SSDs and Social Functioning. Social functioning can be conceptualized as the ability to establish and maintain social relationships as well as the frequency and quality of participation in social interactions through involvement in socially-oriented activities, organizations, and hobbies (Birchwood, Smith, Cochrane, Wetton, & Copestake, 1990). Impairment in social functioning is a hallmark of schizophrenia (Bellack, Morrison, Wixted, & Mueser, 1990). Whether these social functioning deficits are present before illness onset and whether they are present in less severe illnesses along the schizophrenia spectrum remained unclear until recently. Research with UHR and psychometrically at-risk subjects has helped to elucidate the nature of social functioning deficits in SSDs. Moreover, given that successful social interactions rely on the ability to read the emotions of others (Riggio, 1992), the deficits in FAR demonstrated in individuals with SSDs may at least in part explain these individuals’ impaired social functioning (see SSDs, FAR,
and Social Functioning section below), as FAR deficits may interfere with and perhaps even prevent the formation and maintenance of social relationships.

Using the Premorbid Adjustment Scale (Cannon-Spoor, Potkin, & Wyatt, 1982), a number of researchers have tried to ascertain if and to what extent patients with schizophrenia demonstrated functional impairment before illness onset. Cannon et al. (1997) found that patients with schizophrenia were impaired in both premorbid social and academic functioning compared to healthy controls. Furthermore, patients with schizophrenia had significantly worse premorbid academic functioning than patients with bipolar disorder. Cannon et al. (1997) also reported that schizophrenia patients exhibited a greater deterioration in functioning during adolescence than bipolar patients. Similarly, Monte, Goulding, & Compton (2008) reported that patients with schizophrenia experienced a deterioration in academic and social functioning from childhood to early adolescence and then an accelerated deterioration specifically in academic functioning from early to late adolescence.

The “schizophrenia prodome” is believed to commonly occur during late adolescence, and a number of retrospective studies with patients experiencing their first psychotic episode have reported impairments in social and role functioning as a key feature of the prodrome. For instance, in their review, Yung and McGorry (1996) reported that social withdrawal and deterioration in role functioning were among the prodromal features most commonly reported in first episode studies. During open-ended interviews with family members of patients with recent onset of psychosis, Corcoran et al. (2007) found that family members reported that social withdrawal and decline in academic functioning first developed in previously normal children as they entered
adolescence. Møller and Husby (2000) identified four potential dimension of prodromal behavior: 1) quit school, university, or job, or major school truancy, 2) marked and lasting observable shifts of interest, 3) marked and lasting social passivity, withdrawal, or isolation, and 4) marked and lasting change in global appearance or behavior. Among their sample of first episode patients, the authors reported that 17 of the 19 patients experience deterioration in school or work functioning and 14 out of 19 experienced social withdrawal (Møller & Husby, 2000). Similarly, Tan and Ang (2001) reported that 83% of their first episode patients experienced social withdrawal during the prodromal stage while 70% experienced deterioration in academic performance. Moreover, both of these symptoms appeared to be specific to the psychosis prodrome as patient with non-psychotic psychiatric disorders did not report these symptoms before disease onset.

More recent prospective studies with UHR individuals have attempted to further clarify the social and role dysfunction seen during the putative prodromal phase. Much like Møller and Husby (2000), Cornblatt et al. (2003) identified four risk factors in UHR individuals, namely cognitive deficits, affective disturbances, social isolation, and school failure. Lencz et al. (2004) reported that social isolation/withdrawal was the most common presenting symptoms in their sample of UHR patients. On the Structured Interview for Prodromal Symptoms (Miller et al., 1999), social isolation and deterioration in academic performance were the most frequently endorsed items such that virtually all UHR individuals had clinical significant deterioration in these domains (Lencz et al., 2004). Shim et al. (2008) showed that both UHR individuals and those genetically at risk for psychosis had impaired social functioning compared to controls as measured by the Social Functioning Scale (SFS; Birchwood et al., 1990) but UHR individuals exhibited
more social dysfunction than genetically at-risk individuals. Ballon, Kaur, Marks, & Cadenhead (2007) reported that UHR subjects and patients experiencing their first psychotic episode were equally impaired when compared to healthy controls in the domains of peer, family, work and school relationships. Similarly, in a study directly comparing UHR subjects to first episode patients, patients who have experienced multiple psychotic episodes, and healthy controls, Addington, Penn, Woods, Addington, & Perkins (2008b) reported that UHR, first episode, and multiple episode subjects were all impaired on the SFS compared to controls. However, when Addington et al. (2008b) examined specific areas of social functioning, it became clear that while the UHR group was impaired compared to controls in the areas of employment and role functioning, they were significantly less impaired than either patient group. These results suggest that while premorbid social functioning is impaired in at-risk groups, florid psychotic symptoms may further exacerbate deficits at least in some areas of functioning. Previous findings from the same group of authors have shown that while first episode and multiple episode patients were significantly more impaired than controls on several measures of social functioning (including the SFS), first episode and multiple episodes did not differ from one another on the SFS nor on premorbid functioning (Grant, Addington, Addington, & Konnert, 2001). Taken together, these results suggest that some level of social dysfunction is apparent in patients with schizophrenia and those at heightened risk for the disorder regardless of illness chronicity.

Better understanding of social functioning in UHR groups may help us to better predict which at-risk individuals will develop a psychotic disorder and help us to design treatment and potentially even intervention programs. Yung et al. (2007) estimated that
approximately 36.7% of individuals identified as UHR or prodromal using current criteria will convert to diagnosable psychosis within one year. Therefore, our current ability to predict conversion is limited and may benefit from adding premorbid and prodromal social functioning as a predictor variable. By conducting open-ended interviews with family members, Corcoran et al. (2003) were able to identify “declining” prodromal individuals who were characterized by behavioral changes including profound social withdrawal, odd behavior, and changes in school and work performance as well as “never normal” prodromal individuals who were described as having problems since birth, with clear developmental delays and early introduction into special education programs. The “declining” individuals had a higher subsequent rate of conversion to psychosis than the “never normal” individuals (Corcoran et al., 2003). Similarly, Yung, Phillips, Yuen, and McGorry (2004) found that – along with long duration of symptoms, high levels of depression, and reduced attention – poor social and role functioning as measured by the Global Assessment of Functioning (GAF; American Psychiatric Association, 2000) at baseline predicted conversion to psychosis at 12-month follow-up. Mason et al. (2004) conducted a longitudinal study and found that exactly 50% of UHR individuals converted to psychosis at one-year follow-up and – along with presence of magical thinking, flat affect, and auditory hallucinations – premorbid role impairment and asociality improved the ability to predict conversion. Cornblatt et al. (2007) recently developed two new measures of social and role functioning to be used during the prodromal phase of psychosis (Global Functioning: Social and Global Functioning: Role). Using these measures, Cornblatt et al. (2007) found that impairment on social functioning predicted conversion to psychosis while GAF and role functioning did not. Karlsgodt, Niendam,
Bearden, & Cannon (2009) found that social functioning scale score at 15-month follow-up was related to later conversion. Mittal et al. (2011) found that poorer baseline role functioning score predicted conversion at 12-month follow-up at a trend level. Cornblatt et al. (2012) reported that UHR individuals who later converted to psychosis were more likely to demonstrate impairments on the social functioning scale than matched non-converters, and that onset of psychosis did not further disrupt social functioning. Using prediction algorithms, Cannon et al. (2008) reported that five features assessed at baseline contributed uniquely to the prediction of psychosis: a genetic risk for schizophrenia with recent functional decline, higher levels of unusual thought content, higher levels of suspiciousness, greater social impairment (as measured by Cornblatt et al.’s (2007) social functioning scale), and a history of substance abuse. Taken together, these risk factors dramatically increased positive predictive power over the Structured Interview for Prodromal Symptoms diagnostic criteria alone from 35% to 74-81% (Cannon et al., 2008). It should be noted, that while some studies have reported worse social functioning among UHR individuals compared to controls, social functioning at baseline did not predict later conversion (Corcoran et al., 2011). This may be due to methodological issues including small sample size and sample characteristics (i.e., predominately male, more ethnically diverse) as well as resulting from using a social functioning measure that was not developed for UHR individuals and may therefore lack sensitivity or specificity to measure their unique deficits.

While social functioning may be impaired in UHR individuals, these impairments may not be stable or chronic. In another longitudinal study, Niendam et al. (2007) reported that over an 8-month period, 50% of UHR subjects demonstrated improvement
in social and role functioning while the other 50% demonstrated stability or decline in functioning. Improvement in both positive and negative symptoms was associated with functional improvement while stable clinical symptoms were associated with functional stability or decline. Though pharmacologically treating putatively prodromal individuals remains a controversial issue (see Cornblatt, Lencz, & Kane, 2001; McGlashan, 2001; Warner, 2005; McGlashan, 2005; Filakovic, Degmecic, Koic, & Benic, 2007), at least one study has demonstrated that treating UHR individuals with an atypical antipsychotic (amisulpride) not only reduced positive, negative, and depressive symptoms but also improved global functioning (Ruhrmann et al., 2007). Similar to some retrospective studies which showed deterioration in academic performance during late adolescence (Monte et al., 2008), Cornblatt et al. (2007) reported that role functioning (which includes occupational and academic functioning as measured by Cornblatt et al.’s (2007) role functioning scale) declined during the year before assessment. However, role functioning actually improved over a 12-month follow-up period leading the authors to conclude that role functioning is responsive to a combination of psychosocial (i.e., individual psychotherapy, family therapy, group therapy, school-based therapy or counseling, case management) and pharmacological treatment. In contrast, impairment in social functioning was more stable and showed little improvement over time despite treatment, leading Cornblatt et al. (2007) to propose that social impairment is a stable trait in UHR individuals. When changes in social functioning do occur, they may assist in predicting conversion. Those individuals who ultimately go on to experience psychosis may experience declines in social functioning, whereas those who do not convert to psychosis may show improvements in social functioning over one-year follow-up (Jang et al.,
2011). Furthermore, improvements in prosocial behavior appear to be associated with pharmacological treatment in UHR individuals (Jang et al., 2011).

Therefore, there is some evidence that reduction of symptom severity in UHR individuals may be associated with reduction in social impairment. However, is a particular group of psychiatric symptoms related to social impairment in SSDs? Research with patients with schizophrenia suggests that negative symptoms uniquely contribute to the social impairment found among these patients. For instance, Chaves, Seeman, Mari, & Maluf (1993) found that severity of negative symptoms in patient with schizophrenia was positively associated with greater disability and greater role impairment. Similarly, Blanchard, Mueser, & Bellack (1998) reported that poor social functioning in patients was associated with greater physical and social anhedonia, negative affect, and social anxiety. In first episode schizophrenia patients, Addington, van Mastrigt, & Addington (2003) found that poor social and interpersonal premorbid functioning was associated with negative symptoms while Voges & Addington (2005) reported that negative symptoms were a predictor of social functioning and were the only factor assessed to make a unique contribution to social functioning as measured by the SFS. A number of longitudinal studies have also supported the relationship between negative symptoms and social functioning. Dickerson, Boronow, Ringel, & Parente (1999) reported that negative symptoms and age at baseline predicted SFS total scores at 2-year follow-up. After following patients for four to six years, Gorna, Jaracz, Ryabkowski, & Rybakowski (2008) concluded that psychopathological symptoms in general were responsible for the majority of the explained variance in SFS scores and that negative symptoms seem to play the most important role especially as related to the
social engagement/withdrawal and independence-performance SFS subscales as well as the SFS total score.

While there is certainly evidence that negative symptoms contribute to social impairment in patients with schizophrenia, there is also evidence that other psychiatric symptoms play a role as well. For instance, Addington & Addington (1999) found that negative symptoms were related to performance on an interpersonal problem solving task but symptoms were not related to SFS total scores. When examining the subscales of the SFS, the authors reported that both positive and negative symptoms were related to the social engagement/withdrawal subscale, negative symptoms alone were related to the social behaviors and employment subscales, and positive symptoms alone were related to the independence-competence subscale. At 2.5 year follow-up, the authors reported that negative symptoms were associated with performance on the same interpersonal problem solving task but at this point negative symptoms were also associated with SFS total score (Addington & Addington, 2000). Moreover, performance on the interpersonal problem solving task was now also negatively associated with severity of positive symptoms. Therefore, there is evidence that social functioning is related to both positive and negative symptoms in patients with schizophrenia and that the nature of this relationship may change over the disease’s course.

These results are supported by more recent longitudinal studies. For instance, Wittorf, Wiedemann, Buchremer, & Klingberg (2008) assessed social functioning using the GAF and the Social Adjustment Scale II (Schooler, Hogarty, & Weissman, 1979). The authors reported that GAF, and the Social Adjustment Scale’s household functioning, social contacts and leisure, and general adjustment scores at 15-month
follow-up were predicted by fewer negative symptoms at baseline (Wittorf et al., 2008). However, fewer positive symptoms at baseline also predicted GAF, and the Social Adjustment Scale’s social contacts and leisure, and general adjustment scores. Goulding, Franz, Bernger, & Compton (2010) reported that social functioning as measured by the SFS was negatively correlated with negative, depressive, and general psychopathology symptoms in first episode schizophrenia patients. However, the social engagement/withdrawal subscale was uniquely associated with negative symptoms, the interpersonal communication subscale was associated with both negative and general psychopathology symptoms, and the employment/occupation subscale was uniquely associated with positive symptoms. There may also be sex differences in the relationship between symptoms and social functioning. As reported above, Chaves et al. (1993) found that negative symptoms were related to functional impairment in their sample of schizophrenia patients. However, in female patients only, higher positive symptoms were related to greater role impairment. Lastly, beyond positive and negative symptoms, some authors have reported that performance on a social role-playing task and community functioning as measured by the Social Adjustment Scale were inversely related to severity of disorganized symptoms while they failed to find any association with positive and/or negative symptoms and functioning (Cohen, Forbes, Mann, & Blanchard, 2006).

The relationship between functioning and symptomatology appears to be similarly complicated in UHR individuals. Some authors have reported that negative symptoms but not positive, disorganized, or general psychopathology symptoms are related to social and role functioning in UHR individuals (Cornblatt et al., 2007). However, reports that a combination of symptoms is related to poor functioning are more common. As described
above, Cornblatt et al. (2003) reported that cognitive deficits, affective disturbances, social isolation, and school failure formed a vulnerability core in UHR individuals. Moreover, the authors reported that all four risk factors were equally impaired in UHR individuals with attenuated moderate positive, attenuated severe positive, and attenuated negative symptoms (Cornblatt et al., 2003). However, school failure in particular was related to the negative symptom of avolition. In a sample of UHR individuals, familial high-risk individuals, and community controls, Svirskis et al. (2007) reported that GAF decreased linearly as positive symptom severity increased in the total sample. However, in addition to positive symptoms, negative symptoms predicted low GAF scores in the UHR group (Svirskis et al., 2007). Like Cohen et al. (2006), Shim et al. (2008) reported that neither positive nor negative symptoms were correlated with social functioning in a UHR sample but disorganized and general symptoms were strongly correlated with the independence-competence SFS subscale. Finally, in a recent study, Corcoran et al. (2011) concluded that poor social functioning in UHR individuals was primarily explained by negative symptoms. However, poor social functioning was also related to depressive, disorganized, and general psychopathology symptoms, but not positive symptoms. Even more interesting is evidence of ethnic group differences. Specifically, among ethnic minority patients, social functioning was correlated primarily with negative symptoms while in Caucasian patients social functioning was correlated primarily with general psychopathology symptoms.

There is some evidence that amelioration of symptoms in UHR subjects leads to improved social functioning (Niendam et al., 2007; Ruhrmann et al., 2007; Jang et al. 2011); however, while symptomatology is related to functioning in schizophrenia
patients, improvement in symptoms does not lead to improvement in functioning. For instance, in a sample of stable outpatients, Dickerson et al. (1999) found that four out of seven subscales of the SFS and the SFS total score did not significantly change over a two year period. Moreover, Goulding et al. (2010) reported no improvement in SFS scores between baseline and 6-month follow-up despite significant improvement in positive, negative, and general psychopathology symptoms. Though the follow-up periods are limited, these results highlight that early detection and treatment of symptoms during the putative prodromal period may be essential in order to improve social functioning and prevent further decline.

While limited, the available research suggests that psychometrically at-risk individuals also demonstrate impairments in social functioning. In non-clinical volunteers, high SPQ scores were associated with poorer social functioning as measured by the SFS (Henry, Bailey, & Rendell, 2008) and the Social Adaptation Self-evaluation Scale (Fonseca-Pedrero, Lemos-Giráldez, Paño-Piñeiro, Villazón-Garcia, & Muñiz, 2010). Similar to some findings in schizophrenia and UHR patients reviewed above, Henry et al. (2008) reported both the SPQ total score and the SPQ-I scale were negatively associated with social functioning, but only the SPQ-I persisted after adjusting for negative affect. Similarly, Heber, Castorina, & Dinzeo (2011) and Culianez, Dinzeo, Hayes, Bales, & Ciordano (2011) reported that SPQ-I was negatively correlated with all of the SFS subscales but most strongly with interpersonal communication and social engagement in a psychometric at-risk sample. However, Barrantes-Vidal, Lewandowski, & Kwapis (2010) reported that while undergraduates with high levels of negative schizotypal symptoms had impaired social functioning especially in voluntary social
activities and steady romantic relationships, individuals with high levels of positive symptoms were also socially impaired. Moreover, individuals with a mixture of negative and positive symptoms demonstrated the most impairment in social functioning. Using only subclinical paranoia symptoms (positive-like symptoms) to identify their psychometric risk group, Simpson & Pinkham (2011) also reported that individuals with high levels of paranoia had significantly lower scores on the independence-competence, social engagement, and interpersonal communication subscales of the SFS than individuals with low levels of paranoia. Healthy individuals who experienced frequent auditory hallucinations (positive symptom) also reported lower GAF scores and higher SPQ score, and high SPQ scores predicted lower GAF in these individuals (Sommer et al., 2010). Similarly, in health adolescents assessed longitudinally over three years, bizarre experiences and persecutory ideations (positive-like symptoms) were consistently and longitudinally associated with interpersonal functioning in family, peer, and general daily life domains (Collip et al. 2013). Therefore, while negative-like symptoms appear to be related to poorer social functioning in this at-risk population, positive-like symptoms are also contributory.

In summary, among schizophrenia patients there is clear evidence that negative symptoms are related to poor social functioning, though there is also some evidence that positive symptoms may uniquely contribute to social functioning. Among individuals at psychometric and clinical risk for SSDs, the relationship between symptoms and poor social functioning is also supported; though it is less clear whether negative, positive, disorganized, and/or general psychopathology symptom domains are mostly closely related to social functioning in these groups.
SSDs, FAR, and Social Functioning. A number of authors have suggested that the expressive flattening demonstrated by patients with schizophrenia may contribute to poor social functioning (e.g., Martin et al. 1990; Healey, Pinkham, Richard, Kohler, 2010). There is evidence that patients show less intense emotional expressions and display more negative expressions than controls, both of which are associated with poorer social functioning (Brozgold et al., 1998). More recently, Troisi, Pompili, Binello, & Sterpone (2007) reported that patients’ spontaneous facial expressivity was positively correlated with GAF scores and negatively correlated with work and social impairment. Given that successful interpersonal interactions require not only the ability to produce facial expressions but also the ability to recognize and identify the expressions of others, it is not surprising that FAR deficits have also been shown to be related to social functioning impairments in patients with schizophrenia. In long-term inpatients with schizophrenia, performance on a FAR task was related to performance on a conversation probe role-playing task measuring social competence as well as to social interactions and personal appearance and hygiene on the ward (Mueser et al. 1996). Cohen et al. (2006), however, reported that though better FAR performance was related to better community functioning, it was not related to a role-playing task in a similar sample of inpatients. In outpatients with schizophrenia, FAR performance was correlated with performance on a role-playing task and FAR deficits explained unique variance in social skills after adjusting for neurocognitive variables (Meyer & Kurtz, 2009). Accurate perception of both fear and neutral faces was correlated with role functioning, with perception of fear faces specifically accounting for a significant amount of variance in functional status (Brittain, Ffytche, & Surguladze, 2012). Correct recognition of facial expressions was
also associated with higher probability of competitive employment and higher probably of living in a stable partnership in outpatients with schizophrenia (Hofer et al., 2009). Poor ability to recognize emotion from facial expressions and vocal prosody has been related to communication and occupational dysfunction, problems with public appearance and behavior (Hooker & Park, 2002), and impoverished interpersonal relations even after adjusting for cognitive deficits and illness severity (Poole, Tobias, & Vinogradov, 2000). Poor ability to match emotional faces is related to poorer social functioning as measured by the Zigler score (Doop & Park, 2009). In a review of available literature examining the relationship between social cognition (including FAR) and functional outcome, Couture et al. (2006) reported moderate to large effect sizes for the relationship between emotion recognition and community functioning, social behavior in the milieu, and social skills. A more recent meta-analysis also reported medium to large positive correlations between emotion identification and functional outcomes in patients with schizophrenia and schizoaffective disorder in domains involving social problem solving, social skills, and community functioning (Irani, Seligman, Kamath, Kohler, & Gur, 2012). Furthermore, Fett et al. (2011) conducted a meta-analysis showing that social cognition (including FAR) was more strongly associated with community functioning than neurocognition in patients with schizophrenia.

Longitudinal studies have demonstrated that the relationship between FAR and social functioning is relatively stable over time. For example, Kee et al. (2003) reported that emotion recognition based on facial expressions and vocal prosody was related to work functioning and independent living at baseline and one-year follow-up. In addition, emotion recognition at baseline predicted work functioning and independent living at
follow-up. It is interesting to note that in a sample of healthy adolescents and adolescents with personality disorders (including schizotypal, paranoid, and schizoid personality disorders), poor ability to recognize emotion based on facial expression and vocal prosody was related to more social maladjustment as rated by parents 12-18 months later (Wickline et al., 2012). Addington et al. (2006) demonstrated that FAR and social functioning were related and impaired in both first episode and multiple episode patients with schizophrenia compared to controls. Despite improvements in symptoms one year later, both patient groups were still impaired in FAR and social functioning. However, a more recent study suggests that the relationship between FAR dysfunction and social impairment may change with disease progress. Pan, Chen, Chen, & Liu (2009) reported that FAR was not related to social functioning in acute remitting inpatients but was related to functioning in stable outpatients. Furthermore, in these chronic patients FAR deficits contributed independently to impaired functioning even after adjusting for education, neurocognition, IQ, and symptoms.

Beyond just accuracy of FAR performance, Cohen et al. (2009) reported an interesting relationship between response bias and functioning. Specifically, the misattribution of anger to other emotional faces (i.e., a negative bias) was associated with poorer social and global functioning in patients, misattribution of happiness (i.e., a positive bias) was associated with better social functioning, and misattribution of shame was associated with better global functioning (Cohen et al., 2009).

While there is some evidence that neurocognition is inversely related to functioning (see Addington & Addington, 1999; Addington & Addington 2000), a number of authors have proposed that FAR may act as a mediator in the relationship...
between neurocognitive performance and social functioning in schizophrenia. To date, several studies have supported this hypothesis both at baseline and at one-year follow-up (Brekke, Kay, Lee, & Green, 2005a; Addington et al. 2006; for review see Couture et al., 2006). While cognitive remediation may help ameliorate social impairments, specific social cognitive training may be beneficial as well. A recent meta-analytic review of 19 studies using behavioral training programs to improve social cognitive functioning (including FAR) in patients with schizophrenia demonstrated a moderate to large effect size for improved ability to identify facial expressions ($d = 0.71$) and a large effect size for improved ability to discriminate facial expressions ($d = 1.01$), as well as a large effect size for observer-rated community and institutional functioning after training ($d = 0.78$) (Kurtz & Richardson, 2012). The potential to ameliorate both FAR and social functioning deficits in patients with schizophrenia is particularly exciting given the recent interest in (and need for) preventive interventions for individuals at risk for developing psychosis who also show FAR and social functioning deficits, albeit attenuated ones (see Statucka & Walder, 2013).

It should be noted, however, that some authors have argued that the self-report questionnaires, clinical interviews, and laboratory-based tests of social skills and social functioning typically used to date have low ecological validity. Using an ecologically valid experience sampling method to measure social functioning, Janssens et al. (2012) reported that there was no association between FAR ability and social functioning in daily life in either patients or controls. These finding highlight the importance of measurement selection when examining complex constructs such as social functioning.
Research findings with at-risk individuals have not uniformly paralleled the relationship between FAR and social functioning demonstrated in patient populations by most of the research to date. In one study examining FAR and social skills in UHR individuals, patients who had been diagnosed with schizophrenia less than 5 years ago, and more chronic patients, Pinkham et al. (2007b) reported that the UHR group did not differ from healthy controls on a widely used FAR task (the Face Emotion Identification Task; Kerr & Neale, 1993). Both patient groups performed significantly worse on the FAR task than the UHR and control groups, leading the authors to propose that FAR deficits may only appear after illness onset and remain stable thereafter. With regards to social skills, the UHR group and both patient groups performed significantly worse on a conversation probe role-play task compared to controls; however, the UHR group performed significantly better than the chronic patients. Importantly, though FAR performance was unable to predict conversion to psychosis one year later, UHR individuals who converted performed significantly worse on the social skills task at baseline than those who did not convert.

Similarly, using a psychometric at-risk sample, Jahshan & Sergi (2007) found that individuals with a high degree of schizotypal traits as measured by the brief version of the SPQ (Raine & Benishay, 1995) did not show impaired emotion recognition from facial expressions, voice tones, and gestures when compared to individuals with low SPQ scores. The high SPQ group, however, was impaired in academic, peer relationship, and family relationship functioning and their degree of impairment was comparable to that reported for patients with depression and schizophrenia. It should be noted that Jahshan & Sergi (2007) used a social cognition task that has been demonstrated to be sensitive to
deficits after traumatic brain injury but had never been used with individuals with SSDs and may therefore lack sensitivity to detect deficits in this population. Using a different measure of emotional intelligence that has been previously used with SSD patients and was recently incorporated into a test battery designed for use in clinical trials of schizophrenia (MATRICS Consensus Cognitive Battery; Nuechterlein & Green, 2006) in a sample derived from the same population as Jahshan & Sergi (2007), Aguirre et al. (2008) did report the expected deficits in emotion recognition based on photos of faces, landscapes, and art in the high SPQ group. Moreover, in the high SPQ group impaired ability to recognize emotions was associated with poor peer relationship functioning. Finally, Pelletier et al. (2013) identified individuals at high psychometric risk using subclinical hallucinations and reported that these individuals had poorer social and role functioning as measured by Cornblatt et al.’s (2007) instrument, and performed more poorly on the Penn Emotion Recognition Test at trend level and significantly more poorly on fear faces than low-risk individuals. Also, in the high-risk group, FAR performance was positively correlated with social and role functioning while performance on fear faces alone was positively correlated with role functioning (Pelletier et al., 2013).

In summary, examination of the relationship between FAR impairments and social functioning in at-risk populations is limited and preliminary findings are inconclusive as the number of studies reporting that FAR deficits are related to social functioning (Aguirre et al., 2008; Wickline et al., 2012; Pelletier et al., 2013) are nearly matched in number by those reporting no relationship between the two variables in high-risk groups (Jahshan & Sergi, 2007; Pinkham et al., 2007b). However, there is clear evidence to support the relationship between FAR impairments and poor social
functioning in schizophrenia patients. Given the relationship among FAR, psychotic symptoms, and social functioning and that FAR appears to be necessary for successful social interactions (Riggio, 1992), FAR may play a mediating role in the relationship between schizotypal traits and social functioning. To date, however, we are unaware of any studies examining the potential mediating role of FAR deficits in this relationship. Research aimed at filling this gap in the literature holds promise towards 1) better understanding basic perceptual impairments that may accompany psychosis risk, 2) providing clues as to the underlying neural substrates of these impairments, and 3) better understanding how cognitive disruptions may subserve social functioning impairments.

Summary

There is significant evidence of impaired ability to recognize facial affect in patients with schizophrenia (for review see Kohler et al., 2010; Chan et al., 2010). Moreover, similar though attenuated deficits in the accuracy of FAR have been demonstrated in first- and second-degree relatives of patients (e.g., Eack et al., 2010, Lavoie et al., 2013), patients with SPD (e.g., Waldeck & Miller, 2000), and individuals at clinical (e.g., van Rijn et al., 2011) and psychometric high risk (e.g., Brown & Cohen, 2010; Germine & Hooker, 2011) for developing a SSD. Impairment in FAR accuracy is most commonly associated with severity of negative symptoms (for review see Chan et al., 2010) and both FAR impairment and negative symptomatology may reflect amygdala dysfunction associated with SSDs (e.g., Lepage et al., 2011).

Less well understood is the existence of slowed reaction time and negative bias during FAR performance in SSDs. There is some evidence of slower reaction time in patients (e.g., Calkins et al., 2010) and their relatives (e.g., Eack et al., 2010), however,
research in at-risk populations to date has been limited and the findings inconclusive. It may be that slowed reaction time is not present in at-risk individuals and only becomes apparent after disease onset or perhaps slowed reaction time is only apparent among high-risk individuals who later convert to psychosis. The existence of a negative bias during FAR performance in SSDs is more established. Though a negative bias has been demonstrated in patients (e.g., Kohler et al., 2003), their relatives (e.g., Eack et al., 2010), UHR individuals (e.g., van Rijn et al., 2011), and psychometrically at-risk individuals (e.g., Brown & Cohen, 2010), these studies are limited in number and do not address the role of comorbid depressive symptoms in FAR performance.

As with FAR deficits, impairments in social functioning have also been demonstrated in patients with schizophrenia (see Bellack et al., 1990) and these impairments appear to be related to negative symptoms (e.g., Gorna et al., 2008). In UHR individuals, presence of social impairments during the prodromal phase may help predict later conversion to psychosis (e.g., Cannon et al., 2008). Attenuated impairments in social functioning are also present in psychometrically at-risk individuals (e.g., Barrantes-Vidal et al., 2010). Given that 1) both FAR and social functioning impairments appear to be related to severity of negative symptomatology and 2) successful social interactions rely on accurate FAR (Riggio, 1992), FAR impairments in SSDs may at least in part account for the relationship between social functioning impairments and negative symptoms observed in these populations. While these impairments are most severe in patients with schizophrenia, their presentation may be affected by chronic treatment (with medication), hospitalization, and stigma. Therefore, examining similar – though attenuated – deficits in various at-risk populations may hold
potential for elucidating their true nature and etiology by minimizing these confounds, thereby revealing clues that may guide potential avenues for early identification, prevention, and treatment of SSDs.

**Specific Aims and Hypotheses**

Meehl (1962) proposed a dimensional model of psychosis whereby certain individuals may inherit a genetic mutation that predisposes them to schizophrenia, though without the proper constellation of environmental factors, they will never develop a full-blown psychotic disorder. Due to their genetic predisposition, these individuals may possess traits and experience deficits that are similar to those of patients with schizophrenia in an attenuated form. The study of these non-clinical (albeit at-risk) populations presents several methodological advantages. First, studies examining these samples hold promise for elucidation of the degree to which psychopathology lies along a continuum (e.g., Meehl, 1962), with implications for better understanding underlying etiology. Furthermore, when studying young non-clinical populations the possibility exists that some subjects may develop more serious symptoms or forms of psychopathology in the future. This would allow for early identification of important risk factors that may exacerbate existing subthreshold symptoms and point to underlying liability (in the context of longitudinal research). Secondly, studying non-clinical populations eliminates methodological concerns about the potentially confounding effects of psychotropic medications and hospitalization (which otherwise pose challenges when studying clinical populations) on dependent variables.

The specific aims of the proposed study are seven-fold and are as follows.
Specific Aim #1. To replicate findings of reduced accuracy of FAR in a non-clinical sample of late adolescents and young adults posited to be at psychometric high risk for psychosis.

Hypothesis #1: Based on Meehl’s dimensional model of schizotypy and given previous research in patients with schizophrenia (for review see Mandal et al., 1998; Edwards et al., 2002; Kohler et al., 2010; Chan et al., 2010) and those at familial (for review see Lavoie et al., 2013), clinical (Pinkham et al., 2007b; Addington et al., 2008a; van Rijn et al., 2011, Addington et al., 2012; Amminger et al., 2012a; Amminger et al., 2012b; Wölwer et al., 2012; Comparelli et al., 2013) and psychometric (van’t Wout et al., 2004; Williams et al., 2007; Aguirre et al., 2008; Brown & Cohen, 2010; Germine & Hooker, 2011; Roddy et al., 2012; Abbott & Green, 2013; Pelletier et al., 2013) risk for SSDs, it is predicted that there will be a main effect of group such that individuals with elevated SPQ scores who are believed to be at psychometric high risk for psychosis will be less accurate when asked to label emotionally expressive faces than individuals with few schizotypal traits.

Specific Aim #2. To examine whether individuals at psychometric high risk for psychosis are characterized by slower reaction time in response to a FAR paradigm.

Hypothesis #2: The relationship between schizotypal traits and FAR reaction time will be explored. This relationship may prove telling as there may be a significant speed-accuracy tradeoff such that though subtle performance deficits in the psychometrically at-risk population may be difficult to detect, significantly slower processing of emotional stimuli may be present and may be detrimental to social functioning. Generalized psychomotor slowing is well-established in patients with schizophrenia (for review see
Morrens et al., 2007). In individuals at psychometric risk, however, the current literature is mixed with some findings supporting slower reaction time in at-risk individuals during various cognitive tasks (see Lenzenweger, 2001; Asai et al., 2009) while others do not (see Gooding et al., 2006). Therefore, it is unclear whether psychometrically at-risk individuals demonstrated a generalized psychomotor slowing similar to that which has been evidenced in patients. Only recently have studies begun to examine reaction time during FAR tasks in patient and at-risk populations, including psychometrically at-risk samples. Currently, there is evidence that patients (Gur et al., 2002; Habel et al., 2010; Calkins et al., 2010; Li et al., 2010b) and their relatives (Eack et al., 2010; Calkins et al., 2010) respond significantly slower than healthy controls during FAR tasks. However, some research with schizophrenia patients (Fakra et al., 2008; Doop & Park, 2009), with UHR individuals (Seiferth et al., 2008), and with patients with SPD (Dickey et al., 2011) does not support this slowed reaction time and alternatively shows that these individuals did not differ from healthy controls in terms of speed of reaction time during FAR performance. In psychometrically at-risk individuals, Green et al. (2001) found that these individuals were significantly slower than controls only when identifying angry faces while Brown & Cohen (2010) did not find any evidence of slowed reaction time in psychometric at-risk subjects and in fact, reported that high SPQ-I scores were related to quicker reaction times. The dearth of research examining reaction time in psychometrically at-risk individuals precludes any directional hypothesis regarding this variable.

**Specific Aim #3.** To examine whether a negative bias in responding exists during FAR among individuals at psychometric high risk for psychosis.
Hypothesis #3: An increased likelihood to erroneously label neutral faces as negatively valenced emotions has been demonstrated in patients with schizophrenia (Kohler et al., 2003; Pinkham et al., 2011), as well as individuals at familial (Eack et al. 2010), clinical (van Rijn et al. 2011), and psychometric (Brown & Cohen, 2010) risk for developing SSDs. Therefore, it is predicted that there will be a main effect of group such that individuals with high scores on the SPQ who are presumed to be at psychometric high risk for psychosis will demonstrate a negative bias such that they will be significantly more likely to misattribute negative emotions when labeling neutral faces than individuals at psychometric low risk.

Specific Aim #4. To examine the relationships among FAR accuracy, speed, and bias with various dimensions of psychotic symptoms (e.g., positive, negative, disorganized schizotypal traits) and depressive symptomatology in the entire non-clinical college sample as well as in high-risk and low-risk groups.

Hypothesis #4: Findings in patients generally suggest that reduced accuracy on FAR tasks is related to negative symptomatology (Gaebel & Wölwer, 1992; Mueser et al., 1996; Addington & Addington, 1998; Mandal et al., 1999; Kohler et al., 2000; Edwards et al., 2001; Silver & Shlomo, 2001; Kohler et al., 2003; Herbener et al., 2005, Alfimova et al., 2009; Chan et al., 2010; Gilling McIntosh, 2011). Research examining this relationship in at-risk populations is currently limited. All studies that have examined the relationship between FAR accuracy deficits and schizotypal symptoms in psychometrically at-risk samples have reported a significant relationship between deficits and symptoms, however, the specific domain of symptoms found to be related to FAR deficits has varied between studies. Therefore, a relationship between FAR accuracy and
positive, negative, disorganized, and/or general schizotypal traits is hypothesized. Few studies to date have examined the relationships among FAR reaction time, FAR bias, and symptom domains, therefore, examination of these relationships will be exploratory.

**Specific Aim #5.** To examine the degree to which the relationship of psychotic-like traits with FAR negative bias in individuals at psychometric high risk for psychosis holds after adjusting for depressive symptoms.

Hypothesis #5: Negative bias is not exclusive to SSDs and has also been demonstrated in patients with MDD (Leppänen et al., 2004; Gollen et al., 2010; Milders et al., 2010; Naranjo et al., 2011). Given that SSDs and depression have high rates of comorbidity (Buckley et al., 2009) and that there is a large positive correlation between schizotypal traits and depressive symptoms (Fonseca-Pedrero et al., 2011), the observed negative bias in SSDs may be related to negative affect more generally. Therefore, it is predicted that when adjusting for negative affect/depressive symptomatology, while the relationship between high SPQ scores and negative bias will remain significant, the magnitude of this relationship will be significantly reduced, as negative affect/depressive symptomatology will account for a significant portion of the variance in the negative bias scores (see Kessler et al., 2007).

**Specific Aim #6.** To replicate findings of impaired social functioning in a non-clinical sample of late adolescents and young adults posited to be at psychometric high risk for psychosis.

Hypothesis #6: Given previous research in patients with schizophrenia (Bellack et al., 1990) and those at familial (Shim et al., 2008), clinical (Corcoran et al., 2003; Lencz et al., 2004; Yung et al., 2004; Cornblatt et al., 2007; Ballon et al., 2007; Cannon et al.,
and psychometric (Henry et al., 2008; Barrantes-Vidal et al., 2010; Fonseca-Pedrero et al., 2010; Sommer et al., 2010; Heber et al., 2011; Culianez et al., 2011; Simpson & Pinkham, 2011; Collip et al., 2013; Pelletier et al., 2013) risk for SSDs, it is predicted that there will be a main effect of group such that individuals with elevated SPQ scores who are believed to be at psychometric high risk for psychosis will be more impaired on social functioning than individuals with few schizotypal traits.

**Specific Aim #7.** To assess whether FAR accuracy acts as a mediator of the relationship between schizotypal traits and social functioning.

Hypothesis #7: As summarized above, there is evidence that psychometric risk for SSDs – specifically negative symptomatology – is associated with FAR accuracy deficits. There is also evidence that FAR accuracy deficits in patients with schizophrenia are associated with poor social functioning (Mueser et al., 1996; Poole et al., 2000; Hooker & Park, 2002; Kee et al. 2003; Brekke et al., 2005a; Cohen et al., 2006; Couture et al., 2006; Addington et al. 2006; Hofer et al., 2009; Pan et al., 2009). In a psychometric high-risk sample, Aguirre et al. (2008) have also demonstrated that individuals with high SPQ scores are less accurate on FAR tasks and have poor peer relationships and Pelletier et al. (2013) have demonstrated that individuals with sub-clinical hallucinations are less accurate on a FAR task and have poorer social and role functioning. Finally, there is evidence that symptomatology – typically negative symptoms – are related to social functioning in patients (Chaves et al., 1993; Blanchard et al., 1998; Dickerson et al., 1999; Addington et al., 2003; Voges & Addington, 2005; Gorna et al., 2008). While the current research is less clear regarding which symptom domains are related to social
functioning in at-risk populations, past research has consistently demonstrated a
significant relationship between symptomatology and functioning in UHR (Cornblatt et
al., 2003; Cornblatt et al., 2007; Svirskis et al., 2007; Shim et al., 2008; Corcoran et al.,
2011) and – to a lesser extent – in psychometrically at-risk (see Barrantes-Vidal et al.,
2010) individuals. Using the SFS and SPQ in non-clinical samples, a number of
researchers have demonstrated that total SFS score is negatively correlated with total
SPQ score (Henry et al., 2008) and that the SPQ-I subscale is negatively correlated with
the total SFS score (Henry et al., 2008) as well as the interpersonal communication and
social engagement subscales (Heber et al., 2011; Culianez et al., 2011). To date, we are
unaware of any studies examining whether FAR performance may act as a mediator of
the relationship between schizotypal traits and social functioning.

Based on the findings in patient and at-risk samples, it is hypothesized that: a)
more schizotypal personality traits (specifically SPQ-I) will be associated with more
impaired social functioning (specifically SFS total, interpersonal communication, and/or
social engagement), b) more schizotypal personality traits (specifically SPQ-I) will be
associated with less accurate FAR performance, and c) less accurate FAR performance
will be associated with more impaired social functioning (specifically SFS total,
interpersonal communication, and/or social engagement). Furthermore, it is proposed
that FAR accuracy may act as a mediator such that the relationship between negative
schizotypal traits and social functioning impairment (especially in the domains of
interpersonal communication and social engagement) will be significantly partially (if not
completely) accounted for by the indirect pathway from negative schizotypal traits to
FAR accuracy and FAR accuracy to social functioning domains.
CHAPTER II

METHODS

Participants

Nine-hundred and eighty-one (666 Female/315 Male) participants over the age of 18 years were recruited from Introduction to Psychology human subject pools at Baruch College, Brooklyn College, The City College of New York, John Jay College of Criminal Justice, Queens College, and York College of the City University of New York (CUNY) and received course credit as compensation. Individuals who were older than 34 years (n = 16) were excluded from the sample because they were statistical outliers (>3.0 SD above the mean) and because previous research has shown that the peak incidence for schizophrenia in males is between 20 and 24 years and between 29 and 32 years in females (see Stilo & Murray, 2010 for review). Therefore, individuals who are older than 34 years are unlikely to convert to SSDs in the future. Individuals who reported that they do not have normal or corrected-to-normal vision (n = 56) were also be excluded from all analyses involving the FAR task as intact vision is required for this task. In an effort to ensure data reliability, participants with 3 or more randomly answered items on the CIS (n = 108) and those who did not complete the CIS items (n = 7) were excluded from the final sample. Following all of these exclusions, the final sample consisted of 850 participants (584 Female/266 Male).

Based on enrollment during the Fall 2012 semester, across CUNY senior colleges approximately 57.5% of students are expected to be female and 42.5% are expected to be male (CUNY Office of Institutional Research and Assessment, 2013). Furthermore, 30.4% of students are expected to be White, 24.6% Black/African American, 23.6%
Hispanic, 21.2% Asian/Pacific Islander, and 0.2% American Indian/Native Alaskan (CUNY Office of Institutional Research and Assessment, 2013).

Based on previous research, the base rate for schizotypy or psychometric high risk in an undergraduate population is estimated to be .10 (Lenzenweger & Korfine, 1992). Using the SPQ, individuals scoring 41 points or higher are in the top 10% of the distribution of this scale and of these individuals, 55% obtain a diagnosis of SPD based on a semi-structured clinical interview (Raine, 1991). Conversely, individuals obtaining a score of 12 or less are in the bottom 10% of the SPQ distribution and considered to be at low psychometric risk for psychosis (Raine, 1991). Therefore, using these cut-off scores and based on the sample of 850 participants, approximately 85 individuals are expected to be in the psychometrically high-risk group while 85 are expected to be in the psychometrically low-risk group.

Some previous research has demonstrated that 3 out of 5 adolescent participants with a diagnosis of SPD at baseline developed an Axis I psychotic disorder at 5-year follow-up (Walker et al., 2010). Based on previous longitudinal research of college undergraduates identified as psychometrically at high risk (based on the Chapmans’ Social Anhedonia Scale), approximately 24% where diagnosed with SSDs (i.e., schizophrenia, psychosis NOS, SPD, schizoid personality disorder, paranoid personality disorder) at 10-year follow-up as compared to only 1% of a control group (Kwapil, 1998). A more recent 5-year follow-up study, found that psychometrically high-risk individuals (as identified by the Social Anhedonia Scale) had significantly elevated rates of avoidant, schizotypal, and paranoid personality disorders compared to those at low risk (Gooding, Tallent, & Matts, 2005, 2007), leading the authors to conclude that the
psychometric high-risk method is a viable strategy for identifying individuals at risk for the later development of SSDs.

**Procedures**

Participants signed-up for the study via the CUNY Sona System and received an email containing a link to the study website and a username and password to log on to the study website. All participants gave written informed consent (via on-line website) before commencement of study procedures. The study procedures were approved by the Institutional Review Board of Brooklyn College, CUNY.

Following informed consent procedures, participants were asked to provide basic demographic information (e.g., age, sex, handedness, race/ethnicity). Next, participants completed a computerized task assessing FAR accuracy, reaction time, and bias. Finally, participants were administered a valid and reliable self-report measure commonly used to examine schizotypal personality traits in general populations as well as self-report measures to assess social functioning and depressive symptoms. All data collection was completed on a password-protected website, remotely via the internet.

A number of steps were taken to ensure integrity (e.g., validity) of data given it was collected remotely via the internet. Firstly, the email initially sent to participants contained pre-emptive troubleshooting points and a description of optimal conditions for study completion (e.g., quiet space, minimal distractions and interruptions). Secondly, the optimal conditions were repeated after the consent process. Thirdly, participants were notified that if they chose to volunteer for the study, they must complete the study during one session lasting approximately one-hour. Accordingly, once participants began the study they were not be able to log off and log on to the website at a later time to complete
the study. Fourthly, items from the Chapman Infrequency Scale (CIS; Chapman & Chapman, 1983) were presented between self-report measures in order to detect those participants who responded randomly, pseudorandomly or dishonestly. Participants with 3 or more randomly answered items on the CIS (n = 108) and those who did not complete the CIS items (n = 7) were excluded from the final sample based on criteria used in prior studies (see Chmielewski, Fernandes, Yee, & Miller, 1995; Kerns, 2005, 2006; Kwapił, Barrantes-Vidal, & Silvia, 2008; Fonseca-Pedrero et al., 2009, 2011). This procedure ensured reliability of data collected and analyzed. Finally, based on total time taken to complete the FAR task, one participant’s performance was considered invalid because they required over one hour to complete the FAR task. This one participant was excluded from all analyses involving the FAR task. Participants who were statistical outliers (>3.0 SD above the mean) on the total time taken to complete the FAR task (n = 13) were likewise excluded from all relevant analyses involving the FAR task because these participants were perhaps distracted or otherwise not fully engaged in the FAR task, rendering validity of these data questionable.

Materials

**Schizotypal Personality Questionnaire (SPQ).** The SPQ (Raine, 1991) is a 74-item dichotomous (yes-no) self-report questionnaire. It may be used to examine schizotypal personality traits in normal populations or screen for predisposition to SPD and potentially other SSDs in at-risk populations. The SPQ statements are derived from the Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R) diagnostic criteria for SPD. Previous research has shown that in a sample of 195 college undergraduates, 55% of individuals scoring in the top 10% of SPQ total scores had a
DSM-III-R clinical diagnosis of SPD as assessed by the Structured Clinical Interview for DSM Disorders. Individuals who obtained a SPQ total score of 41 or higher were in the top 10% of the distribution (Raine, 1991). In the present study, participants scoring 41 or higher were considered the psychometrically high-risk group, while those scoring at or below the 10th percentile (i.e., cutoff score of 12) were considered the psychometrically low-risk group.

In addition to the total SPQ score, the measure contains nine subscales reflecting the DSM-III-R SPD criteria (i.e., ideas of reference, excessive social anxiety, odd beliefs or magical thinking, unusual perceptual experiences, odd or eccentric behavior, no close friends, odd speech, constricted affect, and paranoid ideation or suspiciousness). Furthermore, three factor scores can be derived by summing of subscale scores: Cognitive-perceptual (SPQ-CP; ideas of reference, odd beliefs or magical thinking, unusual perceptual experiences, and paranoid ideation/suspiciousness); Interpersonal (SPQ-I; excessive social anxiety, no close friends, constricted affect, and paranoid ideation/suspiciousness); and Disorganized (SPQ-D; odd or eccentric behavior, and odd speech). The SPQ has been shown to have high internal reliability with Cronbach’s α internal consistency reliability coefficients for the nine subscales ranging from 0.71 to 0.78 (mean = 0.74) and with a total scale reliability of 0.91 (Raine, 1991). The measure has also been shown to have good criterion validity as demonstrated by correlations between the SPQ total score and clinical interview measures of SPD ($r = 0.68$) as well as between all nine subscales and Structured Clinical Interview schizotypal scores ($r$ range = 0.55 to 0.80, mean $r = 0.65$; Raine, 1991). Correlations between SPQ scores and convergent validity scales were high ($r = 0.65$ to 0.81) while correlations between SPQ
scores and discriminant validity scales were significantly lower than for convergent scales \((r = 0.19 \text{ to } 0.37; \text{Raine, 1991})\). Lastly, the SPQ has also demonstrated good two-month test-retest reliability \((r = 0.82; \text{Raine, 1991})\).

**Social Functioning Scale (SFS).** The SFS (Birchwood et al., 1990) is a 79-item self-report measure specifically developed to be used with outpatients with schizophrenia. The scale has recently been used with UHR samples where it has been shown to be sensitive enough to distinguish UHR subjects from healthy controls (Addington et al., 2008b; Shim et al., 2008; Jang et al., 2011) as well as to distinguish UHR subjects who went on to convert to psychosis from those who did not (Jang et al., 2011). The SFS has also been successfully used with a mixed college and community sample where the SPQ total score and SPQ-I subscale score were significantly negatively correlated with the SFS total score (Henry et al., 2008). In addition to providing a total score, the scale measures abilities or performance in seven areas: 1) social engagement/withdrawal; 2) interpersonal communication/behavior; 3) prosocial activities; 4) recreation; 5) independence-competence; 6) independence-performance; and 7) employment/occupation. The seven subscales have differing means and variances, therefore, each raw score can be standardized and normalized using a T transformation to a mean of 100 and a standard deviation of 15 using an unemployed schizophrenia group as the reference population (Birchwood et al., 1990). Given that an unemployed patient population may not be a relevant comparison group for the healthy college sample used in the present study, raw SFS scores were used in all analyses. The SFS has been shown to have good internal consistency with coefficient alphas ranging from 0.69 to 0.87 for the seven subscales and 0.80 for the total scale (Birchwood et al., 1990). This was
confirmed by a more recent study utilizing the Norwegian version of the SFS which reported that Cronbach’s α coefficients for the subscales ranged from 0.60 to 0.88 and reported a total scale reliability of 0.81 (Hellvin et al., 2010). A factor analysis yielded a single, powerful factor that accounted for 57% of the variance and this factor loaded uniformly and strongly across all subscales, suggesting good construct validity (Birchwood et al., 1990). Finally, schizophrenia outpatients were strongly differentiated from a community sample by the SFS and scores correlated with the presence of negative ($r = -0.44$) and positive ($r = -0.46$) symptoms in patients (Birchwood et al., 1990).

Recently, Heber et al. (2011) as well as Culianez et al. (2011) reported that in a sample of undergraduate students the SPQ-I subscale was negatively correlated with all of these SFS domains but most strongly with the interpersonal communication ($r = -0.50, p < .001$) and the social engagement scales ($r = -0.45, p < .001$). Simpson & Pinkham (2011) reported that undergraduate students with high levels of subclinical paranoia had significantly lower scores on the independence-competence, social engagement, and interpersonal communication scales than participants with low levels of paranoia.

**Center for Epidemiologic Studies Depression Scale (CES-D).** The CES-D (Radloff, 1997) is a 20-item self-report measure that was designed to measure depressive symptomatology in the general population. The measure screens for depressive feelings and behaviors rated on a 4-point Likert scale (i.e., Rarely or none of the time (less than 1 day per week) to Most or all of the time (5-7 days per week)). The CES-D has been demonstrated to have very high internal consistency in the general population (coefficient alpha = 0.85) and in psychiatric patients (coefficient alpha = 0.90). CES-D scores discriminate well between psychiatric inpatients and individuals from the general
population and are correlated with clinician ratings and other self-report measures of depression (Radloff, 1977). Test-retest reliability is adequate (mean $r = 0.57$) given that the CES-D is designed to measure current depressive symptomatology, which is expected to vary over time (Radloff, 1977). The CES-D has also been used with non-clinical community adult (see Gurpegui et al., 2009; Knight, Avery, Janssen, & Powell, 2010; Morin et al., 2011), elderly (see Gerst, Al-Ghatrif, Beard, Samper-Ternent, & Markides, 2010; Zhang et al., 2011), adolescent (see Hales et al., 2006; Anderson et al., 2011), and college samples (see Herman et al., 2011). For example, in nonclinical undergraduate samples, some researchers have demonstrated that maladaptive eating practices were correlated with CES-D scores and were predictive of depression among female students (Hawkins, McDermott, Seeley, & Hawkins; 1992) while others have shown that CES-D scores were related to sleep problems among female students (Regestein, et al., 2010).

**Chapman Infrequency Scale (CIS).** The CIS (Chapman & Chapman, 1983) is a 13-item dichotomous (true/false) measure used to screen out participants who respond in a random, careless, or invalid manner. In keeping with previous research (see Chmielewski et al., 1995; Kerns 2005, 2006; Kwapił et al., 2008; Fonseca-Pedrero et al., 2009, 2011), participants who endorsed three or more items or who did not complete the CIS items were excluded from the final sample. This was done to ensure validity of responses for administered self-report questionnaires.

**Karolinska Directed Emotional Faces (KDEF).** The KDEF (Lundqvist, Flykt, & Öhman, 1998) is a large electronic database containing colored photographs of 35 female and 35 male models, each displaying six different emotional expressions and no emotional expression (neutral), and each expression being photographed from five
different angles. Only photographs for actors looking straight at the camera were used in the present study. Selection of stimuli for this study is based on previous research using the KDEF by Calvo & Lundqvist (2008), Adolph & Alpers (2010), and Gilling McIntosh (2011). Participant were shown 64 faces (8 angry, 8 disgusted, 8 fearful, 8 happy, 8 sad, 8 surprised, and 16 neutral) one at a time. Participants were asked to identify the emotion expressed by the face by selecting with a mouse the word describing the emotion from seven answer choices (i.e., happy, sad, anger, fear, disgust, surprise, and no emotion). Stimuli were balanced for model’s gender (four female and four male faces for each emotion, eight female and eight male faces for neutral). Scores on this task are based on the number of correct responses for each emotion (anger, disgust, fear, happy, sad, surprised, neutral) and the total correct responses as well as the response times for each emotion and the total time taken to complete the task. Bias scores were calculated by summing the number of times neutral faces was labeled as angry, disgusted, happy, frightened, sad, or surprised. Negative bias scores were calculated by summing anger, disgust, fear, and sad biases.

The KDEF photographs have been validated on emotional content, intensity, and arousal and have good test-retest reliability (i.e., 87.96%; Goeleven, De Raedt, Leyman & Verschuere, 2008). They have been successfully used to assess FAR in healthy college samples (Calvo & Lundqvist, 2008; Adolph & Alpers, 2010) and in patients with schizophrenia (Fakra et al., 2008; Doop & Park, 2009; Larøi et al., 2010; Gilling McIntosh, 2011), and were recently used in an online study to assess FAR in individuals with autism and those at familial high risk for autism (Sucksmith, Allison, Baron-Cohen, Chakrabarti, & Hoekstra, 2013). The KDEF appears to be free from ceiling effects in
healthy controls as mean percent correct for each emotion are as follows: happy 97.4%, neutral 97.4%, angry 89.3%, sad 87.2%, disgust 87.4%, surprise 86.2%, and fear 79.3% (Calvo & Lundqvist, 2008).

No data is currently available regarding the reliability and/or validity of administering the KDEF remotely via the Internet, however, there is a growing body of literature to suggest that on-line administration of questionnaires (Davis, 1999; Riva, Teruzzi, & Anolli, 2003; Gosling, Vazire, Srivastava, & John, 2004) and cognitive tasks (Haworth et al. 2007; Silverstein et al., 2007; Man, Chung, & Mak, 2009; Crump, McDonnell, & Gureckis, 2013) yields reliable and empirically valid results comparable to those obtained in traditional laboratory settings.

Statistical Analyses

Participants who scored 41 or higher on the SPQ were included in the psychometrically high-risk group, whereas those who scored at or below 12 were included in the psychometrically low-risk group. These cutoff scores are based on previous literature showing that individuals who score 41 points or higher on the SPQ are significantly more likely to be diagnosed with a SSD than those scoring 12 points or less (Raine, 1991). Two outliers were identified on the SPQ total (>3.0 SD above mean). However, as these individuals were not statistical outliers on other measures of interest and did not fundamentally differ from the rest of the sample in terms of demographic characteristics, they were included in all subsequent analyses involving the SPQ.

The SPQ total score and SPQ-CP, SPQ-I, SPQ-D subscale scores, CES-D total score, KDEF bias scores, KDEF reaction times, and SFS recreation and SFS prosocial scores were positively skewed. KDEF accuracy scores (except fear accuracy), SFS social
engagement/withdrawal, interpersonal communication, independence-performance, independence-competence, and occupation/employment scores were negatively skewed. SFS total score and KDEF fear accuracy were normally distributed. Box-Cox transformations were performed on all non-normally distributed measures (Osborne, 2010). However, SFS social engagement/withdrawal, interpersonal communication, independence-performance, independence-competence, and occupation/employment scores, as well as KDEF accuracy, sad reaction time, and bias scores could not be transformed in a satisfactory manner using this approach. Therefore, analyses involving these variables were conducted using non-parametric tests of difference and correlation.

After removing statistical outliers (<-3.0 SD below the mean), there was very little variability of scores on KDEF happy accuracy, due to a ceiling effect (i.e., no scores >3.0 SD above the mean). This is in keeping with KDEF norms produced by Calvo & Lundqvist (2008), where happy faces were correctly labeled 97.4% of the time and also with previous research where both patients with schizophrenia and controls identified happy faces almost perfectly across trials (see Leung et al., 2011). Similarly, after removing statistical outliers (>3.0 SD above the mean), no one mislabeled neutral faces as disgusted or surprised and only very rarely did anyone mislabel neutral faces as frightened. Again, this is in keeping with Calvo & Lundqvist’s (2008) KDEF norms where no one mislabeled the neutral faces chosen for this study as disgust, afraid, or surprised. Finally, given the nature of the sample, all participants were part- or full-time students. Consequently, separate analyses to address group differences or correlations involving KDEF happy accuracy, disgust bias, fear bias, and surprised bias and SFS employment/occupation were not conducted.
Statistical Analyses for Hypotheses #1, #2, and #3. Mann-Whitney U tests were used (given non-normal distribution of data) to examine differences between low-risk and high-risk groups with regard to total KDEF accuracy, anger, disgust, sad, surprised, and neutral accuracy, total negative bias, anger bias, happy bias, and sad bias (all one-tailed, given directional nature of hypotheses), and sad reaction time (two-tailed). Independent sample t-tests were used to examine differences between low-risk and high-risk groups with regard to KDEF fear accuracy (one-tailed, given directional nature of hypotheses) and reaction times (two-tailed). An alpha level of .05 was used for all comparisons; however, because a large number of comparisons were conducted, post hoc Holm-Bonferroni correction was implemented to control for multiple comparisons. Based on means and standard deviations from a sample of healthy college students where high and low SPQ cutoffs of 42 and 9 were used, respectively (Gassab et al., 2006), an a priori power analysis indicated that a group sample size of 10 subjects would achieve 100% power to detect a medium effect size when employing an alpha level of .05 using a two-tailed, two-sample t-test and a one-tailed, two-sample t-test.

Statistical Analyses for Hypothesis #4. Spearman rho correlations were used to examine the relationships among KDEF accuracy scores (except fear accuracy) and KDEF bias scores with various factors of the SPQ and CES-D scores in the entire sample, in the high-risk group alone, and in the low-risk group alone (using one-tailed tests, given directional nature of hypotheses). Pearson r correlations were used to examine the relationships among KDEF fear accuracy with various factors of the SPQ and CES-D scores in the entire sample, in the high-risk group alone, and in the low-risk group alone (using one-tailed tests given directional nature of hypotheses). Pearson r correlations
were used to examine the relationships among FAR reaction times (except sad reaction time) with various subscales and factors of the SPQ and CES-D scores in the entire sample, in the high-risk group alone, and in the low-risk group alone (two-tailed tests). Spearman rho correlation was used to examine the relationship between sad reaction time and various SPQ subscales and the CES-D in the entire sample, in the high risk-group alone, and in the low-risk group alone (two-tailed tests). An alpha level of .05 was used for all correlations, however, because a large number of correlations were evaluated, post hoc Holm-Bonferroni correction was implemented to control for multiple comparisons. An a priori power analysis indicates that a sample size of 500 will achieve 100% power to detect a difference of 0.300 (similar to the relationships reported by Brown & Cohen; 2010) when employing an alpha level of .05 using a two-tailed correlation while a sample size of 50 will achieve 57% to detect such a difference when employing an alpha level of .05 using a two-tailed correlation.

**Statistical Analyses for Hypothesis #5.** Partial Spearman rho correlations were conducted to assess whether significant relationships among SPQ scores and KDEF bias scores were significantly reduced when adjusting for CES-D scores (using one-tailed tests given directional nature of hypotheses).

**Statistical Analyses for Hypothesis #6.** An independent sample t-test was used to examine group differences (low-risk vs. high-risk) with regard to total SFS score (using one-tailed test given directional nature of hypothesis). A MANOVA was used to examine differences between low-risk and high-risk groups on the various subscales of the SFS (using a one-tailed test given the directional nature of the hypothesis).
Statistical Analyses for Hypothesis #7. Mediation models propose a three-variable chain where the mediator creates an indirect pathway between the independent variable (IV) and dependent variable (DV) whereby the mediator partially or fully explains the direct relationship between the IV and DV. The proposed mediation model was tested using Baron & Kenny’s (1986) statistical procedures for testing mediation. Accordingly, first a series of hierarchical multiple regression analyses was conducted using the entire sample to determine the relationship of the IV and the mediator with the DV. Second, the Sobel test was implemented to assess the mediator model, based on results of the regressions analyses. Specifically, hierarchical regression analyses were implemented to demonstrate that a) variations in schizotypal symptoms significantly account for variations in FAR accuracy (Path A), b) variations in FAR accuracy significantly account for variations in social functioning (SFS total, interpersonal communication, and/or social engagement) (Path B). Then, when both of these relationships (a and b) are adjusted for, a significant relationship between schizotypal symptoms and social functioning (SFS total, interpersonal communication, and/or social engagement) is no longer significant or is significantly reduced (Path C). See Figure 1.

Hierarchical multiple regression using the Enter method were used to calculate unstandardized regression coefficient and the standard error of these coefficients. To calculate the coefficient from SPQ score to FAR accuracy first any significant covariates were entered in Block 1, then the SPQ score was entered into Block 2 with the FAR accuracy score as the DV. To calculate the coefficient from FAR accuracy to SFS subscale (SFS total, interpersonal communication, and/or social engagement), again relevant covariates were entered into Block 1 and SPQ score and FAR accuracy score
were entered into Block 2 with the SFS subscale (SFS total, interpersonal communication, and/or social engagement) as the DV.

Once these regressions were calculated, the unstandardized regression coefficient for the association between SPQ score and FAR accuracy as well as the standard error of this coefficient and the unstandardized coefficient for the association between FAR accuracy and SFS total score, interpersonal communication, and/or social engagement subscale scores (when SPQ score is also a predictor) as well as the standard error of this coefficient were entered into the equation for the Sobel test (Baron & Kenny, 1986) in order to determine whether the indirect effect of SPQ score on SFS total score, interpersonal communication, and/or social engagement subscale scores through the mediator variable (FAR accuracy) is significant:

\[
\sqrt{\frac{b^2 s_a^2 + d^2 s_b^2 + s_a^2 s_b^2}{s_a^2 + s_b^2}}
\]

According to Fritz & MacKinnon (2007), depending on the magnitude of the correlation between the IV and the mediator and the magnitude of the correlation between the mediator and the DV, a sample size between 402 and 562 is necessary to obtain 0.8 statistical power using Baron & Kenny’s statistical procedures for partially mediated models.
CHAPTER III
RESULTS

Demographics

A Mann-Whitney U test was conducted to determine whether the groups significantly differed with respect to age. Categorical demographics, such as gender, ethnicity, and handedness, were examined using chi-square analysis to assess group differences (psychometric high- versus low-risk). There were no significant group differences on demographic characteristics including age, gender, race/ethnicity, and handedness (see Table 1). Thus, subsequent analyses were conducted without covarying for demographic variables.

Group Differences on KDEF (Hypotheses #1, #2, and #3)

Because a large number of comparisons were evaluated, post hoc Holm-Bonferroni correction was implemented to control for multiple comparisons.

Accuracy. Accuracy performance on the KDEF is summarized in Table 2. Between group comparisons indicated that individuals at high psychometric risk performed significantly worse than those at low psychometric risk on overall KDEF accuracy ($p = .004, d = -.360$). Examining each of the individual emotions and neutral, this group difference appeared to be driven by significantly worse ability to recognize neutral faces ($p = .003, d = -.409$) in the high-risk group compared to the low-risk group, as there were no other significant group differences in KDEF accuracy.

Reaction Time. High psychometric risk and low psychometric risk groups did not significantly differ on overall time taken to complete the KDEF nor on mean reaction time for individual emotions or neutral (see Table 3).
Negative Bias. Bias on the KDEF is summarized in Table 4. Between group comparisons indicated that individuals at high psychometric risk were significantly more likely to label neutral faces as negative emotions (i.e., negative bias) than individuals at low psychometric risk ($p = .007$, $d = .332$). Furthermore, those at high psychometric risk were significantly more likely than those at low psychometric risk to label neutral faces as sad ($p = .003$, $d = .358$). High- and low-risk groups did not differ in anger bias or happy bias.

In summary, individuals at high psychometric risk were significantly less accurate overall on the KDEF and less accurate at labeling neutral faces, and they were also significantly more likely to misattribute negative emotions and sadness to neutral faces than low-risk individuals. The two groups did not differ in terms of reaction time.

Correlations among KDEF, SPQ, and CES-D (Hypothesis #4)

Because a large number of correlations were evaluated, post hoc Holm-Bonferroni correction was made to control for multiple comparisons.

See Table 5 for correlations among KDEF and SPQ total and subscales in the entire sample. In the entire sample, SPQ total score was significantly negatively correlated with KDEF total accuracy ($p = .001$). SPQ-CP subscale was significantly negatively correlated with KDEF total accuracy ($p = .001$), sad accuracy ($p = .006$), surprised accuracy ($p = .006$), and neutral accuracy ($p = .001$). SPQ-CP was also significantly positively correlated with negative bias ($p = .002$). SPQ-D subscale was significantly negatively correlated with KDEF total accuracy ($p = .001$), fear accuracy ($p = .008$), and sad accuracy ($p = .010$). SPQ-D was also significantly negatively correlated
with disgust reaction time ($p = .005$). SPQ-I subscale was not correlated with any KDEF variables in the entire sample.

Furthermore, in the entire sample, CES-D was significantly negatively correlated with KDEF total accuracy ($r_s(785) = -.104, p = .002$) and neutral accuracy ($r_s(772) = -.097, p = .004$). CES-D was also significantly positively correlated with negative bias ($r_s(774) = .111, p = .001$) and sad bias ($r_s(779) = .096, p = .004$).

See Table 6 for correlations among KDEF and SPQ total and subscales in the high psychometric risk group. In the high-risk group, SPQ-CP subscale was negatively correlated with KDEF total accuracy ($p = .006$) and positively correlated with neutral reaction time ($p = .003$). SPQ total score, SPQ-I, SPQ-D, and CES-D were not correlated with any KDEF variables in the high-risk group.

See Table 7 for correlations among KDEF and SPQ total and subscales in the low psychometric risk group. In the low-risk group, SPQ total score was significantly negatively correlated with neutral accuracy ($p = .003$) and significantly positively correlated with negative bias ($p = .010$). SPQ-CP, SPQ-I, SPQ-D, and CES-D were not correlated with any KDEF variables in the low-risk group.

**Adjusting for CES-D in relationship between SPQ and KDEF negative bias**

**(Hypothesis #5)**

We proposed to explore whether adjusting for CES-D scores in the high psychometric risk group would attenuate the relationship between SPQ scores and KDEF negative bias. Within the high-risk group alone, however, SPQ total score nor any of the subscale scores were significantly correlated with KDEF negative bias; thus, adjusting for CES-D was not relevant to these analyses. However, within the entire sample, SPQ-CP
was significantly positively correlated with negative bias. Therefore, we explored whether CES-D attenuated this relationship within the entire sample.

The direct correlation between SPQ-CP and KDEF negative bias was significant \((r_{s}(780) = .103, p = .002)\). The partial correlation between SPQ-CP and KDEF negative bias, after covaring for CES-D, was no longer significant \((r_{s}(771) = .053, p = .069)\).

Given the aforementioned findings suggest that CES-D is a significant mediator of the relationship between SPQ and negative bias, we explored this relationship further using a more stringent method including multiple regression and a Sobel test, in line with the strategy proposed to address Specific Aim #7/Hypothesis #7 (see model illustrated in Figure 2). In the previous section it was shown that there was a significant relationship between SPQ-CP and negative bias (Path C), and between CES-D and negative bias (Path B), in the entire sample. Furthermore, in the entire sample, SPQ-CP and CES-D were significantly positively correlated \((r(842) = .442, p = .000)\) (Path A). Therefore, all three pathways in the mediation model were significant and the assumption for testing a mediation model was met. SPQ-CP was a significant predictor of CES-D \((p = .000)\) and SPQ-CP and CES-D were significant predictors of KDEF negative bias \((p = .001;\) see Table 8 and Figure 2). A Sobel test revealed that CES-D was a significant mediator of the relationship between SPQ-CP and KDEF negative bias in the entire sample (Sobel test = 2.44, \(p = .007\)).

**Group Differences on SFS (Hypothesis #6)**

Between group comparisons indicated that individuals at high psychometric risk \((M = 132.55, SD = 22.602)\) reported significantly worse overall social functioning \((t(265) = -8.282, p = .000, d = -1.072)\) than those at low psychometric risk \((M = 154.75, SD =\)
Examining the SFS subscales, there was a statistically significant difference in social functioning based on risk status ($F(6,239) = 30.414, p = .000$; Pillai’s Trace = .433). The high-risk group reported significantly worse social engagement ($p = .000, \eta_p^2 = .255$), interpersonal communication ($p = .000, \eta_p^2 = .269$), independence-performance ($p = .000, \eta_p^2 = .086$), prosocial behavior ($p = .000, \eta_p^2 = .072$), and independence-competence ($p = .000, \eta_p^2 = .215$; see Table 9). Groups did not significantly differ on the SFS recreation subscale.

**KDEF accuracy mediating the relationship between SPQ and SFS (Hypothesis # 7)**

In the entire sample, there were significant direct relationships between the SPQ total score and subscale scores and the SFS total score and subscale scores, except SPQ-CP was not correlated with SFS recreation (see Table 10). In the previous section it was shown that there was a significant relationship between SPQ total score, SPQ-CP, and SPQ-D and KDEF total accuracy in the entire sample. Lastly, KDEF total accuracy was significantly positively correlated with the SFS interpersonal communication subscale (see Table 11), however, this result did not remain significant after Holm-Bonferroni correction. Nonetheless, we concluded that all three pathways in the mediation model were significant and the assumption for testing a mediation model was met.

See Table 12 for summary of the first model. SPQ total score was a significant predictor of KDEF total accuracy ($p = .002$) and SPQ total score and KDEF total accuracy together were significant predictors of SFS interpersonal communication ($p = .000$). However, KDEF accuracy alone was not a significant predictor of SFS interpersonal communication after accounting for SPQ total score ($t(773) = 1.221, p = .112$). A Sobel test revealed that KDEF total accuracy was not a significant mediator of
the relationship between SPQ total score and SFS interpersonal communication (Sobel test = -1.129, \( p = .129 \)).

See Table 13 for summary of the second model. SPQ-CP was a significant predictor of KDEF total accuracy (\( p = .001 \)) and SPQ-CP score and KDEF total accuracy together were significant predictors of SFS interpersonal communication (\( p = .000 \)). KDEF accuracy alone was a predictor of SFS interpersonal communication at trend level after accounting for SPQ-CP (\( t(773) = 1.651, p = .050 \)). A Sobel test revealed that KDEF total accuracy was a mediator of the relationship between SPQ-CP and SFS interpersonal communication at trend level (Sobel test = -1.437, \( p = .075 \)).

See Table 14 for summary of the third model. SPQ-D was a significant predictor of KDEF total accuracy (\( p = .004 \)) and SPQ-D score and KDEF total accuracy together were significant predictors of SFS interpersonal communication (\( p = .000 \)). KDEF accuracy alone was a significant predictor of SFS interpersonal communication after accounting for SPQ-D (\( t(773) = 1.683, p = .047 \)). A Sobel test revealed that KDEF total accuracy was a mediator of the relationship between SPQ-D and SFS interpersonal communication at trend level (Sobel test = -1.437, \( p = .075 \)).
CHAPTER IV
DISCUSSION

To our knowledge, this is the first study to examine FAR in a psychometric high-risk sample with consideration of accuracy, reaction time, variability in performance as a function of type of expression, bias, and the relationship of FAR with symptoms and social functioning.

The Current Study Sample

Based on the existing psychometric high-risk literature and the total number of participants enrolled in the current study for whom valid data were recorded (N = 850), the current recruitment method was expected to yield a sample of approximately 85 (10%) individuals at psychometric high risk for psychosis, using SPQ cut-off score of 41. It is difficult to determine whether the current study yielded a proportion of high-risk individuals consistent with that reported in existing literature given that in the majority of previous psychometric high-risk studies, investigators first screened a large number of individuals from the general (or student) population using screening tools for psychosis proneness and only invited a subsample back to complete all measures. Only two studies examining psychometric risk and FAR to date have reported how many low-risk and high-risk individuals were identified via the screening procedures. Aguirre et al. (2008) screened 2102 college students using the SPQ-Brief and identified 79 (3.8%) high-risk individuals and 170 (8.1%) low-risk individuals. Similarly, Jahshan & Sergi (2007) screened 2108 college students using the SPQ-Brief and identified 104 (4.8%) high-risk individuals and 153 (7.2%) low-risk individuals. The current study sample yielded a sample that is somewhat inconsistent with this distribution; namely 78 (9.2%) high-risk
and 193 (22.7%) low-risk individuals. It is possible that this discrepancy may be attributable to methodological differences due to our on-line task administration or due to our inclusion of the Chapman Infrequency Scale which may have led to the exclusion of false-positives. However, it is difficult to assess consistency of findings because psychometric high-risk studies using the SPQ to identify psychometric risk and examining FAR abilities vary greatly in terms of methodology. That is, though most studies to date have recruited predominantly female, young adult samples from a college population (Jahshan & Sergi, 2007; Shean et al., 2007; Williams et al., 2007; Aguirre et al., 2008; Brown & Cohen, 2010; Germaine & Hooker, 2010; Abbott & Green, 2013), these studies often utilized the SPQ-Brief which contains fewer questions than the original SPQ; therefore, cutoff scores and variability of scores differ from the present study (Jahshan & Sergi, 2007; Aguirre et al., 2008; Germaine & Hooker, 2010). Other researchers have chosen to assess schizotypal traits using a Likert-like scale instead of the traditional dichotomous yes/no SPQ format (Brown & Cohen, 2010), and still others have chosen idiosyncratic and sample-based cutoff points to determine high- versus low-risk groups (Williams et al., 2007; Brown & Cohen, 2010). As a result of these methodological discrepancies, it is difficult to conclude whether or not our current results are consistent with previous literature. That said, our finding that 9.2% of the sample was psychometrically at high risk is in keeping with Lenzenweger & Korfine’s (1992) estimate that the base rate for psychometric high risk in an undergraduate population is 10%.

In terms of racial/ethnic group composition, the current study sample is largely representative of that which would be expected based on the known demographic
characteristics of the CUNY student population. However, it is not possible to determine whether this racial/ethnic group composition is consistent with previous psychometric risk literature examining FAR given that, to date, only one study reported information regarding sample racial/ethnic group composition. Brown & Cohen (2010) reported that 87.6% of their high-risk individuals and 77.8% of their low-risk individuals were Caucasian. Information regarding other racial groups was not provided and it is important to note that Brown & Cohen recruited college students from Louisiana State University where the student population is predominantly Caucasian (77%; Forbes, 2012).

**Group Differences in Overall FAR Accuracy**

Consistent with our stated hypotheses, individuals at psychometric high risk performed significantly less accurately on a FAR task than individuals at low psychometric risk. Thus, results of the present study are consistent with previous literature demonstrating that FAR deficits are exhibited by patients with schizophrenia (for review see Mandal et al., 1998; Edwards et al., 2002; Kohler et al., 2010; Chan et al., 2010) as well as individuals at heightened risk, including familial high risk for SSDs (for review see Lavoie et al., 2013), patients with SPD (Mikhailova et al., 1996; Waldeck & Miller, 2000), those at clinical or ultra-high risk for developing SSDs (Pinkham et al., 2007; Addington et al., 2008a; van Rijn et al., 2011; Addington et al., 2012; Amminger et al., 2012a; Amminger et al., 2012b; Thompson et al., 2012; Wölwer et al., 2012; Comparelli et al., 2013) and those at psychometric high risk (van't Wout et al., 2004; Williams et al., 2008; Aguirre et al., 2008; Brown & Cohen, 2010; Germine & Hooker, 2011; Roddy et al., 2012; Abbott & Green, 2013; Pelletier et al., 2013).
Beyond overall FAR accuracy, we examined accuracy for individual emotional expressions and neutral faces to see whether difficulty identifying and labeling specific emotions led to the overall accuracy finding. Several authors have previously reported that patients with schizophrenia demonstrate specific deficits in labeling faces displaying 1) anger (Goghari & Sphonheim, 2012; Janssens et al., 2012), 2) no expression and fear (Gilling McIntosh, 2011), 3) fear and sadness (Edwards et al., 2001), 4) surprise, fear, and disgust (Leung et al., 2011), and 5) threat emotions such as anger, fear, and disgust (Behere et al., 2011b) compared to healthy controls. Moreover, Bediou et al., (2007) reported that individuals at familial high risk were less accurate on fear and disgust faces while Pelletier et al. (2013) reported that psychometrically high-risk individuals were only less accurate on fear faces compared to low-risk individuals. In the current study, the high psychometric risk group was less accurate than the low psychometric risk group only when asked to label neutral faces. This is in keeping with previous literature which has demonstrated that patients with schizophrenia (Kohler et al., 2003), individuals at familial high risk (Eack et al., 2010), those at UHR (van Rijn et al., 2011), and those at psychometric high risk (Brown & Cohen, 2010) were only less accurate than controls at identifying neutral faces.

These findings hold relevance for the dimensional model of schizophrenia (Meehl, 1962) because they demonstrate that patterns commonly seen in patient populations with severe psychopathology also hold for non-clinical individuals with elevated schizotypal traits. Moreover, they attest to the utility of the psychometric high-risk research paradigm as a methodology well-suited to examining important etiological factors in psychopathology while minimizing the confounding effects of chronic
treatment, hospitalization, and stigma common in symptomatic patients (Lenzenweger, 1994).

**Group Differences in FAR Reaction Time**

In terms of reaction time, the high- and low-risk psychometric groups did not differ on overall time required to complete the FAR task or on reaction times for each emotional expression and neutral. Though this is inconsistent with some existing literature which has shown that patients with schizophrenia perform significantly slower than controls on FAR tasks (Gur et al., 2002; Machado de Sousa & Hallak, 2008; Habel et al., 2010; Calkins et al., 2010; Li et al., 2010b), there is some research which demonstrates that patients do not differ from controls with regard to reaction time on facial affect tasks (Fakra et al., 2008; Doop & Park, 2009). More importantly, previous studies examining various high-risk samples, including familial high risk (Li et al., 2010b), UHR (Seiferth et al., 2008; Dickey et al., 2011) and psychometric high risk (Green et al., 2001; Brown & Cohen, 2010), have demonstrated that these high-risk individuals are not significantly slower from controls on FAR tasks. These findings suggest that while FAR accuracy deficits may represent a trait of SSDs, reaction time during FAR tasks may remain preserved in at-risk populations and reduced reaction time may only become evident with disease progression. Therefore, the current findings do not support the existence of a speed-accuracy tradeoff in individuals at psychometric high risk for SSDs.

**Group Differences in FAR Bias**

Finally in terms of group differences, individuals at high psychometric risk were more likely to misattribute negative emotions to neutral faces than low-risk individuals,
demonstrating a negative bias. Similar findings have been reported in patients with schizophrenia (Kohler et al., 2003; Pinkham et al., 2011), individuals at familial high risk (Eack et al., 2010), and individuals at psychometric high risk (Brown & Cohen, 2010). However, in only one of these studies (Eack et al., 2010) did the authors reported that their high-risk sample was more likely to misattribute sadness to neutral faces, as was the case in the present study. In fact, in two previous studies both utilizing the Penn Emotion Recognition Test, patients with schizophrenia (Kohler et al., 2003) and individuals at psychometric high risk (Brown & Cohen, 2010) were significantly more likely to misattribute disgust to neutral faces. In the present study, it was extremely unlikely for any subjects to mislabel neutral faces as disgusted; that is, in the total sample, only 23 individuals mislabeled a neutral face as disgusted on one or more occasions, and all of these scores were identified as outliers (>3.0 SD above mean). Moreover, in a validation study of the photographs used in the present study, no subjects mislabeled neutral faces as disgusted, whereas sad bias was the most common form of negative bias (Calvo & Lundqvist, 2008). These findings emphasize the possibility that subtle differences in FAR stimuli may at least in part account for inconsistent findings across FAR studies and/or that the nature of negative bias may demonstrate some degree of emotion specificity that is differentiated across populations (e.g., disgust bias in patients versus greater tendency towards sad bias among at-risk individuals).

**Relationship of Symptoms with FAR Accuracy in the Total Sample**

Correlational analyses examining relationships between schizotypal traits and FAR performance in the entire sample, as well as high-risk and low-risk groups alone, suggest that patterns of relationships among variables differ in the different groups. For
instance, in the entire sample correlational analyses mirrored the group differences reported above. That is, total schizotypal traits were negatively correlated with total accuracy. When examining the individual SPQ subscales in the entire sample, positive-like symptoms (SPQ-CP) were negatively correlated with total FAR accuracy as well as sad accuracy, surprised accuracy, and neutral accuracy. These findings are in keeping with previous reports that FAR deficits were related to greater positive and general symptoms in a familial high-risk sample (Eack et al., 2010) as well as previous reports that positive symptoms are associated with FAR deficits in patients with schizophrenia (Mandal et al., 1999; Kohler et al., 2000; Kee et al., 2003; Larøi et al., 2010; Behere et al., 2011b) and positive-like symptoms are related to FAR deficits in psychometrically high-risk samples (van’t Wout et al., 2004; Roddy et al., 2012) and in healthy controls (Alfimova et al., 2009).

However, in the present study, we did not find a significant correlation between negative-like schizotypal traits (SPQ-I) and any FAR variables. This is inconsistent with the large body of existing literature that suggests that FAR deficits are related to severity of negative symptoms in patients with schizophrenia (Gaebel & Wölwer, 1992; Mueser et al., 1996; Silver & Shlomo, 2001; Kohler et al., 2003; Alfimova et al., 2009; Gilling McIntosh, 2011). These findings are also inconsistent with previous research using psychometric high-risk samples which have reported that FAR accuracy was specifically negatively correlated with negative-like (but not positive or disorganized) schizotypal traits as measured by the SPQ (Williams et al., 2007; Abbott & Green, 2013). However, to our knowledge, at least one study has reported that FAR accuracy was not related to negative symptoms in patients with schizophrenia (Kee et al., 2003). It remains possible
that some of this discrepancy is attributable to differences in FAR tasks (e.g., FEEST vs. PFA vs. KDEF).

Moreover, and also in keeping with Kee et al.’s (2003) findings in patients with schizophrenia, disorganized symptoms (SPQ-D) were negatively correlated with overall FAR accuracy. Disorganized symptoms were also specifically correlated with fear and sad accuracy. Overall, regarding FAR accuracy and symptoms, while current findings did not perfectly mirror the literature examining these relationships in schizophrenia, these inconsistencies are not uncommon in this area of research and suggest that facial affect abilities and symptoms may be differentially related at different points along the psychosis spectrum and these relationships require further research.

**Relationship of Symptoms with FAR Bias in the Total Sample**

In the entire sample only positive-like symptoms were positively correlated with negative bias. This is in accordance with previous reports that patients with predominantly positive symptoms (Mandal et al., 1999) and actively paranoid patients (i.e., a positive symptom; Pinkham et al., 2011) were more likely to commit biases than patients with predominantly negative symptoms. Eack et al., (2010) also reported that worse performance on neutral faces was related to attenuated positive symptoms in a familial high-risk sample and van’t Wout et al. (2004) found that positive-like symptoms as measured by the SPQ were associated with labeling happy faces as angry and fearful, a type of negative bias. However, it should be noted that Palmese (2009) reported that patients with positive symptoms actually performed more accurately when asked to judge neutral situations. These inconsistent findings should be interpreted with caution as Palmese (2009) presented patients with neutral situations not simply neutral faces, and
therefore, there are significant methodological differences between her study and the present study.

**Relationship of Symptoms with FAR Reaction Time in the Total Sample**

Only SPQ-D subscale was significantly correlated with any measure of KDEF reaction time. Perhaps surprisingly, SPQ-D was negatively correlated with disgust reaction time, that is, increased disorganized symptoms were correlated with quicker responses to disgusted faces. To date reaction time during FAR performance has only received limited attention in the literature and while patients are typically shown to perform more slowly than controls on FAR tasks (see Gur et al., 2002; Machado de Sousa & Hallak, 2008; Habel et al., 2010; Calkins et al., 2010; Li et al., 2010b), at least one study has demonstrated that patients respond more quickly than controls when asked to label sad faces (Seiferth et al., 2009). Moreover, Brown & Cohen (2010) reported that negative-like symptoms in a college sample were associated with quicker reaction times on a FAR task, which the authors attributed to a lack of investment in the FAR task. Furthermore, though negative-like symptoms were associated with quicker reaction times, individuals with high negative schizotypal traits were not less accurate than those with few negative schizotypal traits, suggesting that a speed-accuracy tradeoff did not occur. Though we were unable to fully replicate this finding in the current study, the negative correlation between SPQ-D and disgust reaction time does lend some further support our proposition above that reaction time during FAR tasks may remain preserved in at-risk populations until the development of florid psychosis.
**Relationships by Group (High vs. Low Psychometric Risk)**

It is interesting to contrast the aforementioned correlational findings in the entire sample with findings within high- and low-risk groups alone. For instance, in the high-risk group, only positive-like symptoms were negatively correlated with total FAR accuracy. Furthermore, positive-like symptoms were positively correlated with neutral reaction time, which is in keeping with Eack et al.’s (2010) finding of slower reaction time for neutral faces among individuals at familial high risk. This finding also suggests that a subtle speed-accuracy tradeoff may occur in psychometrically high-risk individuals such that they must spend significantly more time processing and analyzing a neutral face in order to label it accurately.

Though we were unable to replicate Green et al.’s (2001) findings demonstrating that delusion-prone individuals were significantly slower at identifying angry faces, suggesting an attentional bias for threat-related stimuli, it is important to note that Green et al.’s (2001) methodology differed greatly from that utilized in the current study. Most importantly, the authors asked participants to verbalize the emotional label instead of presenting them with a forced-choice format which may have added a confound given evidence of reduced verbal fluency and expression in psychometrically at-risk individuals (Barrantes-Vidal et al., 2003; Cohen & Hong, 2011; Cohen et al., 2012).

In contrast, in the low-risk group, only total schizotypal traits were negatively correlated with neutral accuracy and positively correlated with negative bias. None of the individual SPQ subscales was correlated with FAR accuracy, reaction time, or bias in the low-risk group. In this group, SPQ subscale scores were characterized by limited
variability and this factor may have reduced our ability to demonstrate significant relationships between the SPQ and other variables within the low-risk group.

**Depressive Symptoms as a Mediator of Relationship Between Schizotypal Traits and FAR Negative Bias**

FAR deficits are not a unique feature of SSDs and also have been demonstrated in a number of psychiatric disorders including major depression and bipolar disorder (see Kohler et al., 2011 for review), anxiety disorders (Sprengelmeyer et al., 1997; Kessler et al., 2008; Demenescu et al., 2011), Body Dysmorphic Disorder (Buhlmann et al., 2004), and autism spectrum disorders (Bölte & Poustka, 2003) as well as neurological conditions such as traumatic brain injuries (see meta-analysis by Babbage et al., 2011), epilepsy (e.g., McClelland et al., 2006; Walpole, Isaac, & Reynders, 2008), and neurodegenerative disorders including frontotemporal dementia, Alzheimer’s disease, and Parkinson’s disease (Bediou et al., 2012). Therefore, it is possible that previous findings of FAR deficits in SSDs are at least in part accounted for by comorbid symptoms and/or conditions. Given the high rates of comorbidity between schizophrenia and major depression (see Buckley et al., 2009 for review) and the strong correlations between schizotypal traits and depressive symptoms in psychometrically high-risk individuals (Fonseca-Peddero et al., 2011), in the present study, we examined the degree to which depressive symptoms accounted for the relationship between psychotic-like symptoms and FAR performance.

In the current total sample, depressive symptoms were negatively correlated with total FAR accuracy as well as positively correlated with negative bias and sad bias. Though a number of previous studies reported that comorbid depressive symptoms were
not associated with FAR accuracy in patients with schizophrenia (Kohler et al., 2000; Bediou et al., 2007), one study examining FAR performance in the general population reported that FAR accuracy was associated with depressive symptoms (Czukly et al., 2008). Therefore, it is possible that in patients with schizophrenia FAR abilities are so severely impaired that comorbid depressive symptoms do not contribute to additional FAR impairment (i.e., a floor effect), whereas in healthy or at-risk populations who experience only attenuated deficits and psychotic-like symptoms – as in the current study – depressive symptoms may yield a more deleterious influence on FAR performance. Moreover, the current finding that depressive symptoms were associated with negative and sad bias is consistent with previous research demonstrating these types of biases in individuals with MDD (Leppänen et al., 2004; Gollan et al., 2010; Milders et al., 2010; Naranjo et al., 2011). Notably, depressive symptoms were not associated with any KDEF variables in the low- or high-risk groups alone, suggesting that when variability of depressive symptoms is low, as might be the case in low-risk individuals who have relatively few depressive symptoms or high-risk and/or psychotic individuals where depressive symptoms may be prevalent, this relationship between depressive symptoms and FAR ability may be masked. Therefore, utilizing a dimensional perspective and examining depressive and psychotic-like symptoms along a continuum may help uncover important relationships among symptoms and between symptoms and cognitive abilities that might otherwise be difficult to discern in patient populations.

We proposed to examine whether depressive symptoms served as a mediator of the relationship between schizotypal traits and negative bias in the high-risk group based on previous research. In the present study, however, none of the schizotypal trait
dimensions were associated with negative bias in the high-risk group. Positive-like symptoms were, however, associated with negative bias in the total sample. Using both partial correlations and a more powerful statistical approach (i.e., multiple regression with Sobel test), we demonstrated that depressive symptoms mediate the relationship between positive-like schizotypal traits and negative bias. This mediation was demonstrated by the fact that when CES-D scores were entered as a covariate, the partial correlation between SPQ-CP and KDEF negative bias was no longer significant. Furthermore, using multiple regression analyses, we demonstrated that SPQ-CP and CES-D were significant predictors of KDEF negative bias, and a follow-up Sobel test demonstrated that CES-D was a significant mediator of the relationship between SPQ-CP and KDEF negative bias. These findings suggest that depressive symptomatology may significantly contribute to negative bias among individuals at high risk for psychosis, and highlights the importance of measuring and adjusting for depressive symptoms in future studies. These findings are also interesting in light of Kessler et al.’s (2007) findings that while patients with panic disorder performed significantly worse on a FAR task compared to healthy controls, after adjusting for depressive symptoms, all group differences disappeared; this suggested that comorbid depressive symptoms may account for at least some of the reported FAR deficits in various patient populations. However, given high rates of comorbidity (Buckley et al., 2009) and significant symptom overlap (Fonseca-Pedrero et al., 2011), it is important to consider that depressive symptoms and psychotic-like traits may share a significant portion of variance and, therefore, adjusting for depression may control for at least some psychotic-like symptoms. Therefore, it may be superficial to adjust for depressive symptoms in SSD research; at the very least, it is
important to consider the advantages and disadvantages (potential confounds) of implementing this approach (for a comprehensive review of the complex issues inherent in covariate analyses, see Miller & Chapman, 2001).

**Group Differences in Social Functioning**

Social functioning impairments are a hallmark of schizophrenia (Bellack et al., 1990) and previous literature in patient populations has demonstrated that social cognition (including FAR) was strongly related to social functioning (Couture et al., 2006; Fett et al., 2011; Irani et al., 2012). Based on these findings, we aimed to examine whether high and low psychometric risk for psychosis groups differed with regard to social functioning.

In the present study, the high psychometric risk group (compared to the low-risk group) reported significantly worse overall social functioning as well as worse functioning in the domains of social engagement, interpersonal communication, independence-performance, prosocial behavior, and independence-competence. This is in accordance with previous retrospective studies with schizophrenia patients (Cannon et al. 1997, Monte et al., 2008, Yung & McGorry, 1996; Corcoran et al., 2007; Møller & Husby, 2000; Tan & Ang, 2001), prospective studies with UHR individuals (Cornblatt et al., 2003; Lencz et al., 2004; Shim et al., 2008; Ballon et al., 2007; Addington et al., 2008b) as well as more recent studies with psychometric high-risk individuals (Jahshan & Sergi, 2007; Henry et al., 2008; Fonseca-Pedrero et al., 2010; Barrantes-Vidal et al., 2010; Simpson & Pinkham, 2011), all of which have demonstrated that patients and those at elevated risk for SSDs demonstrate relatively impaired social functioning. The impairments in psychometrically high-risk individuals are attenuated compared to
patients with florid psychosis; however, these deficits may still have a deleterious impact on everyday functioning and quality of life. Though treating psychometrically high-risk individuals may not currently be feasible or ethical, evidence suggests that social functioning impairments in UHR samples are not permanent and are amendable through treatment (Cornblatt et al., 2007; Niendam et al., 2007; Ruhrmann et al., 2007; Jang et al. 2011). Therefore, the current findings highlight the need for future development of improved methods to identify those at risk for SSDs, as well as the development of safe and feasible interventions to improve social functioning in at-risk populations.

**FAR as a Mediator of Relationship between Schizotypal Traits and Social Functioning**

To review, we have demonstrated that individuals at high psychometric risk demonstrated FAR and social functioning deficits compared to those at low psychometric risk. We also demonstrated that, in line with previous findings using comparable measures in psychometric high-risk studies (Henry et al., 2008; Heber et al., 2011; Culianez et al., 2011), psychotic-like symptoms (namely, total SPQ score and SPQ subscale scores) were consistently negatively correlated with social functioning (i.e., SFS total score and SFS subscale scores). As reported above, specifically, total SPQ score and SPQ-CP and SPQ-D subscale scores were negatively correlated with KDEF total accuracy. Lastly, consistent with previous findings among schizophrenia patients (see meta-analyses by Couture et al., 2006; Fett et al., 2011; Irani et al., 2012), patients with SPD (Wickline et al., 2012) and individuals at high psychometric risk (Aguirre et al., 2008; Pelletier et al., 2013), the current study demonstrated that KDEF accuracy was positively correlated with the interpersonal communication subscale of the SFS. Using
multiple regression analyses and the Sobel test to examine the proposed mediation models, we demonstrated that the magnitude of the relationship between total schizotypal personality traits and interpersonal communication did not decrease after accounting for FAR accuracy. This suggests that FAR does not mediate the direct relationship between psychotic-like symptoms and social functioning. However, FAR accuracy appears to serve as a mediator of the relationship between positive-like schizotypal traits and disorganized schizotypal traits and interpersonal communication at the trend level. This suggests that FAR accuracy may act as a partial mediator between symptoms and functioning, and the relationship between FAR and social functioning in at-risk samples warrants further investigation.

**Treatment Implications**

The present finding that FAR deficits are present among individuals at psychometric high risk for SSDs and are related to poor social functioning has important treatment implications. Some research to date suggests that there is no difference in FAR deficit severity between UHR and first episode patients (Thompson et al., 2012), between first episode and chronic patients (Leung et al., 2011), nor between UHR, first episode, and chronic patients (Comparelli et al., 2013), suggesting that FAR deficits may be a stable trait of SSDs. However, other studies have demonstrated that patients who have experienced multiple episodes of psychosis are more impaired on FAR than first episode patients (Comparelli et al., 2011). Importantly, Malik et al. (2010) demonstrated that patients who had a prolonged duration of untreated psychosis – over 80 weeks – showed more impaired FAR than patients with short duration of untreated psychosis. These findings provide a context for understanding the current finding of a difference in FAR
between high- and low-risk psychometric groups, and highlight the potential importance of early identification and treatment of at-risk individuals with respect to FAR.

Unfortunately, among patients with schizophrenia, FAR deficits remain largely refractory to pharmacological intervention such as antipsychotic medication (Kucharska-Pietura, Mortimer, Tylec, & Czernikiewicz, 2012; see Hempel, Dekker, van Beveren, Tulen, & Hengeveld, 2010 for review). This may be due to the fact that though most antipsychotics work as potent dopamine antagonists, recent research suggests that FAR deficits are independent of the dopaminergic system (Bediou et al., 2012) and may be reliant on glutamatergic system dysfunction (Ebert, Haussleiter, Juckel, Brüne, & Roser, 2012). Furthermore, pharmacologically treating at-risk individuals remains a controversial issue (see Cornblatt et al., 2001; McGlashan, 2001; Warner, 2005; McGlashan, 2005; Filakovic et al., 2007). Until recently, this rendered the field at a loss for treatment options for patients experiencing FAR deficits and subsequent functional deficits. Over the past several years, however, investigators have developed a number of social cognition remediation programs which target FAR deficits in the hopes of improving not only FAR abilities but also social functioning and quality of life. For a recent meta-analysis of existing programs see Kurtz & Richardson (2012). For a complementary comprehensive, qualitative review of these programs with consideration of implications for high-risk research and the role of sex effects, see Statucka & Walder (2013). Though use of these remediation programs with high-risk samples remains extremely limited to date (only one known pilot study currently underway; see Bartholomeusz & Allott, 2012), the rationale for including FAR remediation as part of intervention programs for UHR individuals is strong (see Statucka & Walder, 2013).
Lastly, it is worth noting that a number of noninvasive interventions have been recently demonstrated to improve FAR in patients with schizophrenia and may therefore be well suited for incorporation into preventive interventions targeting individuals at risk for SSDs. For instance, Behere et al. (2011a) reported that patients in a yoga therapy group demonstrated improvement in symptoms, FAR accuracy, as well as socio-occupational functioning over a four-month period. To date, two groups have reported that administration of oxytocin via nasal spray improved FAR ability in patients with schizophrenia (Goldman, Gomes, Carter, & Lee, 2011; Averbeck, Bobin, Evans, & Shergill, 2012). Goldman et al. (2011) speculated that intranasal oxytocin may alter neuroendocrine functioning mediated by regions such as the hippocampus, amygdala, and hypothalamus. Averbeck et al. (2012) reference a growing body of literature suggesting that oxytocin effects are mediated by normalizing amygdala functioning. Studies such as these suggest that improving/altering limbic system functioning can lead to improvement in FAR in SSD populations. These noninvasive interventions hold promise for treatment of UHR and other high-risk individuals and warrant future investigation.

**Neurobiological Implications**

Though a number of social remediation programs and noninvasive interventions targeting FAR deficits currently exist, these programs may benefit from considering recent neuroimaging and neurophysiological findings in the refinement and shaping of existing FAR interventions. Emerging neurobiological evidence may also help clarify neural substrates of FAR deficits in SSDs.

Above we have cited behavioral evidence, which demonstrates that impairments in FAR are present in patients with schizophrenia and those at risk for psychosis. The
current study focuses on clarifying the behavioral relationships among FAR, schizotypal symptoms, and social functioning, with an eye on implications for behavioral interventions. However, there is a growing body of literature, which examines the link between disruptions in neuroanatomical structures and systems with FAR performance in psychosis. Further research is warranted to clarify the neural underpinnings of the FAR deficits in psychosis and in those at risk. Though an extensive review of this literature is beyond the scope of this paper, a brief summary of our current understanding of neurological underpinnings of FAR is provided below.

A distributed and complex neural network is involved in facial affect processing and recognition in healthy individuals including the visual cortex, fusiform gyrus, superior temporal sulcus, amygdala, hippocampus, and cingulate gyrus (Vuilleumier & Poutois, 2007). However, some authors have suggest that FAR deficits are specifically related to dysfunction of mesial temporal regions especially the amygdala in patients with schizophrenia (Kohler et al., 2003; Pinkham, Gur, & Gur, 2007a). The amygdala is involved in complex judgments of emotion and though it is activated by facial stimuli in general, it is preferentially activated by negatively valenced emotions (Vuilleumier & Poutois, 2007). There is evidence from functional magnetic resonance imaging studies that activation of the amygdala during affect recognition tasks is attenuated in schizophrenia patients compared to healthy controls (Gur et al. 2002; Hempel, Hempel, Schönknecht, Stippich, & Schröder, 2003). Later studies by Gur and colleagues (2007) demonstrated that greater amygdala activation in patients during the presentation of fearful faces was positively correlated with flat affect and, paradoxically, with failure to correctly recognize the emotion. A recent study by Lepage et al. (2011) found that
schizophrenia patients and healthy controls equally recruited many brain regions during perception of emotional faces but in the patients the severity of flat affect moderated activity in the amygdala and parahippocampal gyri. Therefore, there is evidence that FAR is impaired in schizophrenia patients and that these impairments are related to negative symptoms, including flat affect (Kohler et al. 2003). Moreover, both FAR impairment and flat affect appear to be related to amygdalar dysfunction.

Beyond amygdala dysfunction, researchers have demonstrated that patients with schizophrenia fail to engage other important components of the facial affect processing network when presented with facial stimuli as compared to healthy controls. For instance, researchers have demonstrated that patients failed to activate 1) the fusiform gyrus (Quintana, Wong, Ortiz-Portillo, Marder, & Mazziotta, 2003; Johnston, Stojanov, Devir, & Schall, 2005; Seiferth et al., 2009), 2) parts of the occipital lobes (Johnston et al., 2005; Seiferth et al., 2009), and 3) relevant parts of the frontal lobes including the inferior frontal cortex (Johnston et al., 2005) and orbital frontal cortex (Quintana et al., 2011). Interestingly, Seiferth et al. (2009) reported that schizophrenia patients also demonstrated a hyperactivation of the right cuneus during FAR which they speculated may reflect compensatory mechanisms. Similarly, Fakra et al. (2008) reported a mixture of hypoactivations and hyperactivations during facial processing tasks in patients with schizophrenia. When compared to healthy controls, schizophrenia patients failed to activate the limbic system (automatic processing of emotions), and instead showed decreased activation in the fusiform gyrus (holistic face processing) and increased activation in the inferior parietal cortex, left middle temporal lobe, and right precuneus (regions associated with feature analysis) during a facial matching task (Fakra et al.,
This led the authors to conclude that patients with schizophrenia adopt a cognitive and feature-based approach when processing emotional faces. Given that many of the currently developed remediation programs encourage a more feature-based approach to FAR, development of programs encouraging more configuration-based processing as demonstrated by healthy controls when completing FAR tasks may prove beneficial.

A number of recent meta-analyses have examined functional neuroimaging data during facial affect processing in patients with schizophrenia. For instance, Surgranyes and colleagues (2011) reported hypoactivation in the prefrontal cortex, posterior cingulate cortex, amygdala, occipito-temporal regions (including fusiform gyrus), and thalamus in schizophrenia patients during FAR. While the finding of widespread hypoactivation is most common in the literature, some authors have reported abnormal hyperactivation as well. Li, Chan, McAlonan, & Gong (2010a) demonstrated that while both patients with schizophrenia and healthy controls activate the bilateral amygdala and right fusiform gyri while processing facial expressions, the extent of activation in bilateral amygdala, parahippocampal gyrus, fusiform gyrus, right superior frontal gyrus, and right lentiform nucleus is more limited in patients. Furthermore, patients with schizophrenia activated the left insula while processing facial expression, which was absent in healthy controls (Li et al., 2010a). In their meta-analysis, Taylor and colleagues (2012) reported that in addition to hypoactivation in the bilateral amygdala, visual processing areas, anterior cingulate cortex, dorsolateral frontal cortex, medial frontal cortex, and subcortical structures (including the thalamus, caudate, and midbrain), patients with schizophrenia also demonstrated hyperactivation of the cuneus, parietal lobule, precentral gyrus, and superior temporal gyrus. The most recent meta-analysis in
this area mirrored these findings and reported hypoactivation in the left precentral and medial frontal gyri, right amygdala and insula, left parahippocampal gyrus and anterior cingulate cortex, right inferior occipital and fusiform gyri, right caudate nucleus, and right medial dorsal thalamus but hyperactivation within the right cuneus (Delvecchio, Sugranyes, & Frangou, 2013).

Though there is a growing body of literature examining neurobiological correlates of FAR in patient populations, only a limited number of studies have reported on imaging findings during FAR in populations at risk for psychosis. In UHR individuals, while behavioral performance did not differ from controls, emotion discrimination was associated with hyperactivation of the right lingual and fusiform gyrus and left middle occipital gyrus and specifically, stronger activation in the inferior and superior frontal gyri, the cuneus, the thalamus, and the hippocampus while viewing neutral faces (Seiferth et al., 2008). Non-psychotic siblings of patients and their psychotic probands showed abnormal activation in the precentral and superior frontal gyri during FAR (Li et al., 2012). Finally, in a community sample of individuals with low versus high social anhedonia (i.e., psychometric risk), the high-risk individuals showed less activation in the rostral medial prefrontal cortex, right superior temporal gyrus, and left somatosensory cortex during an emotion discrimination task relative to the low-risk individuals, and reduced activation was more pronounced in the medial prefrontal cortex (Germine, Garrido, Bruce, & Hooker, 2011). Though none of these studies explicitly examined the relationships between psychotic-like symptoms and functional imaging findings, it is interesting to note that regions identified in all three studies have been consistently associated with specific symptoms domains in patients with schizophrenia. For example,
positive symptoms are related to functioning of the medial prefrontal cortex, amygdala, and hippocampus/parahippocampal region (see meta-analysis by Goghari, Sponheim, & MacDonald, 2010), all of which have been implicated in FAR deficits in at-risk samples. To date, the neuroimaging findings in at-risk samples largely parallel findings in patient populations and demonstrate widespread dysfunction of the neural network involved in facial affect processing. Further investigation of the associations between symptomatology and FAR performance in these samples is warranted.

Recent neurophysiological findings also shed light on etiological factors involved in SSDs and hold implications for future development of effective interventions. For instance, patients with schizophrenia show reduced N170 amplitudes compared to healthy controls. That is, while healthy controls show increased N170 amplitudes when looking at emotional faces compared to neutral faces, patients with schizophrenia show no difference on N170 when looking at neutral and emotional faces (Kirihara et al., 2012; Tsunoda et al., 2012). Individuals at UHR also showed reduced N170 amplitudes as well as reduced P100 and N250 amplitudes (Wölwer et al., 2012). Kirihara et al. (2012) suggested that these finding might reflect impairment in the structural encoding of emotional faces and in the discrimination between emotional and neutral faces. Given these findings and the negative bias commonly demonstrated by patients with schizophrenia when viewing neutral stimuli, social cognition remediation programs may wish to focus on training patients to correctly distinguish neutral from emotional faces. Finally, Linden et al. (2010) demonstrated that while patients with schizophrenia were impaired when asked to explicitly label emotional faces (i.e., a FAR task), they nonetheless showed an enhanced working memory capacity for angry faces just like
healthy controls. The authors suggested that this ability portends that preserved implicit emotional processing exists in patients with schizophrenia and should be incorporated into social cognitive remediation efforts.

**Study Limitations**

The present study has some noteworthy limitations. First, the FAR task used in this study, the KDEF, utilizes only Caucasian faces as part of the stimuli. In healthy individuals, FAR accuracy is higher when emotions are both expressed and recognized by members of the same national, ethnic, or regional group (for meta-analysis see Elfenbein & Ambady, 2002). Similarly, research in patients with schizophrenia has also shown that participants from other ethnic groups do not perform as well as Caucasian patients on measures of emotion perception that use only Caucasian faces (Habel et al. 2000; Brekke, Nakagami, Kee, & Green, 2005b), leading some authors to emphasize the need for using ethnically diverse FAR stimuli in this field of research (Pinkham et al., 2008).

Several lines of evidence, however, suggest that the KDEF accurately assessed FAR abilities in the current sample. Firstly, the current sample was predominately composed of Caucasians, approximately 32.5% of the sample, and the proportion of ethnicities did not statistically differ between the high- and low-risk groups. Secondly, a preliminary one-way ANOVA demonstrated that the different racial groups did not differ from one another in terms of total FAR accuracy \((p = .900)\). This may at least in part be due to the fact that this study was conducted in New York City, one of the most multicultural cities in the world, where participants potentially have many opportunities to be exposed to other racial and ethnic groups. In fact, Elfenbein & Ambady (2002)
demonstrated that the in-group advantage is smaller for cultural groups who have exposure to one another. Furthermore, all KDEF faces were Caucasian (i.e., the majority group in North America) and Elfenbein & Ambady (2002) demonstrated that minority group members were better able to judge emotions of majority group members than vice versa. Examining emotion recognition abilities across ethnic groups especially through the use of ethnically diverse samples and appropriate FAR stimuli is an important avenue for future research.

The second limitation of the current study was that all data collection occurred remotely via the Internet. Although an increasing body of literature has demonstrated that results from samples tested over the internet are reliable and empirically valid (see Davis, 1999; Riva et al., 2003; Gosling et al., 2004; Haworth et al. 2007; Silverstein et al., 2007; Man et al., 2009; Crump et al., 2013), internet data collection may still raise concerns regarding data validity. Germine & Hooker (2010) conducted a study examining FAR in a psychometrically at-risk sample recruited from the general population, entirely via the Internet. Germine & Hooker (2010) reported that performance on their FAR task using the internet-based sample was comparable to data collected using a laboratory-based sample, and that SPQ scores were almost identical to those in a community sample. In the present study, we took a number of precautions to ensure validity of data including 1) informing participants of optimal environmental conditions for study completion to minimize distractions, 2) requiring that participants complete study participation during one session to limit variability between sessions, 3) administering the CIS (Chapman & Chapman, 1983) so that participants who were responding randomly, pseudorandomly, or dishonestly could be excluded from the
sample, and 4) the recording of total time taken to complete the FAR task so that participants who were presumably distracted or otherwise not fully engaged during the task (as determined by extreme reaction time scores) could be identified and excluded from analyses. Despite these precautions, it was not possible to monitor each participant while they completed study participation or to verify the accuracy of information that they provided.

Another limitation of data collection over the Internet is that it largely restricts the type of data that can be collected. For instance, in the present study, we were restricted to collecting only self-report data regarding schizotypal traits, mood symptoms, and social functioning (as opposed to clinician- or family-ratings of symptoms and functioning, and ecologically valid measures of functioning such as the experience sampling method). This limitation is especially salient in terms of assessing social functioning, where some authors have shown that when ecologically valid experience sampling methodologies were used, there was no relationship between FAR ability and social functioning even in patients with schizophrenia (Janssens et al., 2012). Therefore, future research in this field should examine the relationship among schizotypal personality traits, FAR ability, and social functioning using more ecologically valid methods.

**Study Strengths**

The aforementioned study limitations notwithstanding, collecting data over the Internet allowed us to obtain a large overall sample size, which would not have been practically feasible if the study was conducted in a traditional laboratory setting. This sampling method allowed us to identify a large subsample of psychometrically high-risk
individuals and to utilize statistical analyses to examine attenuated deficits and mediation models, which may have lacked statistical power with a smaller sample.

Another strength of the current study was the inclusion of the CIS to help identify participants who responded to self-report measures randomly, carelessly, or dishonestly. Though including an infrequency scale such as the CIS or the Oviedo Infrequency Scale (Fonseca-Pedrero et al., 2011) is not currently standard practice in the psychometric high-risk literature, the results of the present study suggest that inclusion of such measures is warranted in future studies. We collected data from 965 subjects; in this complete sample, 98 individuals (or 10.2% of the sample) were identified as being at psychometric high risk. This is consistent with previous research demonstrating that the base rate for psychometric high risk in an undergraduate populations was 10% (Lenzenweger & Korfine, 1992). Of note, of the initial total sample of 965, 108 participants were excluded because they endorsed three or more items on the CIS, suggesting that their responses on self-report measures may be invalid. Using a similar infrequency measure, Fonseca-Pedrero et al. (2011) reported that they excluded 69 participants out of a sample of 1384 adolescents, or approximately 5%. Most importantly, in our sample, out of the 108 excluded participants, 20 would have been falsely identified as psychometrically high risk based on their SPQ scores. Had these falsely identified individuals remained in the sample, their potentially invalid scores may have added noise to the data and erroneously influenced the results. After excluding these 108 participants, in the remaining sample of 857 individuals, we identified 78 psychometrically high-risk participants or 9.1% of the samples, which is still within the estimate of 10% outlined by Lenzenweger & Korfine (1992).
Conclusion

In conclusion, the present study showed that individuals at high psychometric risk were significantly less accurate on the overall FAR task, less accurate on neutral faces, were more likely to misattribute negative emotions to neutral faces, more likely to misattribute sadness to neutral faces, and reported more impaired social functioning when compared to low-risk individuals. These findings are consistent with past research in various populations along the schizophrenia spectrum.

In the present study, scores on the SPQ were characterized by great variability (i.e., while some subjects reported experiencing no schizotypal personality traits, others endorsed nearly all schizotypal traits on the SPQ). Studies such as this one, which focus on healthy non-clinical populations, lend further support to Meehl’s (1962) dimensional model of schizophrenia and may expand our existing knowledge of psychopathology, and of the function of psychopathology traits within the normal population. Future studies with non-clinical population all along the spectrum of schizophrenia disorders may provide a better understanding of the dimensional nature of these disorders as well as clarify the constellation of genetic and environmental risk factors that contribute to risk for SSDs.

Furthermore, total schizotypal traits, positive-like and disorganized schizotypal traits but not negative-like schizotypal traits were correlated with various aspects of FAR. Although these results are not altogether consistent with previous research, there is some emerging evidence to suggest that positive-like and disorganized schizotypal traits are differentially correlated with FAR performance in SSDs. Neuroanatomical regions associated with positive symptoms of psychosis such as the prefrontal cortex, especially
the medial prefrontal cortex, and temporal regions including the amygdala and hippocampus (see meta-analysis by Goghi et al., 2010) have also been implicated in emerging imaging research demonstrating that impaired facial processing in at-risk samples is associated with abnormal functioning in these areas (see Seiferth et al., 2008; Germine et al., 2011; Li et al. 2012). Though no study to date has explicitly examined the relationship between psychotic-like symptoms and FAR in an at-risk group, the existing evidence suggests that FAR deficits in those at risk may be related to dysfunction of neuroanatomical substrates implicated in positive symptomatology such as the prefrontal cortex and limbic system.

In the present study, some aspects of the FAR deficits demonstrated in this sample were obliterated when adjusting for comorbid depressive symptoms, which suggests that depressive symptomatology may at least in part account for FAR deficits exhibited by SSD samples. Though there may be some overlap between psychotic-like and depressive symptoms, it is important to consider the caveats regarding covariate analyses, as presented by Miller & Chapman (2001), when conducting psychometric high-risk research and when subsequently conducting statistical analyses.

Finally, FAR accuracy appears to act as a partial mediator in the direct relationship between positive-like and disorganized schizotypal traits and interpersonal communication, which suggests that difficulties with FAR may help to at least in part explain how schizotypal traits are associated with impaired social functioning in at-risk individuals.

In sum, individuals at psychometric high risk demonstrate FAR deficits and social functioning impairments that are similar to those seen in patient populations but in an
attenuated form, and difficulties with FAR are related to impaired social functioning. The current findings should be interpreted with caution given that all data was obtained via self-report questionnaires remotely over the Internet and only Caucasian models were used in the FAR task. Despite these potential limitations, we implemented numerous precautions to ensure validity of collected data and, more importantly, internet-based data collection allowed us to collect data from a sample large enough to examine the attenuated impairments found among psychometrically high-risk individuals, as well as more complex mediation models. Future research examining the role of affect recognition and social functioning in the etiology, development, treatment, and prognosis of SSDs using more ecologically-valid measures of affect recognition and social functioning is warranted.
Table 1

Demographic Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Total Sample</th>
<th>High-Risk Group</th>
<th>Low-Risk Group</th>
<th>Group Differences</th>
</tr>
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<tbody>
<tr>
<td><strong>N</strong></td>
<td>850</td>
<td>78</td>
<td>193</td>
<td></td>
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<tr>
<td><strong>MEAN AGE (SD)</strong></td>
<td>21.01 (3.4)</td>
<td>20.97 (3.7)</td>
<td>21.80 (3.9)</td>
<td>$Z = -1.670, p = .095^b$</td>
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<tr>
<td><strong>SEX, n (%)</strong></td>
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<td>$X^2 = 0.442, p = .506$</td>
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<tr>
<td>Male</td>
<td>266 (31.3)</td>
<td>30 (38.5)</td>
<td>66 (34.2)</td>
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<td>584 (68.7)</td>
<td>48 (61.5)</td>
<td>127 (65.8)</td>
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<tr>
<td><strong>ETHNICITY, n (%)</strong></td>
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<tr>
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<td>62 (79.5)</td>
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<td>168 (19.8)</td>
<td>14 (17.9)</td>
<td>32 (16.6)</td>
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<td><strong>RACE, n (%)</strong></td>
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<td>276 (32.5)</td>
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<td>79 (40.9)</td>
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<td>Black/African American</td>
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<td>15 (19.2)</td>
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<tr>
<td>Asian</td>
<td>217 (25.5)</td>
<td>22 (28.2)</td>
<td>43 (22.3)</td>
<td></td>
</tr>
<tr>
<td>Pacific Islander</td>
<td>6 (0.7)</td>
<td>2 (2.6)</td>
<td>2 (1.0)</td>
<td></td>
</tr>
<tr>
<td>Native American/Alaskan</td>
<td>9 (1.1)</td>
<td>1 (1.3)</td>
<td>1 (0.5)</td>
<td></td>
</tr>
<tr>
<td>Other or Mixed Ethnicity</td>
<td>167 (19.6)</td>
<td>17 (21.8)</td>
<td>29 (15.0)</td>
<td></td>
</tr>
<tr>
<td><strong>HANDEDNESS, n (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td>$X^2 = 1.565, p = .457$</td>
</tr>
<tr>
<td>Right</td>
<td>751 (88.4)</td>
<td>71 (91.0)</td>
<td>173 (89.6)</td>
<td></td>
</tr>
<tr>
<td>Left</td>
<td>67 (7.9)</td>
<td>6 (7.7)</td>
<td>12 (6.2)</td>
<td></td>
</tr>
<tr>
<td>Mixed</td>
<td>32 (3.8)</td>
<td>1 (1.3)</td>
<td>8 (4.1)</td>
<td></td>
</tr>
</tbody>
</table>

*a* Data missing for some participants

*b* Mann Whitney $U$ test used for comparison.

* $p < .05$, ** $p < .01$, *** $p < .001$; all tests were two-tailed.
<table>
<thead>
<tr>
<th>Facial Emotion</th>
<th>Groups</th>
<th>N</th>
<th>Mean Accuracy (SD)</th>
<th>Statistic</th>
<th>p-value</th>
<th>Cohen’s d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>High-Risk</td>
<td>73</td>
<td>51.95 (4.143)</td>
<td>Z = -2.670</td>
<td>.004*</td>
<td>-.360</td>
</tr>
<tr>
<td></td>
<td>Low-Risk</td>
<td>184</td>
<td>53.43 (4.071)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angry</td>
<td>High-Risk</td>
<td>73</td>
<td>6.34 (1.336)</td>
<td>Z = -0.147</td>
<td>.442</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>Low-Risk</td>
<td>183</td>
<td>6.34 (1.194)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Disgust</td>
<td>High-Risk</td>
<td>75</td>
<td>5.87 (1.870)</td>
<td>Z = -1.209</td>
<td>.114</td>
<td>-.172</td>
</tr>
<tr>
<td></td>
<td>Low-Risk</td>
<td>184</td>
<td>6.18 (1.726)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fear</td>
<td>High-Risk</td>
<td>75</td>
<td>3.48 (1.622)</td>
<td>t = 1.671</td>
<td>.048</td>
<td>-.229</td>
</tr>
<tr>
<td></td>
<td>Low-Risk</td>
<td>185</td>
<td>3.86 (1.706)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sad</td>
<td>High-Risk</td>
<td>73</td>
<td>6.53 (1.156)</td>
<td>Z = -1.063</td>
<td>.144</td>
<td>-.185</td>
</tr>
<tr>
<td></td>
<td>Low-Risk</td>
<td>183</td>
<td>6.73 (1.001)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Surprised</td>
<td>High-Risk</td>
<td>71</td>
<td>7.56 (0.612)</td>
<td>Z = -0.977</td>
<td>.165</td>
<td>-.320</td>
</tr>
<tr>
<td></td>
<td>Low-Risk</td>
<td>183</td>
<td>7.74 (0.510)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neutral</td>
<td>High-Risk</td>
<td>73</td>
<td>14.58 (1.683)</td>
<td>Z = -2.745</td>
<td>.003*</td>
<td>-.409</td>
</tr>
<tr>
<td></td>
<td>Low-Risk</td>
<td>177</td>
<td>15.19 (1.272)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*a* Mann Whitney U test used for comparisons.

*b* Student’s t test used for comparisons.

*p*-value significant after Holm-Bonferroni correction for multiple comparisons; all tests were one-tailed.
<table>
<thead>
<tr>
<th>Facial Emotion</th>
<th>Groups</th>
<th>N</th>
<th>Mean Reaction Time in millisecond (SD)</th>
<th>Statistic</th>
<th>p-value</th>
<th>Cohen’s d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total(^a)</td>
<td>High-Risk</td>
<td>73</td>
<td>3.247 (0.011)</td>
<td>(t = 0.365)</td>
<td>.715</td>
<td>-.091</td>
</tr>
<tr>
<td></td>
<td>Low-Risk</td>
<td>180</td>
<td>3.248 (0.011)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angry(^a)</td>
<td>High-Risk</td>
<td>74</td>
<td>3.036 (0.037)</td>
<td>(t = 1.229)</td>
<td>.220</td>
<td>-.189</td>
</tr>
<tr>
<td></td>
<td>Low-Risk</td>
<td>179</td>
<td>3.043 (0.037)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Disgust(^a)</td>
<td>High-Risk</td>
<td>73</td>
<td>3.052 (0.037)</td>
<td>(t = 0.079)</td>
<td>.937</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>Low-Risk</td>
<td>179</td>
<td>3.052 (0.033)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fear(^a)</td>
<td>High-Risk</td>
<td>75</td>
<td>3.071 (0.036)</td>
<td>(t = 1.553)</td>
<td>.112</td>
<td>-.219</td>
</tr>
<tr>
<td></td>
<td>Low-Risk</td>
<td>182</td>
<td>3.079 (0.037)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Happy(^a)</td>
<td>High-Risk</td>
<td>74</td>
<td>3.949 (0.104)</td>
<td>(t = -1.213)</td>
<td>.226</td>
<td>.164</td>
</tr>
<tr>
<td></td>
<td>Low-Risk</td>
<td>181</td>
<td>3.932 (0.103)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sad(^b,c)</td>
<td>High-Risk</td>
<td>75</td>
<td>4380.69 (3081.874)</td>
<td>(Z = -0.805)</td>
<td>.421</td>
<td>.070</td>
</tr>
<tr>
<td></td>
<td>Low-Risk</td>
<td>185</td>
<td>4198.60 (2037.015)</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Surprised(^a)</td>
<td>High-Risk</td>
<td>75</td>
<td>1.960 (0.010)</td>
<td>(t = -0.334)</td>
<td>.739</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>Low-Risk</td>
<td>179</td>
<td>1.960 (0.009)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neutral(^a)</td>
<td>High-Risk</td>
<td>73</td>
<td>2.394 (0.021)</td>
<td>(t = -0.137)</td>
<td>.891</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>Low-Risk</td>
<td>179</td>
<td>2.394 (0.021)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\(^a\) Student’s \(t\) test used for comparisons.

\(^b\) Mann Whitney \(U\) test used for comparisons.

\(^c\) Sad reaction time could not be normalized in a satisfactory manner using Box-Cox transformation and therefore, raw scores were used for sad reaction time analyses; all other reaction times normalized via Box-Cox transformations.

\(*\) \(p\)-value significant after Holm-Bonferroni correction for multiple comparisons; all tests were two-tailed.
<table>
<thead>
<tr>
<th>Facial Emotion</th>
<th>Groups</th>
<th>N</th>
<th>Mean Bias (SD)</th>
<th>Statistic</th>
<th>p-value</th>
<th>Cohen’s d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Negative</td>
<td>High-Risk</td>
<td>73</td>
<td>1.01 (1.296)</td>
<td>Z = -2.452</td>
<td>.007*</td>
<td>.332</td>
</tr>
<tr>
<td></td>
<td>Low-Risk</td>
<td>178</td>
<td>0.62 (1.041)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angry</td>
<td>High-Risk</td>
<td>75</td>
<td>0.23 (0.481)</td>
<td>Z = -1.525</td>
<td>.064</td>
<td>.148</td>
</tr>
<tr>
<td></td>
<td>Low-Risk</td>
<td>181</td>
<td>0.16 (0.462)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Happy</td>
<td>High-Risk</td>
<td>70</td>
<td>0.19 (0.490)</td>
<td>Z = -0.681</td>
<td>.248</td>
<td>.139</td>
</tr>
<tr>
<td></td>
<td>Low-Risk</td>
<td>183</td>
<td>0.13 (0.364)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sad</td>
<td>High-Risk</td>
<td>73</td>
<td>0.75 (1.077)</td>
<td>Z = -2.749</td>
<td>.003*</td>
<td>.358</td>
</tr>
<tr>
<td></td>
<td>Low-Risk</td>
<td>180</td>
<td>0.41 (0.804)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Mann Whitney U test used for comparisons.

*b Total negative bias consists of sum of angry, disgust, fear, and sad biases.

*p-value significant after Holm-Bonferroni correction for multiple comparisons; all tests were one-tailed.
Table 5
Correlations of KDEF Accuracy, Reaction Time and Bias with Symptoms for Total Sample

<table>
<thead>
<tr>
<th></th>
<th>SPQ total Correlation</th>
<th>SPQ-CP Correlation</th>
<th>SPQ-I Correlation</th>
<th>SPQ-D Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>p-value</td>
<td>p-value</td>
<td>p-value</td>
<td>p-value</td>
</tr>
<tr>
<td>Total Accuracy&lt;sup&gt;a&lt;/sup&gt; (n = 793)</td>
<td>-0.117</td>
<td>-0.123</td>
<td>-0.052</td>
<td>-0.107</td>
</tr>
<tr>
<td>Anger Accuracy&lt;sup&gt;a&lt;/sup&gt; (n = 789)</td>
<td>-0.035</td>
<td>-0.051</td>
<td>0.022</td>
<td>-0.054</td>
</tr>
<tr>
<td>Disgust Accuracy&lt;sup&gt;a&lt;/sup&gt; (n = 799)</td>
<td>-0.050</td>
<td>-0.029</td>
<td>-0.053</td>
<td>-0.032</td>
</tr>
<tr>
<td>Fear Accuracy&lt;sup&gt;b&lt;/sup&gt; (n = 801)</td>
<td>-0.048</td>
<td>-0.029</td>
<td>-0.012</td>
<td>-0.086</td>
</tr>
<tr>
<td>Sad Accuracy&lt;sup&gt;a&lt;/sup&gt; (n = 794)</td>
<td>-0.059</td>
<td>-0.090</td>
<td>0.007</td>
<td>-0.083</td>
</tr>
<tr>
<td>Surprised Accuracy&lt;sup&gt;a&lt;/sup&gt; (n = 788)</td>
<td>-0.068</td>
<td>-0.080</td>
<td>-0.052</td>
<td>-0.017</td>
</tr>
<tr>
<td>Neutral Accuracy&lt;sup&gt;a&lt;/sup&gt; (n = 780)</td>
<td>-0.085</td>
<td>-0.117</td>
<td>-0.061</td>
<td>-0.063</td>
</tr>
<tr>
<td>Total Reaction Time&lt;sup&gt;b&lt;/sup&gt; (n = 787)</td>
<td>0.009</td>
<td>0.034</td>
<td>-0.002</td>
<td>-0.065</td>
</tr>
<tr>
<td>Anger RT&lt;sup&gt;b&lt;/sup&gt; (n = 789)</td>
<td>0.016</td>
<td>0.002</td>
<td>-0.024</td>
<td>-0.022</td>
</tr>
<tr>
<td>Disgust RT&lt;sup&gt;b&lt;/sup&gt; (n = 781)</td>
<td>0.648</td>
<td>0.945</td>
<td>0.504</td>
<td>0.534</td>
</tr>
<tr>
<td>Fear RT&lt;sup&gt;b&lt;/sup&gt; (n = 792)</td>
<td>-0.028</td>
<td>-0.006</td>
<td>0.001</td>
<td>-0.101</td>
</tr>
<tr>
<td>Happy RT&lt;sup&gt;b&lt;/sup&gt; (n = 790)</td>
<td>0.347</td>
<td>0.852</td>
<td>0.972</td>
<td>0.054</td>
</tr>
<tr>
<td>Sad RT&lt;sup&gt;a&lt;/sup&gt; (n = 800)</td>
<td>-0.030</td>
<td>0.024</td>
<td>-0.018</td>
<td>-0.080</td>
</tr>
<tr>
<td>Surprised RT&lt;sup&gt;b&lt;/sup&gt; (n = 785)</td>
<td>0.007</td>
<td>0.032</td>
<td>0.030</td>
<td>-0.074</td>
</tr>
<tr>
<td>Neutral RT&lt;sup&gt;b&lt;/sup&gt; (n = 782)</td>
<td>0.850</td>
<td>0.043</td>
<td>0.713</td>
<td>0.600</td>
</tr>
<tr>
<td>Total Negative Bias&lt;sup&gt;a&lt;/sup&gt; (n = 782)</td>
<td>0.077</td>
<td>0.103</td>
<td>0.059</td>
<td>0.049</td>
</tr>
<tr>
<td>Anger Bias&lt;sup&gt;a&lt;/sup&gt; (n = 784)</td>
<td>0.053</td>
<td>0.075</td>
<td>0.031</td>
<td>0.042</td>
</tr>
<tr>
<td>Happy Bias&lt;sup&gt;a&lt;/sup&gt; (n = 788)</td>
<td>0.029</td>
<td>0.043</td>
<td>0.018</td>
<td>0.023</td>
</tr>
<tr>
<td>Sad Bias&lt;sup&gt;a&lt;/sup&gt; (n = 787)</td>
<td>0.052</td>
<td>0.062</td>
<td>0.063</td>
<td>0.039</td>
</tr>
</tbody>
</table>

<sup>a</sup>Spearman’s rank correlation coefficient (\( \rho \)) used.

<sup>b</sup>Pearson’s product-moment correlation coefficient (\( r \)) used.
Table 5 (continued)

*Note:* KDEF = Karolinska Directed Emotional Faces; SPQ total = Schizotypal Personality Questionnaire total; SPQ-CP = Schizotypal Personality Questionnaire, cognitive-perceptual subscale; SPQ-I = Schizotypal Personality Questionnaire, interpersonal subscale; SPQ-D = Schizotypal Personality Questionnaire, disorganized subscale; RT = reaction time.

* p-value significant after Holm-Bonferroni correction for multiple comparisons: accuracy tests were all one-tailed; reaction time tests were all two-tailed; bias tests were all one-tailed.
Table 6  
Correlations of KDEF Accuracy, Reaction Time and Bias with Symptoms for High-Risk Group

<table>
<thead>
<tr>
<th></th>
<th>SPQ total Correlation</th>
<th>SPQ-CP Correlation</th>
<th>SPQ-I Correlation</th>
<th>SPQ-D Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>p-value</td>
<td>p-value</td>
<td>p-value</td>
<td>p-value</td>
</tr>
<tr>
<td>Total Accuracy</td>
<td>-0.022</td>
<td>-0.291</td>
<td>0.148</td>
<td>-0.006</td>
</tr>
<tr>
<td>(n = 73)</td>
<td>.427</td>
<td>.006*</td>
<td>.105</td>
<td>.480</td>
</tr>
<tr>
<td>Anger Accuracy</td>
<td>-0.094</td>
<td>-0.131</td>
<td>0.030</td>
<td>-0.117</td>
</tr>
<tr>
<td>(n = 73)</td>
<td>.215</td>
<td>.135</td>
<td>.401</td>
<td>.163</td>
</tr>
<tr>
<td>Disgust Accuracy</td>
<td>0.095</td>
<td>-0.060</td>
<td>0.041</td>
<td>0.036</td>
</tr>
<tr>
<td>(n = 75)</td>
<td>.209</td>
<td>.303</td>
<td>.362</td>
<td>.379</td>
</tr>
<tr>
<td>Fear Accuracy</td>
<td>-0.120</td>
<td>-0.145</td>
<td>0.050</td>
<td>-0.100</td>
</tr>
<tr>
<td>(n = 75)</td>
<td>.153</td>
<td>.108</td>
<td>.336</td>
<td>.197</td>
</tr>
<tr>
<td>Sad Accuracy</td>
<td>-0.027</td>
<td>-0.144</td>
<td>0.060</td>
<td>0.157</td>
</tr>
<tr>
<td>(n = 73)</td>
<td>.411</td>
<td>.169</td>
<td>.308</td>
<td>.093</td>
</tr>
<tr>
<td>Surprised Accuracy</td>
<td>0.042</td>
<td>-0.096</td>
<td>0.038</td>
<td>0.015</td>
</tr>
<tr>
<td>(n = 71)</td>
<td>.364</td>
<td>.213</td>
<td>.376</td>
<td>.451</td>
</tr>
<tr>
<td>Neutral Accuracy</td>
<td>-0.060</td>
<td>-0.223</td>
<td>0.084</td>
<td>-0.061</td>
</tr>
<tr>
<td>(n = 73)</td>
<td>.308</td>
<td>.029</td>
<td>.239</td>
<td>.306</td>
</tr>
<tr>
<td>Total Reaction Time</td>
<td>0.202</td>
<td>0.252</td>
<td>-0.002</td>
<td>0.061</td>
</tr>
<tr>
<td>(n = 73)</td>
<td>.087</td>
<td>.031</td>
<td>.988</td>
<td>.606</td>
</tr>
<tr>
<td>Anger RT</td>
<td>0.049</td>
<td>0.060</td>
<td>-0.034</td>
<td>0.103</td>
</tr>
<tr>
<td>(n = 74)</td>
<td>.680</td>
<td>.613</td>
<td>.774</td>
<td>.383</td>
</tr>
<tr>
<td>Disgust RT</td>
<td>0.149</td>
<td>0.226</td>
<td>0.091</td>
<td>-0.065</td>
</tr>
<tr>
<td>(n = 73)</td>
<td>.209</td>
<td>.055</td>
<td>.445</td>
<td>.584</td>
</tr>
<tr>
<td>Fear RT</td>
<td>0.248</td>
<td>0.131</td>
<td>0.160</td>
<td>0.056</td>
</tr>
<tr>
<td>(n = 75)</td>
<td>.032</td>
<td>.262</td>
<td>.170</td>
<td>.630</td>
</tr>
<tr>
<td>Happy RT</td>
<td>0.021</td>
<td>0.179</td>
<td>-0.092</td>
<td>-0.046</td>
</tr>
<tr>
<td>(n = 74)</td>
<td>.659</td>
<td>.128</td>
<td>.436</td>
<td>.700</td>
</tr>
<tr>
<td>Sad RT</td>
<td>0.164</td>
<td>0.271</td>
<td>-0.073</td>
<td>0.033</td>
</tr>
<tr>
<td>(n = 75)</td>
<td>.160</td>
<td>.019</td>
<td>.536</td>
<td>.781</td>
</tr>
<tr>
<td>Surprised RT</td>
<td>0.242</td>
<td>0.188</td>
<td>-0.008</td>
<td>0.165</td>
</tr>
<tr>
<td>(n = 75)</td>
<td>.037</td>
<td>.106</td>
<td>.946</td>
<td>.157</td>
</tr>
<tr>
<td>Neutral RT</td>
<td>0.208</td>
<td>0.344</td>
<td>-0.074</td>
<td>0.047</td>
</tr>
<tr>
<td>(n = 73)</td>
<td>.078</td>
<td>.003*</td>
<td>.533</td>
<td>.693</td>
</tr>
<tr>
<td>Total Negative Bias</td>
<td>-0.007</td>
<td>0.147</td>
<td>-0.070</td>
<td>0.025</td>
</tr>
<tr>
<td>(n = 73)</td>
<td>.476</td>
<td>.107</td>
<td>.278</td>
<td>.416</td>
</tr>
<tr>
<td>Anger Bias</td>
<td>-0.104</td>
<td>0.145</td>
<td>-0.184</td>
<td>-0.038</td>
</tr>
<tr>
<td>(n = 75)</td>
<td>.188</td>
<td>.107</td>
<td>.058</td>
<td>.373</td>
</tr>
<tr>
<td>Happy Bias</td>
<td>0.088</td>
<td>0.038</td>
<td>0.004</td>
<td>0.091</td>
</tr>
<tr>
<td>(n = 70)</td>
<td>.235</td>
<td>.377</td>
<td>.486</td>
<td>.227</td>
</tr>
<tr>
<td>Sad Bias</td>
<td>-0.078</td>
<td>0.084</td>
<td>-0.057</td>
<td>-0.092</td>
</tr>
<tr>
<td>(n = 73)</td>
<td>.257</td>
<td>.241</td>
<td>.316</td>
<td>.220</td>
</tr>
</tbody>
</table>

*a* Spearman’s rank correlation coefficient (ρho) used.

*b* Pearson’s product-moment correlation coefficient (r) used.
Table 6 (continued)

*Note 1: KDEF = Karolinska Directed Emotional Faces; SPQ total = Schizotypal Personality Questionnaire total; SPQ-CP = Schizotypal Personality Questionnaire, cognitive-perceptual subscale; SPQ-I = Schizotypal Personality Questionnaire, interpersonal subscale; SPQ-D = Schizotypal Personality Questionnaire, disorganized subscale; RT = reaction time.

* p-value significant after Holm-Bonferroni correction for multiple comparisons: accuracy tests were all one-tailed; reaction time tests were all two-tailed; bias tests were all one-tailed.
Table 7
Correlations of KDEF Accuracy, Reaction Time and Bias with Symptoms for Low-Risk Group

<table>
<thead>
<tr>
<th></th>
<th>SPQ total Correlation p-value</th>
<th>SPQ-CP Correlation p-value</th>
<th>SPQ-I Correlation p-value</th>
<th>SPQ-D Correlation p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total Accuracy</strong></td>
<td>0.041</td>
<td>-0.105</td>
<td>0.106</td>
<td>0.003</td>
</tr>
<tr>
<td>(n = 184)</td>
<td>.290</td>
<td>.078</td>
<td>.076</td>
<td>.483</td>
</tr>
<tr>
<td>Anger Accuracy</td>
<td>0.118</td>
<td>0.006</td>
<td>0.131</td>
<td>0.014</td>
</tr>
<tr>
<td>(n = 183)</td>
<td>.056</td>
<td>.468</td>
<td>.039</td>
<td>.425</td>
</tr>
<tr>
<td>Disgust Accuracy</td>
<td>-0.049</td>
<td>-0.059</td>
<td>0.003</td>
<td>-0.032</td>
</tr>
<tr>
<td>(n = 184)</td>
<td>.256</td>
<td>.215</td>
<td>.484</td>
<td>.334</td>
</tr>
<tr>
<td>Fear Accuracy</td>
<td>0.087</td>
<td>-0.035</td>
<td>0.141</td>
<td>-0.014</td>
</tr>
<tr>
<td>(n = 185)</td>
<td>.119</td>
<td>.321</td>
<td>.028</td>
<td>.426</td>
</tr>
<tr>
<td>Sad Accuracy</td>
<td>0.022</td>
<td>-0.037</td>
<td>0.068</td>
<td>-0.015</td>
</tr>
<tr>
<td>(n = 183)</td>
<td>.386</td>
<td>.308</td>
<td>.180</td>
<td>.419</td>
</tr>
<tr>
<td>Surprised Accuracy</td>
<td>-0.074</td>
<td>-0.164</td>
<td>-0.043</td>
<td>0.011</td>
</tr>
<tr>
<td>(n = 183)</td>
<td>.161</td>
<td>.013</td>
<td>.284</td>
<td>.440</td>
</tr>
<tr>
<td>Neutral Accuracy</td>
<td>-0.207</td>
<td>-0.157</td>
<td>-0.148</td>
<td>-0.108</td>
</tr>
<tr>
<td>(n = 177)</td>
<td><strong>.003</strong></td>
<td>.019</td>
<td>.025</td>
<td>.076</td>
</tr>
<tr>
<td><strong>Total Reaction Time</strong></td>
<td>0.087</td>
<td>0.111</td>
<td>0.039</td>
<td>-0.019</td>
</tr>
<tr>
<td>(n = 180)</td>
<td>.246</td>
<td>.139</td>
<td>.602</td>
<td>.802</td>
</tr>
<tr>
<td>Anger RT</td>
<td>0.039</td>
<td>0.079</td>
<td>0.009</td>
<td>-0.013</td>
</tr>
<tr>
<td>(n = 179)</td>
<td>.605</td>
<td>.295</td>
<td>.909</td>
<td>.861</td>
</tr>
<tr>
<td>Disgust RT</td>
<td>0.016</td>
<td>0.046</td>
<td>0.015</td>
<td>-0.093</td>
</tr>
<tr>
<td>(n = 179)</td>
<td>.833</td>
<td>.537</td>
<td>.841</td>
<td>.216</td>
</tr>
<tr>
<td>Fear RT</td>
<td>0.061</td>
<td>0.110</td>
<td>0.002</td>
<td>-0.043</td>
</tr>
<tr>
<td>(n = 182)</td>
<td>.417</td>
<td>.140</td>
<td>.974</td>
<td>.568</td>
</tr>
<tr>
<td>Happy RT</td>
<td>0.086</td>
<td>0.100</td>
<td>0.082</td>
<td>-0.061</td>
</tr>
<tr>
<td>(n = 181)</td>
<td>.250</td>
<td>.181</td>
<td>.273</td>
<td>.413</td>
</tr>
<tr>
<td>Sad RT</td>
<td>0.057</td>
<td>0.074</td>
<td>0.050</td>
<td>-0.065</td>
</tr>
<tr>
<td>(n = 185)</td>
<td>.442</td>
<td>.320</td>
<td>.502</td>
<td>.381</td>
</tr>
<tr>
<td>Surprised RT</td>
<td>0.056</td>
<td>0.020</td>
<td>0.095</td>
<td>-0.070</td>
</tr>
<tr>
<td>(n = 179)</td>
<td>.453</td>
<td>.794</td>
<td>.207</td>
<td>.352</td>
</tr>
<tr>
<td>Neutral RT</td>
<td>0.084</td>
<td>0.053</td>
<td>0.103</td>
<td>-0.032</td>
</tr>
<tr>
<td>(n = 179)</td>
<td>.266</td>
<td>.477</td>
<td>.170</td>
<td>.670</td>
</tr>
<tr>
<td><strong>Total Negative Bias</strong></td>
<td>0.176</td>
<td>0.106</td>
<td>0.136</td>
<td>0.125</td>
</tr>
<tr>
<td>(n = 178)</td>
<td><strong>.010</strong></td>
<td>.081</td>
<td>.036</td>
<td>.048</td>
</tr>
<tr>
<td>Anger Bias</td>
<td>0.073</td>
<td>-0.005</td>
<td>0.042</td>
<td>0.150</td>
</tr>
<tr>
<td>(n = 181)</td>
<td>.165</td>
<td>.471</td>
<td>.288</td>
<td>.022</td>
</tr>
<tr>
<td>Happy Bias</td>
<td>0.002</td>
<td>-0.003</td>
<td>-0.012</td>
<td>-0.025</td>
</tr>
<tr>
<td>(n = 183)</td>
<td>.491</td>
<td>.486</td>
<td>.437</td>
<td>.369</td>
</tr>
<tr>
<td>Sad Bias</td>
<td>0.123</td>
<td>0.082</td>
<td>0.128</td>
<td>0.059</td>
</tr>
<tr>
<td>(n = 180)</td>
<td>.050</td>
<td>.136</td>
<td>.043</td>
<td>.215</td>
</tr>
</tbody>
</table>

* Spearman’s rank correlation coefficient ($\rho$) used.

b Pearson’s product-moment correlation coefficient ($r$) used.
Table 7 (continued)

Note 1: KDEF = Karolinska Directed Emotional Faces; SPQ total = Schizotypal Personality Questionnaire total; SPQ-CP = Schizotypal Personality Questionnaire, cognitive-perceptual subscale; SPQ-I = Schizotypal Personality Questionnaire, interpersonal subscale; SPQ-D = Schizotypal Personality Questionnaire, disorganized subscale; RT = reaction time.
* p-value significant after Holm-Bonferroni correction for multiple comparisons: accuracy tests were all one-tailed; reaction time tests were all two-tailed; bias tests were all one-tailed.
Table 8

**Multiple Regression for SPQ-CP and CES-D on KDEF Negative Bias**

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE B</th>
<th>β</th>
<th>R</th>
<th>R²</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Path A</strong>&lt;sup&gt;a&lt;/sup&gt; (SPQ-CP on CES-D)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>2.960</td>
<td>.130</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SPQ-CP</td>
<td>0.323</td>
<td>.023</td>
<td>.451</td>
<td>.451</td>
<td>.203</td>
<td>197.385***</td>
</tr>
<tr>
<td><strong>Path B</strong>&lt;sup&gt;b&lt;/sup&gt; (CES-D on KDEF negative bias accounting for SPQ-CP)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>0.288</td>
<td>.128</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SPQ-CP</td>
<td>0.032</td>
<td>.020</td>
<td>.065</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CES-D</td>
<td>0.067</td>
<td>.027</td>
<td>.097</td>
<td>.139</td>
<td>.019</td>
<td>7.650***</td>
</tr>
</tbody>
</table>

*Note: SPQ-CP = Schizotypal Personality Questionnaire, cognitive-perceptual subscale; CES-D = Center for Epidemiologic Studies Depression Scale; KDEF = Karolinska Directed Emotional Faces.*

<sup>a</sup> df(1, 774); <sup>b</sup> df(2, 773).

* p < .05, ** p < .01, *** p < .001, all tests were one-tailed.
Table 9
*Social Functioning Scale (SFS) Subscales by Group*

<table>
<thead>
<tr>
<th>SFS subscale</th>
<th>Groups</th>
<th>N</th>
<th>Mean Score (SD)</th>
<th>F</th>
<th>p-value</th>
<th>$\eta^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Social Engagement</td>
<td>High-Risk</td>
<td>61</td>
<td>9.93 (2.040)</td>
<td>83.691</td>
<td>.000***</td>
<td>.255</td>
</tr>
<tr>
<td></td>
<td>Low-Risk</td>
<td>185</td>
<td>12.22 (1.564)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interpersonal</td>
<td>High-Risk</td>
<td>61</td>
<td>7.38 (1.128)</td>
<td>89.586</td>
<td>.000***</td>
<td>.269</td>
</tr>
<tr>
<td></td>
<td>Low-Risk</td>
<td>185</td>
<td>8.61 (0.781)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Independence-</td>
<td>High-Risk</td>
<td>61</td>
<td>29.52 (4.979)</td>
<td>23.090</td>
<td>.000***</td>
<td>.086</td>
</tr>
<tr>
<td>Performance</td>
<td>Low-Risk</td>
<td>185</td>
<td>32.77 (4.430)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recreation</td>
<td>High-Risk</td>
<td>61</td>
<td>10.27 (4.153)</td>
<td>0.948</td>
<td>.166</td>
<td>.004</td>
</tr>
<tr>
<td></td>
<td>Low-Risk</td>
<td>185</td>
<td>10.79 (3.422)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prosocial</td>
<td>High-Risk</td>
<td>61</td>
<td>18.96 (7.871)</td>
<td>18.856</td>
<td>.000***</td>
<td>.072</td>
</tr>
<tr>
<td></td>
<td>Low-Risk</td>
<td>185</td>
<td>23.68 (7.194)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Independence-</td>
<td>High-Risk</td>
<td>61</td>
<td>34.90 (3.510)</td>
<td>66.782</td>
<td>.000***</td>
<td>.215</td>
</tr>
<tr>
<td>Competence</td>
<td>Low-Risk</td>
<td>185</td>
<td>37.71 (1.784)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* $p < .05$, ** $p < .01$, *** $p < .001$, all tests were one-tailed.
Table 10
Correlations of Schizotypal Personality Questionnaire (SPQ) with Social Functioning Scale (SFS)

<table>
<thead>
<tr>
<th></th>
<th>SPQ total</th>
<th>SPQ-CP</th>
<th>SPQ-I</th>
<th>SPQ-D</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Correlation</td>
<td>p-value</td>
<td>Correlation</td>
<td>p-value</td>
</tr>
<tr>
<td>SFS Total^b</td>
<td>-0.335</td>
<td>.000*</td>
<td>-0.109</td>
<td>.001*</td>
</tr>
<tr>
<td>(n = 846)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Social Engagement^b</td>
<td>-0.375</td>
<td>.000*</td>
<td>-0.202</td>
<td>.000*</td>
</tr>
<tr>
<td>(n = 845)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interpersonal Communication^b</td>
<td>-0.382</td>
<td>.000*</td>
<td>-0.186</td>
<td>.000*</td>
</tr>
<tr>
<td>(n = 832)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Independence- Performance^b</td>
<td>-0.253</td>
<td>.000*</td>
<td>-0.117</td>
<td>.001*</td>
</tr>
<tr>
<td>(n = 844)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recreation^a</td>
<td>-0.127</td>
<td>.000*</td>
<td>0.030</td>
<td>.000*</td>
</tr>
<tr>
<td>(n = 845)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prosocial^a</td>
<td>-0.266</td>
<td>.000*</td>
<td>-0.087</td>
<td>.006*</td>
</tr>
<tr>
<td>(n = 845)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Independent- Competence^b</td>
<td>-0.349</td>
<td>.000*</td>
<td>-0.228</td>
<td>.000*</td>
</tr>
<tr>
<td>(n = 838)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

^a Pearson’s product-moment correlation coefficient (r) used.
^b Spearman’s rank correlation coefficient (rho) used.

Note 1: SPQ-CP = Schizotypal Personality Questionnaire, cognitive-perceptual subscale; SPQ-I = Schizotypal Personality Questionnaire, interpersonal subscale; SPQ-D = Schizotypal Personality Questionnaire, disorganized subscale.

* p-value significant after Holm-Bonferroni correction for multiple comparisons; all tests were one-tailed.
<table>
<thead>
<tr>
<th></th>
<th>Total Accuracy</th>
<th>Correlation</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>SFS Total&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.016</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(n = 789)</td>
<td></td>
<td>.327</td>
<td></td>
</tr>
<tr>
<td>Social Engagement&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.042</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(n = 788)</td>
<td></td>
<td>.122</td>
<td></td>
</tr>
<tr>
<td>Interpersonal Communication&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.064</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(n = 776)</td>
<td></td>
<td>.038</td>
<td></td>
</tr>
<tr>
<td>Independence- Performance&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.057</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(n = 787)</td>
<td></td>
<td>.054</td>
<td></td>
</tr>
<tr>
<td>Recreation&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-0.009</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(n = 789)</td>
<td></td>
<td>.403</td>
<td></td>
</tr>
<tr>
<td>Prosocial&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-0.010</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(n = 789)</td>
<td></td>
<td>.394</td>
<td></td>
</tr>
<tr>
<td>Independence- Competence&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-0.002</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(n = 781)</td>
<td></td>
<td>.498</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup>Spearman’s rank correlation coefficient (rho) used.

* p-value significant after Holm-Bonferroni correction for multiple comparisons; all tests were one-tailed.
### Table 12

**Multiple Regression for SPQ Total and KDEF Total Accuracy on SFS Interpersonal Communication Subscale**

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE B</th>
<th>β</th>
<th>R</th>
<th>R²</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Path A&lt;sup&gt;a&lt;/sup&gt; (SPQ total on KDEF total accuracy)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>54.182</td>
<td>.366</td>
<td></td>
<td></td>
<td></td>
<td>8.562**</td>
</tr>
<tr>
<td>SPQ total</td>
<td>-0.112</td>
<td>.038</td>
<td>-.105</td>
<td>.105</td>
<td>.011</td>
<td></td>
</tr>
<tr>
<td>Path B&lt;sup&gt;b&lt;/sup&gt; (KDEF total accuracy on SFS interpersonal communication accounting for SPQ total)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>8.540</td>
<td>.518</td>
<td></td>
<td></td>
<td></td>
<td>66.292***</td>
</tr>
<tr>
<td>SPQ total</td>
<td>-0.113</td>
<td>.010</td>
<td>-.376</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>KDEF total</td>
<td>0.011</td>
<td>.009</td>
<td>.041</td>
<td>.383</td>
<td>.146</td>
<td></td>
</tr>
</tbody>
</table>

**Note:** SPQ = Schizotypal Personality Questionnaire; KDEF = Karolinska Directed Emotional Faces; SFS = Social Functioning Scale.

<sup>a</sup> df(1, 774); <sup>b</sup> df(2, 773).

* p < .05, ** p < .01, *** p < .001, all tests were one-tailed.
Table 13

*Multiple Regression for SPQ-CP and KDEF Total Accuracy on SFS Interpersonal Communication Subscale*

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE B</th>
<th>β</th>
<th>R</th>
<th>R^2</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Path A: (SPQ-CP on KDEF total accuracy)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>54.114</td>
<td>.314</td>
<td></td>
<td></td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>SPQ-CP</td>
<td>-0.183</td>
<td>.056</td>
<td>-.117</td>
<td>.117</td>
<td>.014</td>
<td>10.731**</td>
</tr>
<tr>
<td>Path B: (KDEF total accuracy on SFS interpersonal communication accounting for SPQ-CP)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>7.680</td>
<td>.547</td>
<td></td>
<td></td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>SPQ-CP</td>
<td>-0.081</td>
<td>.016</td>
<td>-.184</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>KDEF total accuracy</td>
<td>0.016</td>
<td>.010</td>
<td>.059</td>
<td>.200</td>
<td>.040</td>
<td>16.070***</td>
</tr>
</tbody>
</table>

*Note:* SPQ-CP = Schizotypal Personality Questionnaire, cognitive-perceptual subscale; KDEF = Karolinska Directed Emotional Faces; SFS = Social Functioning Scale.

* df(1, 774);  ** df(2, 773).

* p < .05,  ** p < .01,  *** p < .001, all tests were one-tailed.
Table 14
Multiple Regression for SPQ-D and KDEF Total Accuracy on SFS Interpersonal Communication Subscale

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE B</th>
<th>β</th>
<th>R</th>
<th>R²</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Path A&lt;sup&gt;a&lt;/sup&gt; (SPQ-D on KDEF total accuracy)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>53.771</td>
<td>.257</td>
<td></td>
<td>.096</td>
<td>.009</td>
<td>7.217**</td>
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<tr>
<td>SPQ-D</td>
<td>-0.309</td>
<td>.115</td>
<td>-0.096</td>
<td>.096</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Path B&lt;sup&gt;b&lt;/sup&gt; (KDEF total accuracy on SFS interpersonal communication accounting for SPQ-D)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>7.634</td>
<td>.536</td>
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<td></td>
<td>.054</td>
<td>21.922***</td>
</tr>
<tr>
<td>SPQ D</td>
<td>-0.197</td>
<td>.032</td>
<td>-0.218</td>
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<td></td>
</tr>
<tr>
<td>KDEF total accuracy</td>
<td>0.017</td>
<td>.010</td>
<td>0.059</td>
<td>.232</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: SPQ-D = Schizotypal Personality Questionnaire, disorganized subscale; KDEF = Karolinska Directed Emotional Faces; SFS = Social Functioning Scale.
<sup>a</sup> df(1, 774); <sup>b</sup> df(2, 773).

* p < .05, ** p < .01, *** p < .001, all tests were one-tailed.
FIGURES

Figure 1. Mediation Model: Facial Affect Recognition (Karolinska Directed Emotional Faces; KDEF) Mediating the Relationship between Schizotypal Traits (Schizotypal Personality Questionnaire; SPQ) and Social Functioning (Social Functioning Scale; SFS)

Figure 2. Mediation Model: Depressive Symptoms (Centers for Epidemiologic Studies Depression Scale; CES-D) Mediating the Relationship between Schizotypal Traits (Schizotypal Personality Questionnaire; SPQ) and Negative Bias (Karolinska Directed Emotional Faces; KDEF)
REFERENCES CITED


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