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Social Connectedness Deficits in College Students with Schizotypy

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SOCIAL CONNECTEDNESS DEFICITS IN COLLEGE STUDENTS WITH SCHIZOTYPY

A Dissertation

Submitted to the Graduate Faculty of the
Louisiana State University and
Agricultural and Mechanical College
In partial fulfillment of the
Requirements for the degree of
Doctor of Philosophy
in

The Department of Psychology

by
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Abstract

Schizophrenia is a devastating disorder characterized by a variety of bizarre behaviors as well as deficits in neurocognition, social cognition, and functioning. This study focuses on individuals with schizotypy—those with the purported genetic liability for schizophrenia that do not display the full disorder. Prior research has identified potential risk factors for schizophrenia by studying this population, including deficits in social cognition. Studies of social cognition in individuals with schizotypy, however, have yielded inconsistent findings that have failed to fully explain the range of functional deficits seen in these individuals. Social connectedness, in contrast, may be a more useful risk factor and may better explain these deficits. Specifically, individuals with schizotypy may have low levels of social connectedness which leads to poor functioning, odd social behaviors, and social cognitive deficits. To examine this hypothesis, 39 individuals with schizotypy and 41 healthy controls were included in this study. Individuals with schizotypy reported significantly lower levels of social connectedness than controls. A model of the relationship between schizotypy and outcome— defined as social competence, quality of life, and general psychopathology symptoms as measured by the Brief Symptom Inventory (BSI; Derogatis & Melisaratos, 1983)—in which social connectedness was a mediator was evaluated. Social connectedness mediated the relationship between schizotypy and poorer objective and subjective quality of life, but not when social competence and BSI symptoms were the outcomes. Finally, specific schizotypy traits and their relationship to social connectedness were considered. Negative schizotypy was significantly related to social connectedness. Social connectedness appears to be an important feature of the schizophrenia spectrum especially when considering quality of life. Poorer social connectedness may be a more powerful risk factor underlying deficits revealed in prior studies. A primary deficit in social connectedness may also explain

why research examining specific deficits on performance based tasks such as in social cognition studies has found inconsistent evidence for deficits in individuals with schizotypy. Results and implications for the conceptual understanding of schizotypy are discussed, and recommendations are made for future studies of social connectedness in the schizophrenia spectrum.

Literature Review

Schizophrenia

Schizophrenia is a devastating disorder characterized by impaired reality testing, odd behaviors, and marked social dysfunction (American Psychiatric Association, 2000). It is fairly uncommon, with an estimated 0.4% prevalence (Wu, Shi, Birnbaum, Hudson, & Kessler, 2006). The impact of this disorder on society as a whole, however, is substantial. In 2001, the World Health Organization named schizophrenia among the top ten causes of healthy life lost to disability (WHO, 2001). Individuals with schizophrenia may experience severe and debilitating symptoms that lead to extended periods of psychiatric hospitalization, a lifetime of treatment with psychotropic medication, lack of educational and occupational attainment, and poor interpersonal relationships. The disorder has been recognized by physicians and researchers for over a decade (Bleuler, 1950; Kraepelin, 1919/1971; Morel, 1890). It was once thought to entail progressive deterioration and was originally named *démence précoce* or “early dementia” (Morel, 1890). The disorder was given its present name, schizophrenia meaning “split mind,” by Bleuler (1950) who described its manifestation as the “splitting of psychic functions.” At present, the American Psychiatric Association’s diagnostic manual, generally considered the authority for diagnosis of psychiatric disorders in the United States, defines schizophrenia by the presence of delusions, hallucinations, disorganized speech and behavior, and other symptoms causing social or occupational dysfunction which are present for at least six months with at least one month of active symptoms (APA, 2000).

Symptoms in schizophrenia.

Individuals with a diagnosis of schizophrenia may display dramatically different symptom presentations and illness courses. They may experience acute episodes of psychosis

with prominent delusions and hallucinations, chronic and less blatant symptoms such as lack of motivation and social withdrawal, and/or severe cognitive and emotional deficits (Andreasen, 1997). A range of different symptoms may be manifest in an individual simultaneously, not all patients display all symptoms, and there is no single symptom that is always present or is specific to the disorder (APA, 2000). In fact, efforts to identify disorder-specific criteria have been largely unsuccessful (e.g. first rank symptoms, Schneider, 1959 and Nordgaard, Arnfred, Handest, & Parnas, 2008). The illness course also varies over time, for example, one may be afflicted by striking but intermittent acute psychotic episodes followed by periods of clinical stability or by chronic symptoms such as lack of motivation and emotional expression with transient delusions and hallucinations (Gerbaldo, Cassady, & Helisch, 1995).

There have been two broad strategies for reducing and characterizing this remarkable heterogeneity of symptoms. The first involves identifying different disease processes within schizophrenia that reflect different etiologies, raising the argument of “lumping” versus “splitting” (McKusick, 1969). “Splitters” argue that schizophrenia is not a single disorder but a collection of disorders with separate etiologies (Crow, 1980). Absent evidence of separate etiologies and considering that separate syndromes can occur simultaneously in the same individual, the most parsimonious solution may be to “lump” these syndromes into a single category reflecting a single disorder (McKusick, 1969; Crow, 1985; Gottesman, McGuffin, & Farmer, 1987). Nevertheless, a number of taxonomies have been put forward delineating distinct subtypes of the illness. For example, very early in the history of schizophrenia, distinctions were made between “process” and “reactive” types (Kantor, Wallner, & Winder, 1953). Crow (1985) also noted two types of schizophrenia: Type I and Type II. The DSM-IV-TR addresses the issue of heterogeneity by defining subtypes including paranoid, disorganized, residual, and

undifferentiated (APA, 2000). DSM-IV-TR subtypes, however, are not particularly reliable or stable (Blashfield, 1973; Gruenberg, Kendler, & Tsuang, 1985), and the majority of patients fall into the “undifferentiated” category (Kendler, 1985).

A second strategy for understanding and potentially reducing heterogeneity is by use of a statistical method. Research suggests that particular symptoms tend to cluster together. The most accepted model distinguishes three different symptom clusters and comes from a factor analysis done by Liddle (1987) that found positive, negative, and disorganization factors. There has been substantial research support for this factor structure (Andreasen, Arndt, Alliger, Miller, & Flaum, 1995; Malla, Norman, Williamson, & Cortese, 1993). Positive symptoms include delusions and hallucinations and reflect an exaggeration of behaviors present in non-disordered individuals. Negative symptoms reflect the absence of behaviors normally present in non-disordered individuals. These include flat or blunted affect (diminished emotional expression), avolition (lack of motivation), anhedonia (decreased ability to experience pleasure), and alogia (diminished speech). Disorganization symptoms include disorganized speech, disorganized or bizarre behaviors, and inappropriate affect. Positive symptoms are the least stable (Fenton & McGlashan, 1991), respond best to medications (Tandon et al., 2008), and are not a good indicator of prognosis (Addington & Addington, 1991; Strauss, Carpenter, & Bartko, 1975). Negative symptoms are the most stable and generally do not respond well to treatment (Arndt et al., 1995) with the exception of those secondary negative symptoms that occur in response to positive or other mood symptoms (Arango, Buchanan, Kirkpatrick, & Carpenter, 2004; Carpenter, Heinrichs, & Alphas, 1985; Goldberg, 1985). Less is known about how disorganization symptoms relate to functioning and other symptoms. They are positively correlated with deficits in executive functioning and attention (Kerns & Berenbaum, 2002;

Moritz et al., 2001) as well as social information processing deficits (Brune, 2003; Shean, Murphy, & Meyer, 2005).

Neurocognition in schizophrenia.

Neurocognitive deficits are a hallmark of schizophrenia. Early in the history of schizophrenia research, these deficits were identified as important features. Bleuler (1950) and Kraepelin (1971) observed deficits in attention, perception, and cognition. Other researchers described a generalized deficit in all neurocognitive tasks (Chapman & Chapman, 1973). Specific domains of deficiency in schizophrenia include executive functioning (Hutton, Puri, Duncan, Robbins, Barnes, & Joyce, 1998), verbal memory (Braff, 1993), and attention (Stirling, Hellewell, & Hewit, 1997). A seminal review article emphasized the importance and impact of neurocognitive deficits (Green, 1996). This review revealed that negative symptoms predict functional outcome, but positive symptoms do not. Neurocognitive deficits, however, were the best single predictor of functional outcome in schizophrenia. Specifically, Green (1996) suggested there were neurocognitive “rate limiting factors” that prevent disordered individuals from acquiring more advanced skills. The effect size of the relationship between neurocognition and functioning is not overwhelming (effect sizes range from $d = 0.20$ to 0.40 in cross-sectional studies [Green, Kern, Braff, & Mintz, 2000] and are smaller in longitudinal studies [Miley, Ho, Arndt, & Andreasen, 2005]) but is stable (Addington & Addington, 2000). Further research has shown that the pathway from neurocognitive deficits to functioning may be best understood as mediated by social cognition.

Social Cognition

Social cognition is the way people think about themselves and others (Penn, Sanna & Roberts, 2008) and includes social perception, interpretation, and processing (Penn, Corrigan,

Bentall, Racenstein, & Newman, 1997). A major theory from the social psychology literature about the relationship between social and nonsocial cognition is the *building-block* theory (Ostrom, 1984; Penn et al., 1997). According to this theory, nonsocial and social cognition are related but represent different levels of analysis. Basic cognitive processes provide the foundation for social cognitive processing. Neurocognitive deficits are a limiting factor in functioning, but social cognition has a more precise relationship to functioning because how an individual understands social behavior and interprets the world is more closely related to his or her behavior. This theory contrasts with other social cognition theories that conceptualize social and nonsocial cognition as identical processes (Ostrom, 1984).

Social cognition in schizophrenia.

Across the board, studies have revealed broad cognitive deficits in schizophrenia, including deficits in social cognition. This includes deficits in emotion perception (Edwards, Jackson, Pattison, 2002; Kohler & Brennan, 2004; Mandal, Pandey & Prasad, 1998), social knowledge (Hellewell & Whittaker, 1998), theory of mind (Brune, 2005; Harrington, Siegert, & McClure, 2005), and attribution style (Bentall, Corcoran, Howard, Blackwood, & Kinderman, 2001; Garety & Freeman, 1999). As mentioned above, social cognitive deficits are more closely related to the functional deficits seen in schizophrenia. This has been supported in studies of the relationship between social cognitive abilities and functional skills such as behavior in the hospital (Mueser et al. 1996; Penn et al, 1996), social skills (Bellack et al, 1992; Ihnen, Penn, Corrigan, & Martin, 1998), and interpersonal problem solving (Toomey, Wallace, Corrigan, Schuldberg, & Green, 1997). Although it is possible that social cognitive impairment in schizophrenia is redundant with deficits in basic neurocognition, a body of literature suggests that this is not the case. Green and Horan (2010) summarize some of this literature and conclude

that while social cognition and neurocognition may be inextricably linked, social cognition does appear to play a unique role in schizophrenia.

Green and Nuechterlein (1999) proposed a model of the relationship between neurocognition and functional outcome in which social cognition was a mediator. Further research supports this model in that social cognition and neurocognition are related but separate factors, and social cognition contributes variance to functioning above and beyond the contribution of neurocognition (Brekke, Kay, Lee, & Green, 2005; Sergi, Rassovsky, Nuechterlein, & Green, 2006). The evidence, therefore, seems to suggest that neurocognitive abilities may affect the quality of an individual's social abilities and interactions, and this, in turn, is something that influences overall functioning and life quality.

Social cognition and risk for schizophrenia.

As researchers began to examine the impact of social cognition on symptoms and functioning in schizophrenia, interest emerged in the idea that social cognitive deficits could be a potential endophenotype for schizophrenia spectrum disorders. The notion of endophenotypes originated when researchers began to look for genetic bases of psychiatric disorders. Disorders classified based on phenotypes consisting of various combinations of symptoms were not useful in identifying the supposed underlying genetic bases. Endophenotypes, in contrast to phenotypes, are more closely connected to genes, lying between genotype and phenotype, reflecting the intermediate connection between complex behaviors and biological and genetic underpinnings. Endophenotypes are not detectable to the naked eye or ear but can be measured with more complex methods. They can be physiological, psychological, biochemical, neuroanatomical, or endocrinological (Gottesman & Gould, 2003; Gottesman, 1991). By deconstructing phenotypes into specialized and more elementary components, one purportedly

reduces the number and complexity of the underpinning genes, making the search for candidate genes simpler. Gottesman and Gould (2003) laid out specific criteria for determining whether a particular a trait is a useful endophenotype. Potential endophenotypes should: 1) be associated with the trait in the population, 2) be heritable, 3) be present whether the trait is present or not, 4) co-segregate with the trait imperfectly within families, and 5) be found in unaffected relatives at a higher rate than in the general population (Gottesman & Hanson, 2005).

Empirical studies have identified several possible endophenotypes for schizophrenia including sensory motor gating, eye-tracking dysfunction, and working memory (Gottesman & Gould, 2003). In light of the research suggesting that social cognition might be more closely linked to dysfunction in schizophrenia, research began to seek out social cognitive endophenotypes for schizophrenia as well. Studies have examined a number of social cognitive abilities including theory of mind, facial emotion recognition, and social knowledge as potential endophenotypes for schizophrenia. Results have been mixed, and social cognitive endophenotypes have failed to fulfill Gottesman and Hanson's (2005) endophenotype criteria. While studies have shown that social cognitive deficits are associated with schizophrenia (Penn et al., 2008), and evidence of some heritability exists (Alfimova, Abramova, Barhatova, Yumatova, Lyachenko, & Golimbet, 2009; Eack et al., 2010; van Buuren, Vink, Rapcencu, & Kahn, 2011), a number of studies have failed to demonstrate that the deficits are present in situations where the disorder is not—namely in at-risk populations (Gibson, Penn, Prinstein, Perkins, & Belger, 2010; Janssen, Versmissen, Campo, Myin-Germeys, van Os, & Krabbendam, 2006; Jahshan, & Sergi, 2007). The deficits that are seen in relatives also appear to manifest to a lesser degree than in those with the disorder (Alfimova et al., 2009; Bass, van't Wout, Aleman, & Kahn, 2008). If social cognition were indeed an endophenotype, one would expect to find

similar social cognitive deficits in individuals who possessed the genetic diathesis for schizophrenia across tasks, regardless of whether symptoms were present. Several issues complicate this body of literature.

Schizotypy

It has been proposed that schizophrenic symptoms are multidimensional and are present at subclinical levels in individuals who possess the underlying genetic vulnerability. Rado (1956; Rado & Daniels, 1956) first used the term “schizotype” to refer to individuals who manifested the schizophrenic phenotype. Rado (1956) viewed schizophrenia symptoms as continuous, manifesting in an attenuated form in some individuals. Meehl (1962) refined this theory, focusing on what he called the “schizogene,” a gene affecting brain development. Having this gene results in an integrative neural deficit and produces a central nervous system anomaly, which Meehl (1962) termed “schizotaxia.” The effect of schizotaxia in interaction with social learning and other environmental influences produces a particular personality organization—schizotypy. Three phenotypic outcomes are possible for a schizotype: 1) asymptomatic, 2) schizophrenia spectrum disorder, or 3) schizophrenia. According to Meehl’s model, about ten percent of the population has the genetic vulnerability, but only a small subset will develop clinically-defined schizophrenia (Meehl, 1962; Lenzenweger & Korfine, 1992). It is notable also that later conceptualizations of this model include polygenetic influences rather than a single gene (Lenzenweger, 2006). The most relevant aspect of Meehl’s model is that those who do not decompensate show the genetic liability as subtle aberrations in psychological and neurocognitive processes that can be detected with specialized measurement techniques. This theory emphasizes the notion of endophenotypes detectable in healthy but genetically vulnerable individuals. Meehl’s risk signs included cognitive slippage, interpersonal

aversiveness, anhedonia, and ambivalence (Meehl, 1962; 1990). These signs result from the central nervous system anomaly and are present in all who possess the genetic vulnerability regardless of whether symptoms are manifest. Empirical research in schizotypy has supported Meehl's model. Schizotypes can be reliably identified (Raine, 1991), taxometric studies indicate that a subset of the population exhibits a schizotypal personality organization (Horan, Blanchard, Gangestad, & Kwapil, 2004), and this subset is at increased risk for schizophrenia and related disorders (Chapman, Chapman, Kwapil, Ekblad, & Zinser, 1994; Gooding, Tallent, & Matts, 2005).

Schizotypy traits.

Schizotypy research has revealed dimensional traits similar to schizophrenia symptoms (Rossi & Daneluzzo, 2002; Wuthrich & Bates, 2006). Kerns (2006) found that a three factor model of schizotypy including positive, negative, and disorganized traits exhibited a good fit. This study included extensive schizotypy measures, assessing the full range of dimensional traits. Schizotypy dimensions closely resemble those of schizophrenia, only not severe enough to meet clinical threshold (Raine, Reynolds, Lencz, Scerbo, Triphon, & Kim, 1994). Positive traits include ideas of reference, magical thinking, and paranoid ideation. Negative schizotypy includes having no close friends and constricted affect. Disorganized schizotypy is characterized by oddities of speech and behavior such as speech that uses vague or unclear references. Just as in schizophrenia, individuals may exhibit different degrees of each trait with different levels of functional impairment, including some that show almost no impairment.

Measurement of schizotypy.

Current research methods usually identify individuals with schizotypy in one of four ways: biological relatives of individuals with schizophrenia, individuals in the prodromal phase

(or ultra-high risk), individuals with schizotypal personality disorder, or individuals identified by psychometric methods. Studies using biological relatedness identify individuals with schizophrenia and assume biological relatives possess the genetic vulnerability (twins, siblings, parents). This method finds support in studies that have found relatives are at increased risk for schizophrenia and display subclinical symptoms (Baron et al., 1985; Erlenmeyer-Kimling & Cornblatt, 1987; Thaker, Adami, Moran, Lahti, & Cassidy, 1993). Another method is the ultra-high risk method which identifies individuals who are on the verge of decomposition into schizophrenia (Simon et al., 2006). These individuals are already experiencing psychotic symptoms and some functional impairment but have not yet deteriorated to the point of meeting diagnostic criteria. This includes individuals with intermittent psychosis of a short duration, symptoms not intense enough to meet criteria, and individuals with combinations of trait and state related factors that place them at high risk (Yung et al., 2003). Some research also uses diagnostic criteria for schizotypal personality disorder in which there is a clear diagnostic threshold, with symptoms either being present or not. Particular problems with these methods are a lack of efficiency and that they capture only those individuals on the more severe end of the schizotypy spectrum, largely ignoring those who never decompensate. This is important because, according to Meehl's model, the majority remain healthy. Finally, researchers also use a psychometric risk paradigm. Psychometric methods identify schizotypes based on behavioral or self-report measures of the signs of schizotypy. This method has been used in many studies and has proven to efficiently capture a sample of individuals who are at greater risk for developing schizophrenia and schizophrenia-spectrum disorders (Chapman et al., 1994; Gooding, Tallent, & Matts, 2007; Kwapil, Miller, Zinser, Chapman, & Chapman, 1997).

Moreover, a broad range of relatively healthy individuals are included, allowing one to study genuine risk factors that are not likely to be confounded with symptomatic dysfunction.

One of the most widely used psychometric measures for identifying schizotypal individuals is the Schizotypal Personality Questionnaire (SPQ). Raine (1991) designed this questionnaire to mirror DSM-III symptoms of schizotypal personality disorder. While other scales measure a limited number of symptoms (positive symptoms, speech disturbances, etc.), the SPQ assesses a range of symptoms, including all nine features of schizotypal personality disorder: ideas of reference, excessive social anxiety, odd beliefs/magical thinking, unusual perceptual experiences, odd/eccentric behavior, no close friends, odd speech, constricted affect, and suspiciousness. Although the items mirror schizotypal personality disorder symptoms, the instrument assesses a broad range of subclinical pathology rather than just clinically significant symptoms. The SPQ has demonstrated good internal reliability ($\alpha = .91$) and test-retest reliability ($r = .82$). Further, it demonstrates criterion related validity in that high scorers are much more likely to meet criteria for a diagnosis of schizotypal personality disorder, and all individuals with schizotypal personality disorder obtain high scores (Raine, 1991). Raine (1991) also examined correlations between other schizotypy scales and scales related to schizotypy but not measuring DSM-III schizotypal symptoms and found evidence for convergent and discriminant validity. The SPQ has also been factor analyzed, resulting in three separate dimensions similar to those found in schizophrenia (Reynolds, Raine, Mellinger, Venables, & Mednick, 2000; Wuthrich & Bates, 2006).

This body of research indicates schizotypy is a construct similar in structure to schizophrenia, it reflects a vulnerability to schizophrenia, and it can be identified via relatively brief self-report questionnaires. Research in schizotypy allows researchers to avoid many

confounds associated with chronic mental illness, including severe cognitive deficits, medication effects, hospitalization, and acute psychosis. One can also begin to examine potential endophenotypes of the disorder for the purpose of determining etiology and predicting who will develop the full disorder.

Neurocognition in schizotypy.

Studies of schizotypy have revealed a wide range of neurocognitive deficits, although these deficits are not as large as those found in schizophrenia (Siever & Davis, 2004). Particular areas of impaired neurocognition are similar to those found in individuals with schizophrenia: verbal memory, attention, and executive functioning (Sitskoorn, Aleman, Ebisch, Appels, & Kahn, 2004). This is true in studies that have examined individuals with schizotypal personality disorder (Roitman, Bergman, & Obuchowski, 1997; Siever et al., 2002; Voglmaier, Siedman, Niznikiewicz, Dickey, Shenton, & McCarley, 2005), biological relatives of individuals with schizophrenia (Laurent et al., 2000; Sitskoorn et al., 2004;), ultra-high risk samples (Brewer et al., 2005; Wood et al., 2003), and psychometric high risk samples (Barrantes-Vidal, Fananas, Rosa, Caparros, Riba, & Obiols, 2002; Bergida & Lenzenweger, 2006). One caveat of this literature is that studies of college students with schizotypy have often not found evidence for general neurocognitive deficits relative to normal controls (Chun, Minor, & Cohen, 2013; Jahshan & Sergi, 2007; Lenzenweger & Gold, 2000). In summary, although broad neurocognitive deficits have been identified in individuals with schizotypy and appear to be associated with risk for schizophrenia, these deficits are not as profound as those found in individuals with schizophrenia.

Social cognition in schizotypy.

Investigation of social cognition in individuals at risk for schizophrenia has offered less than satisfying results. While at-risk individuals show deficits in some studies, these deficits are not consistent across studies, are small in magnitude, and are often limited to selected domains. Studies have offered evidence for abnormalities in emotion recognition (Abbott & Byrne, 2013; Amminger et al., 2012; Brown & Cohen, 2010; Germine & Hooker, 2011; Kee, Horan, Mintz, & Green, 2004; Pinkham et al., 2007), but this is often not in the form of performance deficits and may instead reflect biased processing (Couture, Penn, Addington, Woods, & Perkins, 2008; Eack et al., 2010; Mikhailiova et al., 1996;). Other studies have demonstrated no deficit in schizotypal individuals (Toomey & Schulberg, 1995; Toomey, Seidman, Lyons, Faraone, & Tsuang, 1999; Li, Chen, Tang, Li, Xiao, Yin, & Wang, 2010; Pinkham, Penn, Perkins, Graham, & Siegel, 2007; Jashan & Sergi, 2007; Thompson et al., 2012) or a deficit that may be better explained by neurocognitive deficiencies (Pooreh et al., 1994). With respect to social inferences and theory of mind, the literature has been similarly inconclusive with many studies showing deficient performance (Irani, Platek, Panyavin, Calkins, Kohler, Siegel, 2006; Johnstone, & Lawrie, 2006; Kelemen, Must, & Benedek, 2004; Marjoram, et al., 2008; Pickup, 2006) but not all (Couture et al., 2008; Fernyhough, Jones, Whittle, Waterhouse, & Bentall, 2008; Jashan & Sergi, 2007). Another domain of social cognition that has been examined is social perception. Many of these studies have revealed poorer performance (Pinkham et al., 2007; Toomey et al., 1999; Miller & Lenzenweger, 2012) but not all (Thompson et al., 2012), and this is an area that has been less frequently studied. Finally, research has considered attribution style. There have been a couple studies that reported evidence that individuals with schizotypy make deviant attributions for social or interpersonal events (Levine, Jonas, & Serper, 2004; An, Kang, Park, Kim, Lee, & Lee,

2010). Taking all of these results into consideration, it is possible social cognitive deficits represent state-like variations linked to psychotic exacerbation, individuals are able to employ compensatory mechanisms to accomplish social cognitive tasks, or that social cognitive deficits are either not present or are very mild. This is disconcerting given the wealth of research showing that functional social deficits are a major feature of schizophrenia independent of acute symptoms (Addington & Addington, 2003; Combs, Waguspack, Chapman, Basso, & Penn, 2011; Johnstone, MacMillan, Frith, & Benn, 1990; Wykes, Sturt, & Katz, 1990). Furthermore, early theories of schizophrenia risk posit that deficits arise primarily in the social domain due to the nature of the dysfunction and characteristics of the social world (Meehl, 1990). Less severe presentations such as Cluster A Personality disorders are also characterized by interpersonal dysfunction and odd social behaviors (APA, 2000; O'Donohue, Fowler, & Lilienfeld, 2007). It seems that a deficit in specific social cognitive abilities fails to adequately explain the range of social dysfunctions seen in the schizophrenia spectrum. Potentially, social cognition and social abilities may be linked to the schizophrenia spectrum less directly. One possible explanation of the discrepancy between results obtained in individuals at risk and decompensated individuals may be that another, lower order or more intuitive variable functions as the predisposing factor. A less specific, trait-like factor present early in development may more adequately explain the broad and variable social deficits exhibited in schizophrenia. It may be this variable that, in combination with poor or waning social cognitive and neurocognitive abilities, contributes to the range of symptoms and social difficulties seen in the schizophrenia spectrum. Social connectedness is this type of variable.

Social Connectedness

Social connectedness is a stable individual difference that reflects the awareness and internalized experience of interpersonal closeness in relationships with family, friends, strangers, community, and society (Lee & Robbins, 2000). Social connectedness can be conceptualized as the way an individual views his or her self in relation to the social world, as emotionally connected or disconnected. Lee and Robbins describe connectedness as “the ability to feel comfortable within a social context larger than family or friends.” The roots of connectedness are in self psychology, a psychoanalytic theory focusing on the development of self-concept (Kohut, 1984).

According to Lee and Robbins (1995; 2000), connectedness is a piece of the larger construct of belongingness. It begins in infancy and continues developing throughout life. The initial stage, companionship, occurs when the infant bonds with a nurturing parent. This later extends to close others or objects such as toys. The next stage, affiliation, emerges in response to the demands of adolescence in which the sense of self must extend beyond the primary caregiver to similar peers. The final and most advanced stage, connectedness, characterizes an individual comfortable in social roles and responsibilities and able to identify with others perceived as different.

Problems arise when needs are not met somewhere in the developmental progression of connectedness. An infant whose companionship needs are not fulfilled develops a fragile sense of self, low self-esteem, and isolates to avoid rejection. Children whose affiliation needs are not met may be able to maintain relationships with single close others but have difficulty maintaining a sense of self in larger groups of friends or family without the reassuring presence of the close other. Finally, if connectedness needs are not met, individuals experience feelings of being different or distant from others and frustration with the sense that others do not understand

him or her. In addition, individuals may isolate or develop fantasies about finding a place to belong and reject more realistic social roles.

Social connectedness is conceptualized as primarily subjective in nature. As such, social connectedness has been measured via self-report. The measure created by Lee and Robbins was specifically developed as part of a larger effort to measure belongingness. Items were written based on Kohut's (1984) definition of connectedness and evaluated by a panel of judges for content validity. The entire scale was administered to a large sample of undergraduates ($n = 626$). The sample was split, and a principal components analysis was performed to extract the significant factors. Items were also evaluated based on social desirability with the Marlowe Crown Social Desirability Scale (Crowne & Marlowe, 1960). Those deemed to be influenced by this response style were eliminated along with those not loading on a major factor. Next, the scale was cross validated with a confirmatory factor analysis on the second half of the sample. Based on these analyses, the authors identified two separate scales, one measuring social connectedness and the other social assurance. The social connectedness scale includes those items reflecting a "general emotional distance between self and others" and applies to relationships even with close friends (Lee & Robbins, 1995).

Though there has been little research to date on this conceptualization of social connectedness, some findings have emerged. First and foremost, empirical studies offer evidence for connectedness as a construct distinct from loneliness (Lee & Robbins, 1998), which is thought of as a result of poor connectedness, rather than a personality characteristic. Furthermore, connectedness is distinct from social support (Lee & Robbins, 1995), which is an environmental variable rather than an internal trait. Though connectedness seems akin to Bowlby's attachment theory (Ainsworth, 1989; Bowlby, 1988), it is much broader. Attachment

is one aspect of connectedness or connectedness in one specific domain (primary caregiver).

Finally, studies of connectedness in healthy adults reveal sex differences. Men tend to derive connectedness from relationships that emphasize social comparison and women from relationships that emphasize intimacy and physical proximity (Lee & Robbins, 2000).

Connectedness in relation to psychopathology has also been examined in a few studies. Poor social connectedness is associated with increased trait anxiety (Lee & Robbins, 2000), loneliness, social distress and avoidance, depression, hostility, social discomfort, difficulty with intimacy, sociability and assertiveness, and submissiveness (Lee, Draper, & Lee, 2001). Lee and colleagues (2001) theorized that individuals low in connectedness engage in dysfunctional interpersonal behaviors as a protective mechanism against social rejection. Another study (Williams & Galliher, 2006) showed that connectedness was important for understanding the relationship of depression and self-esteem to social support, suggesting that this relationship was mediated by connectedness. Finally, social connectedness was tested as a mediator of the relationship between extraversion and well-being (Lee et al., 2008). These authors found that, in a normal college student sample, social connectedness indeed was a mediator and explained that social connectedness allows extraverted individuals to maintain well-being across different social situations. These studies suggest that connectedness may be important for understanding a range of psychopathology.

Social connectedness in the schizophrenia spectrum.

So how might social connectedness play a role in the schizophrenia spectrum? First and foremost, the social domain represents an important aspect of how theorists conceptualize schizophrenia spectrum symptomology. Meehl (1962; 1990) hypothesized that the unique aspects of the social world and its reinforcement patterns makes social situations the major area

in which schizotypic tendencies emerge. First, these situations tend to provide random reinforcement, making learning more difficult. Second, Meehl's concept of "aversive drift," which is a characteristic feature of schizotypy (1962), makes these individuals more sensitive to aversive conditioning and, therefore, their behavior may be unduly influenced by unpleasant experiences. Individuals with schizotypy tend to be more readily conditioned by negative social feedback, and social stimuli tend to drift toward being experienced as neutral or negative. Finally, social withdrawal, another feature of schizotypy, limits opportunities for positive reinforcement and corrective learning experiences.

Descriptions of schizophrenia and related disorders also emphasize poor social relatedness (negative symptoms) and abnormal social behaviors (disorganized behavior, odd speech, suspiciousness of others) as defining symptoms. A primary deficiency in social relatedness may be an especially powerful factor contributing to these symptoms. The major area of functional disruption in the schizophrenia spectrum is undoubtedly the social realm. One might hypothesize that poorly connected individuals fail to perform specifically in the social domain and show symptoms that make them appear socially odd or awkward. It could be specifically the lack of connectedness that leads to deviant social behaviors. Lower connectedness may prevent individuals from developing the emotional and cognitive attachments that aid in learning social skills and interpreting social information. Developmental and neuropsychological brain damage studies suggest that specific structures have evolved for processing social stimuli, such as the fusiform gyrus for facial processing (McCarthy, Puce, Gore, & Allison, 1997; Onitsuka et al., 2003; Wojciulik, Kanwisher, & Driver, 1998;) and the increased salience of social/emotional stimuli that aids in attention and memory (Corrigan & Penn, 2001; Fiske & Taylor, 1991; Forgas, 1995; Penn et al., 1997). Strong connections to the

social world may be essential for normal and advanced development of these abilities. Given the range of traits seen in schizotypy (i.e. no close friends, social anhedonia, suspiciousness, constricted affect, and odd speech and behavior), the hypothesis of a primary deficit in social connectedness is potentially a powerful explanation. This hypothesis also allows for the possibility that poorly connected individuals may develop cognitive strategies to compensate for these deficits. For example, someone who has difficulty intuitively determining facial affect and body language could develop particular rules for these situations that may function, albeit awkwardly, or he may learn to rely more on contextual information rather than perceiving relevant social cues.

As stated above, research has yet to examine social connectedness in schizotypy and how it may contribute to functional deficits and symptoms. First and foremost, however, any examination of schizotypy must contend with the problem of social cognitive and neurocognitive deficits. It has been demonstrated that these are major features of the schizophrenia spectrum even though their causal roles have yet to be elucidated. In investigating social connectedness, it is important to consider these variables and their relationships to symptoms and social competence. In the current study, these will be studied as control variables when considering the relationship between schizotypy and social connectedness. Also, functioning can be measured in a number of ways. Objective measures are necessary to assess real world, observable functional deficits, however, self-report measures have the potential to offer a more sensitive assessment of problems as individuals may be able to report difficulties that do not manifest consistently or for which individuals may have developed compensatory behaviors. Since a schizotypy population is hypothesized to be relatively healthy without severe deficits, both types of measures are informative. The current study is designed to consider these factors and the role of social

connectedness in the schizotypy population. This study first examines whether social connectedness reflects an important feature of the schizotypy. It next considers whether social connectedness contributes to understanding of social and life dysfunction seen in individuals with schizotypy. Specifically, this study considers whether dysfunction might arise from a primary deficit in social connectedness such that social connectedness mediates the relationship between schizotypy and important functional variables: social competence, quality of life, and general psychopathology symptoms. This is considered while controlling for deficits thought to be characteristic of schizotypy population: neurocognition and social cognition—measured via an emotion recognition task as further discussed in the measures section.

Hypotheses

1. Based on prior research in schizotypy, I predict there will be differences between the schizotypy and control groups in neurocognition, emotion recognition, quality of life, social competence, and general psychopathology symptoms.
 - a. Individuals with schizotypy will perform significantly more poorly on a measure of neurocognition than healthy controls
 - b. Individuals with schizotypy will perform significantly more poorly on a measure of emotion recognition than healthy controls.
 - c. Individuals with schizotypy will perform significantly more poorly on a measure of social competence than healthy controls.
 - d. Individuals with schizotypy will report significantly lower quality of life than normal controls.
 - e. Individuals with schizotypy will report significantly more severe general psychopathology symptoms than normal controls.
2. I predict there will be differences between the schizotypy and control groups on social connectedness.
 - a. Schizotypy group membership will be associated with lower social connectedness.
 - b. This will remain significant when controlling for neurocognition and emotion recognition.
3. I predict there will be significant relationships between social connectedness, neurocognition, emotion recognition, social competence, quality of life, and general psychopathology symptoms within the entire sample.

- a. Higher performance on measures of neurocognition and emotion recognition will be significantly correlated with increasing social connectedness, increasing social competence, increasing quality of life, and decreasing severity of general psychopathology symptoms.
 - b. Increasing social connectedness will be significantly correlated with increasing social competence, increasing quality of life, and decreasing severity of general psychopathology symptoms.
4. I predict social connectedness will be a significant mediator of the relationship between schizotypy and the outcome variables when controlling for neurocognition and emotion recognition.
 - a. Social connectedness will significantly mediate the relationship between schizotypy and social competence.
 - b. Social connectedness will significantly mediate the relationship between schizotypy and quality of life
 - c. Social connectedness will significantly mediate the relationship between schizotypy and general psychopathology symptoms.
5. Within the schizotypy group, I predict there will be a relationship between schizotypy traits and social connectedness.
 - a. I will conduct an exploratory investigation of the correlations between positive, negative, and disorganized schizotypy traits and social connectedness. No specific associations are hypothesized.

Method

Participants

An online questionnaire was sent via email to 10257 freshman and sophomore undergraduates at Louisiana State University as part of a larger study to which 2303 (22.5%) students responded. Most respondents were female (61.4%) and Caucasian (77.0%). Those who completed the questionnaire were entered into a lottery of ten possible \$25 prizes. The questionnaire consisted of a consent form, demographic questions, and the Schizotypal Personality Questionnaire-Brief Revised (SPQ-BR; Cohen, Matthews, Najolia, & Brown, 2010, described further below). Profile validity was checked with the Infrequency Scale (Chapman & Chapman, 1976). This is a thirteen-item validity measure consisting of infrequently endorsed items (e.g. I believe that most light bulbs are powered by electricity, or I find that I often walk with a limp, which is the result of a skydiving accident,) answered in a dichotomous true/false format. The items were embedded in the larger questionnaire in order to identify random or dishonest responders. Participants endorsing more than three items in the unexpected direction were excluded ($n = 23$) resulting in 2277 valid profiles. When invalid profiles and those with missing items were removed resulting in 1816 profiles, the mean SPQ-BR total score was 50.01 ($SD = 20.32$). The distribution of SPQ-BR scores in the larger sample of respondents approximated a normal distribution (skew = 0.06, kurtosis = -0.28). Total SPQ-BR scores were similar for males ($M = 42.97$, $SD = 25.95$) and females ($M = 41.73$, $SD = 25.37$), $t(1748) = 1.12$, $p = 0.26$). SPQ-BR negative factor scores, however, were significantly higher for males ($M = 7.09$, $SD = 5.66$) than females ($M = 6.17$, $SD = 5.20$), $t(1692.02) = 3.89$, $p < 0.00$). There were no sex differences for the positive or disorganization factors. Age was not significantly related to SPQ-BR scores when evaluated with Pearson's correlations (all p 's > 0.10). Internal

reliability for the SPQ-BR in this sample was also good ($\alpha = 0.93$). The factors were highly correlated. All factors were positively correlated with total scores at $r = 0.73$ or greater. Positive scores were most highly correlated with disorganization scores ($r = 0.63$), and negative scores were most highly correlated with positive scores ($r = 0.50$). All factors were positively correlated with each other at $r = 0.45$ or greater.

Participants for the schizotypy group were identified by positive, negative, or disorganized scale scores in the 95th percentile (based on gender and ethnicity norms of the larger sample). To address concerns that depressive symptoms can give “false positives” on negative schizotypy subscales, individuals scoring high on the negative factor were considered for the schizotypy group if they: 1) also showed elevation on the positive or disorganization factors, or 2) had a depression subscale score from the Brief Symptom Inventory (BSI; Derogatis & Melisaratos, 1983, further described below) below gender and ethnicity determined means. Two hundred and six individuals were approached for participation in the schizotypy group, 40 (19%) agreed to participate, and 39 completed the measures included in this study. The final schizotypy group was similar to the group approached for participation. The larger group was 60% female and 80% Caucasian with a mean age of 19.18 ($SD = 2.90$) compared to 59% female and 77% Caucasian with a mean age of 18.64 ($SD = 1.22$) in the final schizotypy sample.

A control group was also recruited based on SPQ-BR factor scores below gender and ethnicity means. Four hundred eighty five individuals were approached for participation in the control group, 43 agreed to participate (9%), and 41 completed the measures included in this study. The larger group was 63% female and 81% Caucasian with a mean age of 19.10 ($SD = 3.00$) compared to 61% female and 81% Caucasian with a mean age of 19.02 ($SD = 2.27$) in the final control sample.

All participants in the laboratory phase of the study received \$20 cash compensation and the possibility of extra credit toward psychology courses. Participants were tested by trained undergraduate research assistants using the instruments noted below along with a variety of other instruments as part of the larger study. Testing sessions lasted approximately two hours. This study was approved by the Louisiana State University Human Subject Review Board, and all participants offered written informed consent prior to completing the surveys.

Measures

Schizotypy traits: Schizotypal Personality Questionnaire Brief-Revised (SPQ-BR).

In order to select participants and measure symptomology, the SPQ-BR (Cohen et al., 2010) was used. The original SPQ is a 74-item, self-report questionnaire that assesses the full range of schizotypal personality disorder symptomatology (DSM IV-TR; Raine, 1991). The SPQ is easy to administer and yields a large amount of data. It has been used in a number of studies and is preferred over other similar instruments because it has superior psychometric properties (Raine, 1991), assesses a broad range of symptoms that are closely related to DSM IV-TR symptoms, and is relatively brief. This study employed the most recent brief version, created based on a factor analysis of the original SPQ. This version improves on the original in that it is briefer (34 items), shows high reliability (internal consistency of factors range $\alpha = 0.75$ to 0.97), and possesses evidence for validity based on factor analytic procedures and relationship to measures of life quality. The scale is composed of seven subscales reflecting the original SPQ subscales which mirror symptoms of schizotypal personality disorder: ideas of reference/suspiciousness, no close friends/constricted affect, eccentric behavior, social anxiety, magical thinking, odd speech, and unusual perceptions. Based on prior studies of schizotypy and factor analytic results, three factors reflecting positive or cognitive-perceptual traits (ideas of

reference/suspiciousness, magical thinking, and unusual perceptions subscales), negative or interpersonal traits (no close friends/constricted affect and social anxiety subscales), and disorganized traits (eccentric behavior and odd speech subscales) were employed to identify potential participants and to quantify schizotypal traits. Responses are made on a five-point Likert scale ranging from 1 (strongly disagree) to 5 (strongly agree). Increasing scores reflect increasing amounts of the particular trait. Items, factors, and scales are listed in Appendix A.

General psychopathology symptoms: Brief Symptom Inventory (BSI). The BSI (Derogatis & Melisaratos, 1983) is a 53-item self-report inventory of a broad range of psychological symptoms including depression, anxiety, physical complaints, and psychotic symptoms. The scale has been used in numerous studies to date (a PsychINFO search revealed 922 peer-reviewed empirical studies in English). Research has demonstrated adequate reliability and evidence of construct validity in terms of relationship to other scales such as the Symptom Check List-90 and Minnesota Multiphasic Inventory, and the internal structure has been supported by factor analysis (Derogatis & Melisaratos, 1983). Participants rate symptoms on a five point Likert scale ranging from (0 = not at all, 1 = a little bit, 2 = moderately, 3 = quite a bit, and 4 = extremely). For the purpose of this study, two separate scores will be used. The Depression subscale was employed as mentioned above to recruit participants, and the Global Severity subscale was used as a measure of general symptom severity. The Global Severity subscale reflects the average intensity rating for all of the items. Increasing scores reflect increasing levels of symptoms. This measure was chosen based on its ease of administration, substantial research support, and the broad range of symptoms measured.

Social connectedness: Social Connectedness Scale-Revised (SCS-R). The SCS-R (Lee & Robbins, 1995) is a twenty-item self-report scale with a dichotomous true/false response format. Items are designed to assess an individual's subjective sense of connectedness or disconnectedness from the social world. As discussed above, the scale was developed and evaluated on a large group of undergraduate students as part of a larger effort to measure the theoretical construct of belongingness. The scale has demonstrated adequate reliability (internal reliability $\alpha = 0.91$, test-retest $r = 0.96$; Lee & Robbins, 1995). Original items were theoretically derived based on Kohut's (1984) definition of connectedness and evaluated for content validity by a panel of judges. Items not cohering based on the results of a principal components analysis were eliminated along with those significantly associated with social desirability. The scale has been evaluated in follow-up studies, with good support for convergent and divergent validity (Lee, Draper & Lee, 2001; Lee & Robbins, 1998; 2000). Some items are reverse scored, and increasing scores reflect higher connectedness. Items and scoring are listed in Appendix B.

Neurocognition: Brief Assessment of Cognition in Schizophrenia (BACS).

Neurocognition was assessed with the BACS (Keefe, Goldberg, Harvey, Gold, Poe, & Coughenour, 2004). The BACS is a measure designed specifically for assessing those deficits that research has typically found to be related to functional outcome in individuals with schizophrenia including verbal memory (List Learning), working memory (Digit Sequencing), motor speed (Token Motor Task), attention (Symbol Coding), executive functions (Tower of London), and verbal fluency (Category Instances and Controlled Oral Word Association Test). A description of the different scales from the test manual is contained in Appendix C. It takes about 35 minutes to complete and yields seven subtest scores as well as a composite score. The measure is highly reliable (test-retest ICC's range from 0.86 to 0.95), shows convergence with

other measures of the same constructs, and demonstrates comparable sensitivity to deficits (Keefe et al., 2004). Total scores are employed in this study.

Emotion recognition: Penn Emotion Recognition Test (PERT). To assess social cognitive abilities, a measure of emotion recognition was used. This social cognitive domain has been examined more frequently than the other domains schizophrenia and schizotypy samples, and prior research supports it as a valid and reliable measurement construct (Gur et al., 2002; Kohler et al., 2003). Moreover, studies within schizotypy suggest this may be an area in which deficits are particularly likely to be captured with available measures (Kee et al., 2004; Brown & Cohen, 2010). The 40-item PERT (Gur et al., 2002; Kohler et al., 2003) was employed here. This measure is brief but adequately sensitive for assessing less pronounced deficits in emotion recognition. It includes items of both high and low intensity and depicts angry, fearful, happy, sad, and neutral faces. The faces represent a diversity of ethnicity and age and include both posed and evoked expressions. Each face is presented one at a time, and participants are asked to choose which emotion is being expressed from a list of six choices (happy, sad, disgust, fear, anger, no emotion) reflecting five of the six universal emotions according to Ekman and Friesen (1975). The PERT authors did not include one universal emotion, surprise, in the list because they claimed it is not a “pure” emotion, saying “its valence depends entirely on the triggering event and it can be any of the other emotions, with a rapid onset” (Kohler et al., 2003). Scores reflect the percent of expressions identified correctly. Examples of stimuli are available in Appendix D.

Social competence: UCSD Performance-Based Skills Assessment-2 (UPSA-2). The UPSA-2 is the brief version of UCSD performance-based social functional skills assessment and assesses an individual’s functional capacity in five domains of daily living: 1) financial skills 2)

communication 3) organization/planning 4) transportation 5) household management. More information on these subscales is contained in Appendix F. The test is a performance-based measure of social functional skills and assesses an individual's ability to perform these tasks rather than actual social functioning outside of the testing environment. The tasks employed, however, reflect typical tasks employed in real life situations. The measure takes approximately 30 minutes to administer and has been shown to have high test-retest reliability and participant tolerability above and beyond other measures (Harvey, Green, & Nuechterlein, 2010). In each subsection participants role play tasks that are involved in activities of daily living such as making a doctor's appointment, reading a utility bill, planning a day trip, and cooking a dessert. This measure has frequently been used for identifying deficits in individuals with schizophrenia-spectrum disorders and was designed to assess deficits specific to the schizophrenia spectrum (Mausbach et al., 2008; Mausbach et al., 2011; Mausbach et al., 2010; Patterson, Goldman, McKibbin, Hughs, & Jeste, 2001). This scale was chosen because it is a relatively brief and ecologically valid measure of functional deficits expected in the schizophrenia spectrum with substantial research support.

Quality of life: Lehman's Quality of Life Brief Interview (QoL-I). Quality of life was assessed with the QoL-I, a self-report questionnaire that includes items that assess an individual's subjective perception of his or her quality of life as well as objective items assessing activities and social supports (Lehman, 1995). This measure has previously been used in research involving psychiatric populations (Anderson, McNeil, & Reddon, 2002; Wasserman, Sorensen, Delucchi, Masson, & Hall, 2006; Heider et al., 2007) and has demonstrated good psychometric properties (Lehman, 1996). The brief version includes 78 items, and the amount of administration time was not feasible for this study. The current study employs the even briefer

version used by Bellack, Bennett, Gearon, Brown, and Yang (2006) and Cohen and Davis (2009) which includes 33 items, allowing for computation of seven scales: home concerns, daily activities, family relationships, social relationships, financial concerns, legal concerns, health concerns, and global life quality in two domains: objective quality of life and subjective quality of life. This scale is especially useful because it is easy to administer to large numbers of individuals and yields a wealth of information. Additionally, subjective and objective scales allow for separate assessment of the individual's subjective appraisal (How satisfied are you with . . . ?) and objective behaviors (How often do you . . . ?). Twenty three items comprise the objective scale (internal consistency estimates for the different domains range from $\alpha = 0.48$ to 0.67), and nine items rated on a seven point Likert scale comprise the subjective scale ($\alpha = 0.84$) (Cohen & Davis, 2009). Though measuring "objective" quality of life with a self-report instrument may cause some concern, Cohen and Davis (2009) demonstrated that objective and subjective quality of life have different correlates within schizotypy and seem to represent separate constructs. Items and response options are listed in Appendix E. Increasing scores reflect increasing quality of life.

Internal reliability for those measures expected to be internally reliable was generally good for this sample. The SPQ-BR was highly reliable (Cronbach's $\alpha = 0.96$), and the subjective quality of life scale on the QoL-I ($\alpha = 0.80$) and PERT ($\alpha = 0.62$) were somewhat less reliable. For the objective quality of life scale of the QoL-I, internal reliability for the different domains was acceptable and comparable to that found by Cohen and Davis (2010): family $\alpha = .50$, social/friends $\alpha = .74$, financial $\alpha = .55$, safety/home $\alpha = .51$. Internal reliability for this sample is not reported for standard clinical instruments as these have established psychometric support and multiple scales assessing different constructs (i.e. BSI, UPSA-2, and BACS).

Analyses

Analyses were conducted in several steps. First, the data were checked for normality and corrected as needed. Next, the groups were compared on demographic and other relevant variables. Third, the main variable of interest, social connectedness, was examined to determine whether schizotypy group membership was associated with social connectedness. This was done with a hierarchical regression examining the relationship between schizotypy and social connectedness. A regression model was chosen to evaluate the relationship between these variables because these reflect measured rather than experimentally manipulated variables. Regressions have traditionally been used to address questions of relationship or correlation between variables rather than questions of cause and effect, although regression can be used for both types of questions (Field, 2005). The mathematically equivalent ANOVA or ANCOVA procedures are conceptually suited for experimentally-manipulated variables (Field, 2005). First, social connectedness was entered in step one of the model to predict schizotypy group membership. In the next step, neurocognition and emotion recognition were entered into step two as covariates to determine whether the relationship between social connectedness and schizotypy group membership would still be significant when controlling for these variables. After group comparisons were examined, correlations between included variables were examined. Next, the hypothesized mediation models were evaluated. Lastly, exploratory analyses were conducted examining the correlations between social connectedness and schizotypy traits within the schizotypy group.

To examine whether social connectedness mediated the relationship between schizotypy and the various outcomes, Preacher and Hayes's bootstrapping (Preacher & Hayes, 2004) method of examining indirect effects was employed. This method is considered more powerful

than the traditional Baron and Kenny three-step approach (Baron & Kenny, 1986) and the Sobel Test (Hoyt, Imel, & Chan, 2008; Mackinnon, Lockwood, Hoffman, West, & Sheets, 2002; Sobel, 1986). Bootstrapping is a nonparametric method that uses resampling with replacement. A large number of samples are generated, and the indirect effect is computed from each sample. Next, a sampling distribution of the indirect effects is created. From this distribution, a confidence interval for the indirect effect based on the distribution of indirect effects is created. The confidence interval is checked to determine whether it contains zero. If it does not, the researcher can conclude that the population value of the indirect effect is not likely to be zero, or that there is an indirect effect. For these analyses, the recommended 1000 repetitions of the bootstrap process were performed (Preacher & Hayes, 2004). The authors have written a specific macro available for use with the latest edition of Predictive Analytics Software (PAWS v.18; formerly SPSS) available for download from the author's website (<http://www.afhayes.com/introduction-to-mediation-moderation-and-conditional-process-analysis.html>). This method is preferred over other methods of mediation analysis because it does not focus on the significance of the direct relationship between the predictor and outcome variable (the $x \rightarrow y$ path) before or after the inclusion of the mediation. Focus on the direct path may unnecessarily restrict the analysis because indirect effects can be revealed in the absence of a direct relationship between the predictor and outcome (Hayes, 2009; MacKinnon et al., 2002). There may also be differential power for detecting total and direct effects as opposed to indirect effects and suppression effects (or opposing indirect effects) may obscure total effects in a model (Preacher & Kelley, 2011; Rucker, Preacher, Tormala, & Petty, 2011). Finally, the notion of "full" and "partial" mediation is not very informative. Full mediation seems to suggest that a single mediator fully accounts for the $x \rightarrow y$ relationship. Preacher and Kelly (2011)

demonstrated with simulated and empirical data that this is not necessarily the case. Even with a full mediation model, other mediators may be operating. Instead, mediation effects are better communicated by quantifying the effect sizes of indirect effects. The variables included in the mediational analyses were measured rather than manipulated, and, therefore, causality cannot definitively be established (Mathieu & Taylor, 2006). The temporal precedence of the predictor and mediator has not been established, which means the included variables may be related via a different pattern. In order to provide further support for and evaluate the specificity of the significant mediation models, therefore, alternative models were tested. This was done by reversing the order of the variables. These alternative models were then evaluated for evidence of significant mediation.

Power analysis was conducted for the analyses planned in this study. These were computed with statistical software G*power 3.1.3. When examining group differences, to detect medium effects with a power of 0.80 and alpha level of 0.05 with a one-tailed test, a total of 51 participants per group or twenty one per group for large effects would be required. For the one-tailed correlational analyses within the schizotypy group with the same parameters, 64 participants in the schizotypy group would be needed to detect medium effects or twenty one to detect large effects. Medium to large effects are expected for these analyses based on prior research showing these types of effects on social/functional variables such as quality of life and general psychopathology symptoms. For the regression analysis, a total sample size of 77 is required to detect medium effects. The total sample size is within these ranges ($n = 80$).

Power to detect the mediated effect is a huge concern in mediation studies. Fritz and Mackinnon (2007) surveyed the literature employing mediation tests and reported that across 189 independent samples, the median sample size employed was $n = 187$ (range from 20 to 16,466).

Most studies used the Baron and Kenny method of determining mediation (70.9%), despite the low power of this method. These authors also analyzed the required sample sizes to detect mediated effects for the different analytic methods used in the literature. They estimated the required sample sizes needed to detect mediation with a power of $\beta = 0.80$. Using the Baron and Kenny (1986) approach, this study would not be powered to detect full mediation even with large size parameters specified in the model (the $X \rightarrow M$ path and the $M \rightarrow Y$ path adjusted for X). The study could, however, detect partial mediation with medium to large parameters specified. Similarly, using the Sobel (1986) method, the study would be powered to detect mediation with medium and large parameters specified. Employing the bootstrap method to test indirect effects, this study equipped to detect mediation with a power of $\beta = 0.80$ when the parameters are medium in size (13% of the variance) or larger. Therefore, provided that the relationship between schizotypy and social connectedness and the relationship between social connectedness and the outcome adjusting for schizotypy are at least medium in size, this study is adequately powered to detect mediation.

Results

Data were checked for normality by examination of histograms and scatterplots, skewness and kurtosis, and statistical tests of normality (Kolmogorov-Smirnov tests). Non-normal data (PERT and BSI) were transformed with log transformations with the exception of objective quality of life. This variable could not be transformed to meet the assumption of normality. Upon examination of the data, this variable was judged to approximate normality closely enough to proceed with the analyses (skew = -0.86; kurtosis = 1.53). Means and standard deviations are reported for non-transformed data in Table 1.

The sample was mostly female (60%) and Caucasian (78.8%) with a mean age of 18.84 ($SD = 1.84$). The control group was relatively healthy as reflected by BSI scores (BSI GSI $M = 1.5$, $SD = 0.37$ with a total possible range of 0 to 5; BSI Depression $M = 7.42$, $SD = 1.59$ with a total possible range of 0 to 30). Most participants completed all the measures administered in this study. Any measures with missing participants are noted in Table 1.

Group Comparisons

Groups were similar with respect to demographic variables. The groups did not differ in age (Mann Whitney $U = 744.5$, $p = 0.53$, control age $M = 19.02$, $SD = 2.27$ schizotypy age $M = 18.64$, $SD = 1.22$). Both groups were composed primarily of Caucasian females (control group 81% Caucasian, 61% female and schizotypy group 77% Caucasian, 59% female). The control and schizotypy groups were also compared on all variables included in this study. These comparisons are depicted in Table 1. The schizotypy group scored significantly higher on all self-report symptom measures than the control group. The schizotypy group also reported significantly lower objective and subjective quality of life. Scores were not significantly different between groups on neurocognition, emotion recognition, and social competence.

Table 1
Group comparisons with means (M) and standard deviations (SD).

	Control	Schizotypy	<i>t</i> (df)	<i>p</i>	<i>d</i>
	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)			
SPQ-BR Total	26.51 (11.85)	78.79 (14.37)	-17.79 (78)	< 0.001	-3.97
SPQ-BR Positive	8.46 (5.03)	30.87 (9.28)	-13.33 (57.91)	< 0.001	-3.00
SPQ-B Disorganization	8.34 (4.70)	25.64 (4.87)	-16.17 (78)	< 0.001	-3.61
SPQ-BR Negative	3.27 (2.42)	11.92 (6.12)	-8.24 (49.09)	< 0.001	-1.86
Neurocognition ¹	281.05 (31.37)	290.26 (26.61)	-1.41(77)	0.17	-0.32
Emotion Recognition ²	0.78 (0.08)	0.76 (0.08)	1.25 (78)	0.22	0.25
QoL Objective	-1.15 (4.42)	-4.51 (7.20)	2.51 (62.54)	0.02	0.56
QoL Subjective	38.79 (3.82)	33.33 (4.93)	5.56 (78)	< 0.001	1.24
Social Competence ³	44.69 (3.93)	44.38 (3.53)	0.36 (76)	0.72	0.08
BSI Symptoms ²	1.46 (0.36)	2.82 (0.87)	-9.59 (64.66)	< 0.001	-2.04

Note. *d* = Cohen's *d* effect size. SPQ-BR = Schizotypal Personality Questionnaire Brief-Revised. QoL = quality of life. ¹ data were missing for one control group member for this measure, so all analyses employing this measure contain *n* = 40 in the control group. ²This variable was log transformed. Means and standard deviations are reported for the original scale. ³ Data were missing for two control group members for this measure, so all analyses employing this measure contain *n* = 39 in the control group

Social Connectedness

Social connectedness was measured with the SCS-R, a self-report measure with a Likert scale response format. Reliability of the measure in this sample was high (Cronbach's $\alpha = 0.96$) and similar to that reported by Lee and colleagues ($\alpha = 0.92$). The overall mean score of the sample ($M = 85.95$, $SD = 21.08$) was also similar to that reported by Lee and colleagues (2001; $M = 88.02$). Examining group differences, the schizotypy group reported being much less social

connected ($M = 71.72$, $SD = 17.72$) than controls ($M = 99.49$, $SD = 13.92$) at a large effect size ($d = 1.74$). It is notable that the control group here scored higher than Lee and colleagues' participants ($M = 99.49$), which were also recruited from a university setting. At least one other study examining social connectedness in college students also reported scores similar to those in this sample and higher than Lee and colleagues' participants' mean score ($M = 93.07$, $SD = 15.04$; Armstrong & Oomen-Early, 2009).

To further examine social connectedness, a hierarchical linear regression was performed using group membership (schizotypy vs. control) to predict social connectedness. As neurocognition and emotion recognition were identified as theoretically important covariates in the hypotheses, these variables were entered in step 2 of the model to examine whether the relationship between social connectedness and schizotypy would change. The first model was significant and explained a large amount of the variance ($R^2 = 0.43$, $F(1, 77) = 59.98$, $p < 0.001$). The second model with the covariates included was also significant ($R^2 = .42$, $F(3, 75) = 19.95$, $p < 0.001$). The change in R^2 was not significant ($\Delta R^2 = 0.02$, $p = 0.67$). Schizotypy significantly predicted social connectedness, and controlling for neurocognition and emotion recognition did not affect this relationship. Given that neurocognition and emotion recognition were not significant predictors of schizotypy and that the control and schizotypy groups did not differ on these variables, the subsequent analyses reported here do not include covariates. It should be noted, however, that when the analyses including these covariates were performed, results did not change significantly. Results are depicted in Table 2.

Table 2
Regression models using group (schizotypy vs. control) to predict social connectedness.

Variable	<i>b</i>	SE <i>b</i>	β	Adjusted R^2	ΔR^2
Step 1					
Group	-27.86	3.60	-0.66	0.43	
Step 2					
Group	-28.30	3.71	-0.67		
Neurocognition	0	0.06	-0.01		
Emotion Recognition	-20.95	23.56	-0.08	0.42	0.02

Correlations

The next set of analyses focused on the predicted relationships between social connectedness and the outcome variables. First, the relationships between the hypothesized covariates, neurocognition and emotion recognition, and the other variables of interest were examined. Pearson's correlations are provided in Table 3. The only significant relationship was between social competence and neurocognition. Next, correlations between all the variables of interest for the mediational analyses were examined (see Table 4). Social connectedness was significantly positively correlated with objective and subjective quality of life and significantly negatively correlated with BSI symptoms. Social connectedness was not significantly related to social competence.

Table 3
Pearson's correlations between hypothesized covariates and variables of interest.

	Neurocognition	Emotion Recognition
Social Connectedness	-0.11	0.01
Qol Objective	0.11	-0.15
Qol Subjective	-0.01	0.04
Social Competence	0.24*	0.03
BSI Symptoms	0.12	0.02

Note. Qol = quality of life
* $p < 0.05$

Table 4
Pearson's correlations between variables used in mediational analyses.

	Social Connectedness	Qol Objective	Qol Subjective	Social Competence
Qol Objective	0.49**	--		
Qol Subjective	0.61**	0.40**	--	
Social Competence	0.15	0.13	-0.03	--
BSI Symptoms	-0.57**	-0.15	-0.43**	0.05

Note. Qol = quality of life
** $p < 0.01$

Mediational Analyses

Next, mediational hypotheses were examined. Distributions of residuals were examined for homoscedasticity—whether the variance within the groups was similar for each of the variables. This was done by visually examining scatterplots of the residuals for each of the variables. There were some concerns about the distributions of residuals for the BSI symptoms and objective quality of life. The bootstrapping method of analyzing indirect effects, unlike the three-step approach (Baron & Kenny, 1985) or Sobel (1986) test, however, is robust to non-normal distributions (Preacher & Kelly, 2011; Rucker et al., 2011). In addition, given that the prior analyses showed no difference between the groups and no change in correlations between the included variables when covariates were included, mediational analyses including the hypothesized covariates (neurocognition and emotion recognition) are not included here. When these analyses were performed, however, there was no meaningful change. Additionally, further statistics quantifying the size of the indirect effects were examined. Preacher and Kelley (2011) recommend reporting effect sizes of indirect effects in mediation models as κ^2 which can be understood as “the proportion of the maximum possible indirect effect that could have occurred, had the constituent effects been as large as the design and data permitted” (0 = no indirect effect and 1 = as large as possible). The authors do not provide guidelines for interpretation of ranges of effects, but they state that these effects may be interpreted according to Cohen’s (1988) guidelines (0.02 = *small*, 0.15 = *medium*, 0.35 = *large*).

The model of the total effect of group on social competence was not significant, $F(1, 76) = 0.13, p = 0.72, R^2 = 0$. When social connectedness was added to the model, it was still not significant, $F(2, 75) = 1.13, p = 0.32, \Delta R^2 = 0.03$. The total effect without the mediator was -0.31 ($SE = 0.85$), $p = 0.72$, 95% CI [-0.20, 1.38]. The direct effect while controlling for the

mediator was 0.81 ($SE = 1.13$), $p = 0.48$, 95% CI [-1.45, 3.07]. Analysis of the indirect effect using the bootstrap method showed no evidence of mediation (bootstrap mean = -1.21, 95% CI [-3.25, 0.60]). Group did not predict social competence, and there was no evidence for social connectedness as a mediator. B 's and indirect effects for all mediation analyses are reported in Table 5.

The model of the total effect of group on objective quality of life was significant, $F(1, 78) = 6.42$, $p = 0.01$, $R^2 = 0.08$. When social connectedness was added to the model, the model was still significant, $F(2, 77) = 12.26$, $p < 0.001$, $\Delta R^2 = 0.24$. The total effect without the mediator was -3.37 ($SE = 1.33$), $p = .01$, 95% CI [-6.01, -0.72]. The direct effect while controlling for the mediator was 1.03 ($SE = 1.62$), $p = 0.53$, 95% CI [-2.19, 4.25]. The effect of group decreased with the mediator added. There was a significant indirect effect (bootstrap mean = -4.40, 95% CI [-7.20, -2.03]). The κ^2 effect size was 0.29 ($SE = 0.07$, 95% CI [0.14, 0.43]). Social connectedness significantly mediated the relationship between schizotypy and objective quality of life, at a medium effect size.

The model of the total effect of group subjective quality of life was significant, $F(1, 78) = 30.87$, $p < 0.001$, $R^2 = 0.28$. When social connectedness was added to the model, the model was still significant, $F(2, 77) = 26.23$, $p < 0.001$, $\Delta R^2 = 0.13$, 95% CI [-7.45, -3.47]. The total effect without the mediator was -5.46 ($SE = 0.98$), $p < 0.001$, 95% CI [-7.42, -3.50]. The direct effect while controlling for the mediator was -2.30 ($SE = 1.20$), $p = 0.06$, 95% CI [-4.69, -0.10]. The effect of group decreased with the mediator added. There was a significant indirect effect (bootstrap mean = -3.16, 95% CI [-5.36, -1.59]) using the bootstrap method. The κ^2 effect size was 0.27 ($SE = 0.07$, 95% CI [0.15, 0.42]). Social connectedness significantly mediated the relationship between schizotypy and subjective quality of life at a medium effect size.

The model of the total effect of group on BSI symptoms was significant, $F(1, 71) = 91.12, p < 0.001, R^2 = 0.56$. When social connectedness was added, the model was still significant, $F(2, 70) = 46.42, p < 0.001$ with almost no change in the amount of variance explained ($\Delta R^2 = 0.01$). The total effect without the mediator was 0.21 ($SE = .03$), $p < 0.001$, 95% CI [0.25, 0.33]. The direct effect while controlling for the mediator was 0.27 ($SE = 0.04$), $p < 0.001$, 95% CI [0.17, 0.32]. Analysis of the indirect effect showed no evidence of mediation (bootstrap mean = 0.03, 95% CI [-0.03, 0.08]). Social connectedness did not significantly mediate the relationship between schizotypy and BSI symptoms.

Table 5
Regression models examining the effect of group and social connectedness outcome variables.

	Regression			Bootstrap			
	B	SE B	R^2	ΔR^2	Indirect Effect	SE	95% CI
<hr/>							
Social Competence							
<hr/>							
Step 1:			0				
Group	-0.31	0.85					
Step 2:			0.03	0.02			
Group	0.81	1.33					
Social Connectedness	0.04	0.03			-1.12	0.93	-3.12, 0.49

Table 5 continued

Regression models examining the effect of group and social connectedness outcome variables.

	Regression			Bootstrap			
	B	SE B	R ²	Δ R ²	Indirect Effect	SE	95% CI
Objective QoL							
Step 1:			.08				
Group	-3.37	0.93					
Step 2:			0.24	0.16			
Group	1.02	1.35					
Social Connectedness	0.16	0.04			-4.40	1.30	-7.19, -2.03
Subjective QoL							
Step 1:			0.28				
Group	-5.46	0.98					
Step 2:			0.41	0.13			
Group	-2.30	1.20					
Social Connectedness	0.11	0.03			-3.16	0.92	-5.36, -1.59

Table 5 continued

Regression models examining the effect of group and social connectedness outcome variables.

	Regression		Bootstrap				
	B	SE B	B	SE B	B	SE B	B
Symptoms							
Step 1:			0.56				
Group	0.27	0.03					
Step 2:			0.57	0.01			
Group	0.24	0.04					
Social Connectedness	0	0			0.03	0.03	-0.03, 0.08

Note. SE = standard error. CI = Confidence Interval

As evidence for mediation was found with the quality of life outcome variables, these variables were examined further. Proper model specification is a major concern for mediational analyses. Given that the variables included were measured rather than experimentally manipulated, it is impossible to establish a chain of causality (Mathieu & Taylor, 2006). It is possible that the data might be better explained by a different relational pattern between the predictor, mediator, and outcome. To further test the validity of the hypothesized models, therefore, alternative models were tested. For the quality of life outcome variables, two other models were tested. First, a model in which quality of life mediated the relationship between schizotypy and social connectedness was tested for each domain of quality of life (the mediator and outcome were switched). Next, another model was tested in which schizotypy mediated the

relationship between social connectedness and quality of life (the predictor and the mediator were switched). Group could not be used as a mediator because it is a dichotomous variable. SPQ-BR total score was employed instead as a continuous measure of schizotypy.

The model of the total effect of schizotypy on social connectedness was significant, $F(1, 78) = 61.08, p < 0.001, R^2 = 0.44$. When objective quality of life was added to the model, the model was still significant, $F(2, 77) = 45.13, p < 0.001, \Delta R^2 = 0.10$. The total effect without the mediator was $-27.78 (SE = 3.55), p < 0.001, 95\% CI [-6.01, -0.72]$. The direct effect while controlling for the mediator was $-23.96 (SE = 3.37), p < 0.001, 95\% CI [-30.67, -17.25]$. There was no evidence for an indirect effect (bootstrap mean = $-3.81, SE = 1.98, 95\% CI [-8.79, -0.85]$). Objective quality of life did not mediate the relationship between schizotypy social connectedness.

The model of the total effect of social connectedness on objective quality of life was significant, $F(1, 78) = 24.30, p = 0.01, R^2 = 0.24$. When schizotypy was added to the model, the model was still significant, $F(2, 77) = 11.99, p < 0.001, \Delta R^2 = 0$. The total effect without the mediator was $0.14 (SE = 0.03), p = 0.01, 95\% CI [-6.01, -0.72]$. The direct effect while controlling for the mediator was $0.14 (SE = 0.04), p < 0.001, 95\% CI [-2.19, 4.25]$. There was no evidence for an indirect effect using the bootstrap method (bootstrap mean = $0, SE = 0.03, 95\% CI [-0.05, 0.05]$). Schizotypy did not mediate the relationship between social connectedness and objective quality of life.

The model of the total effect of schizotypy on social connectedness was significant, $F(1, 78) = 61.08, p < 0.001, R^2 = 0.44$. When subjective quality of life was added to the model, the model was still significant, $F(2, 77) = 45.13, p < 0.001, \Delta R^2 = 0.09$. The total effect without the mediator was $-27.78 (SE = 3.55), p < 0.001, 95\% CI [-6.01, -0.72]$. The direct effect while

controlling for the mediator was -19.63 ($SE = 3.85$), $p < 0.001$, 95% CI $[-27.30, -11.97]$. There was no evidence for an indirect effect (bootstrap mean = -8.14 , $SE = 2.78$, 95% CI $[-15.30, -3.67]$). Subjective quality of life did not mediate the relationship between schizotypy social connectedness.

The model of the total effect of social connectedness subjective quality of life was significant, $F(1, 78) = 47.22$, $p < 0.001$, $R^2 = 0.38$. When schizotypy was added to the model, the model was still significant, $F(2, 77) = 23.57$, $p < 0.001$, $\Delta R^2 = 0$, $p = 0$, 95% CI $[-7.45, -3.47]$. The total effect without the mediator was 0.15 ($SE = 0.02$), $p < 0.001$, 95% CI $[0.11, 0.19]$. The direct effect while controlling for the mediator was 0.14 ($SE = 0.03$), $p < 0.001$, 95% CI = $0.17, 0.32]$. There was no evidence of an indirect effect using the bootstrap method (bootstrap mean = 0.01 , $SE = 0.02$, 95% CI = $[-0.03, 0.05]$). Schizotypy did not mediate the relationship between social connectedness and subjective quality of life.

The alternative mediation models including objective and subjective quality of life were not significant. These analyses, therefore, provide additional support for the hypothesis that social connectedness is a mediator in the relationship between schizotypy and quality of life. Although a chain of causality cannot be established, this supports the hypothesized specification of the models in which social connectedness is a mediator.

Exploratory Correlations

Finally, the last set of analyses concerned only the schizotypy group. To examine the relationship between schizotypy traits and social connectedness, correlations between scores on SPQ-BR factors and social connectedness were examined. These were computed with Pearson's correlations. The only factor scores significantly correlated with social connectedness

were the negative SPQ-BR scores. Higher negative schizotypy scores were associated with poorer social connectedness. These data are presented in Table 6.

Table 6
 Pearson's correlations between SPQ-BR variables and social connectedness with and without covariates (neurocognitive and emotion recognition performance)

	SPQ-BR	SPQ-BR	SPQ-BR	SPQ-BR
	Total	Positive	Negative	Disorganization
Social	-0.37*	-0.07	-0.41*	0
Connectedness				

Note. SPQ = Schizotypal Personality Questionnaire Brief-Revised

* $p < 0.05$

** $p < 0.01$

Discussion

This study was designed to examine whether individuals with schizotypy exhibit deficits in social connectedness—a variable that has not yet been examined in this population—and how these deficits relate to functioning. It was hypothesized that lower social connectedness may be a risk factor for schizophrenia that underlies deficits in other areas such as social cognition, social behavior, and social functioning. A finding of lower connectedness in a schizotypy sample would support this idea. A sample of 39 individuals with psychometrically-defined schizotypy and 41 controls were recruited from a university and compared on a number of variables including connectedness. Then specific models of the relationship between schizotypy, social connectedness, and various measures of functioning were examined.

Schizotypy and control groups were chosen based on scores from an online administration of the SPQ-BR (Cohen et al., 2010). The final samples were similar to those that initially responded to the online survey, and there is no evidence to suggest that those who participated in the laboratory portion of the study were different from those who did not participate. The entire sample was largely female and Caucasian and relatively healthy. The schizotypy group was composed of college students that were determined to be at risk for schizophrenia with the understanding that most will never develop a clinical disorder. As such, the results here may not generalize to clinical samples or even to samples recruited in community settings. These individuals represent the higher functioning individuals on the schizophrenia spectrum. Findings in this sample, therefore, are very unlikely to be tainted by the effects of clinical schizophrenia symptoms. In order for the results here to be extended to lower functioning samples and to schizophrenia as a whole, this study should be replicated in those groups.

The schizotypy and control groups were compared on a number of variables in this study. In terms of demographics, the groups were quite similar. The schizotypy group also reported much lower quality of life in both the objective and subjective domains and more severe symptoms on the BSI, as expected based on prior studies of emotional functioning in schizotypy (Miller & Lenzenweger, 2012; Najolia, Buckner, & Cohen, 2012; Rey, Jouvent, & Dubal, 2009; Seghers, McCleery, & Docherty, 2011). Not expected, however, was the finding of no difference between the groups on the performance measures—neurocognition, emotion recognition, and social competence. When examining the effect sizes, this does not appear to be explained by low power, with the exception of a possible small deficit in emotion recognition performance ($d = 0.25$). Though unexpected, other studies of neurocognition in schizotypy have found similar results (Jahshan & Sergi, 2007; Lenzenweger & Gold, 2000). This may be an area of deficit that present measures are not adequately sensitive to detect or an area that is not actually deficient when individuals are healthy. A recent study has also suggested neurocognitive impairment in schizotypy is more complicated (Chun, Minor, & Cohen, 2013). Specifically, though individuals with schizotypy do not show consistent impairment compared to controls, they do self-report being impaired. This may reflect the fact that neurocognitive measures are not sensitive enough to detect small impairments or that individuals may perceive difficulties before they are evident in the laboratory. They may be experiencing daily life problems as a result of cognitive deficiencies even though they are not affected when required to perform structured, time-limited test in a laboratory setting. Also possible is the fact that there are no impairments and their self-report reflects a negative view of the self or something akin to thought disorganization.

This sample was also not impaired on emotion recognition. This is less surprising because other studies have found that at-risk samples are not impaired relative to controls in emotion recognition (Jashan & Sergi, 2007; Li et al., 2010; Pinkham et al., 2007; Toomey & Schuldberg, 1995; Toomey et al., 1999). These results also suggest that social cognition deficits may not be an endophenotype for schizophrenia even though they may sometimes be present in at-risk individuals. If poor social cognition were the result of some underlying construct such as poor social connectedness that affected the development of normal social cognitive abilities, one might expect that studies examining social cognitive performance would find inconsistent results. Individuals may be able to perform some tasks in some settings but have more trouble with others. Relatedly, this schizotypy group also performed similarly to the control group on the social competence measure. This measure in particular required participants to perform structured tasks with clearly defined correct and incorrect answers. These tasks were typical tasks that a person would need to perform in a real world setting. Yet again, at least one other study has suggested that such abilities are sometimes intact in schizotypy laboratory studies (Thompson et al., 2012). Healthy individuals who are functioning at a level necessary to attend a major university would likely be able to perform these activities even if they had subtle social difficulties or abnormalities. This is further support for the idea that structured tasks requiring a correct or incorrect response may not be the best method for assessing social difficulties in the schizotypal population. Social deficiencies may be better evaluated with more sensitive measures or with more ambiguous requirements (Quirk, Submaranian, & Hoerger, 2007).

The major variable of interest in this study was social connectedness. The schizotypy group reported less social connectedness than the control group, and schizotypy group membership explained a large amount of the variance in social connectedness. This was not

something that could be accounted for by deficits in neurocognition and emotion recognition.

This is the first study to examine social connectedness in this population, and the data here offer support for social connectedness as a major deficit in schizotypy. The substantial effect size found here suggests that social connectedness may be more closely related to the central features underlying schizotypy and could reflect a risk factor for schizophrenia. Several aspects of this concept, including its logical connection to schizotypal and schizophrenic traits and its clear link to understanding of the social world in general, make it an especially plausible risk factor. Social connectedness deficits that give rise to difficulties in the natural or typical development of social cognitive abilities would explain why prior studies in risk populations have yielded inconsistent findings regarding social cognitive deficits. Poor connectedness may only negatively affect some domains of social cognition, it may affect these abilities to different degrees depending on the illness progression, and individuals may develop compensatory strategies to perform social cognitive tasks.

The most interesting part of this study examined the specific relationships between schizotypy, social connectedness, and four separate functional outcome measures—social competence, objective quality of life, subjective quality of life, and BSI symptoms. The hypothesized model was one in which social connectedness mediated the relationship between schizotypy and functioning. For social competence, there was no significant mediating relationship. This was not surprising given that the schizotypy group did not show a deficit in this area. As stated above, social competence is a performance measure. It is possible that these skills are not affected by connectedness deficits because individuals may have developed strategies necessary to function in the social world so that they are able to accomplish tasks. These individuals may still appear socially odd, require more effort to perform tasks, or have a

subjective sense of distress in the social world. This would not be evident in a laboratory measure of performance.

For the quality of life variables, the hypothesized mediation models were supported by the data. First, when the proposed models were tested, there was a significant total effect of schizotypy on quality of life, with a large amount of the variance in quality of life accounted for by schizotypy group membership. The direct effect of schizotypy on quality of life decreased when social connectedness was added to the model, and there was evidence of an indirect effect of moderate size. The validity of these models were further evaluated by comparing them to alternative models in which schizotypy was related to social connectedness through quality of life (switching the mediator and the outcome) and in which social connectedness was related to quality of life through schizotypy (switching the predictor and the mediator). These alternative models were not supported by the data, offering further evidence for the validity of the hypothesized models. Social connectedness, therefore, appears to mediate the relationship between schizotypy and quality of life. Individuals with schizotypy report lower life quality in terms of how much time they spend doing things with others or having leisure time and activities (objective) and report feeling less satisfied with this (subjective). This appears to occur largely through poor social connectedness.

For BSI symptoms, there was a direct relationship between schizotypy and BSI symptoms, however, adding social connectedness to the model did not change this relationship, and there was no mediation effect. This was an interesting finding. While schizotypy was associated with having more severe BSI symptoms, social connectedness did not play a role in this relationship. When considered in light of the fact that social connectedness did play a role in the relationship between schizotypy and life quality, it becomes even more meaningful. Social

connectedness is an important factor in functioning, but not symptoms. This may be because BSI symptoms are less socially based and do not have the same relationships to schizotypy traits as the quality of life variables. General psychopathology symptoms, instead, are directly related to schizotypy (Najolia et al., 2012; Seghers et al., 2011). In this study, poor connectedness, was at least somewhat uniquely associated with lower life quality. Based on these results, it seems that social connectedness deficits may underlie poorer quality of life, but not overall psychopathology symptoms.

Finally, the relationship between schizotypal traits and social connectedness was examined within the schizotypy group. Social connectedness was significantly negatively correlated only with negative schizotypy traits, and this was true even when controlling for neurocognitive and emotional recognition performance. This was a medium-size effect. Negative schizotypal traits seem to be the important aspect of schizotypy in relation to social connectedness. This is not surprising because negative traits are those that pertain most closely to interpersonal situations (no close friends, constricted affect, and social anxiety). In fact, there is much overlap in the way negative schizotypy and poor social connectedness are defined conceptually and measured in the scales included in this study (Cohen et al., 2010; Lee et al., 2001). Some of this association is probably due to conceptual overlap, and some is undoubtedly due to measurement redundancy. It is unclear why connectedness was not related to disorganized traits (odd speech and behavior); however, the data suggest that poorer connectedness is associated with more social withdrawal than oddness.

Overall, social connectedness appears to reflect an important aspect of schizotypy. This has been the first study to examine this concept in schizotypy. Other studies have explored the related concept of attachment (Berry, Band, Corcoran, Barrowclough, & Wearden, 2007;

Fernyhough, Hurndall, & Koronis, 2008; Meins, Jones, Tiliopoulos & Goodall, 2009), and many studies have explored social cognition (Gibson et al., 2010; Miller & Lenzeweger, 2002; Thompson et al., 2012) or traits such as social anhedonia in this group (Brown, Silvia, Myin-Germeys, Lewandowski, & Kwapil, 2008; Horan, Brown, & Blanchard, 2007; Kwapil, 1998). These concepts overlap somewhat with the idea of social connectedness, but they are not the same. Social connectedness is a broader construct than attachment extending beyond the primary caregiver, it reflects a stable individual difference that develops through social learning rather than a specific ability, and is not defined or conceptualized as a symptom-like trait. The high correlations between social connectedness and negative traits suggest some substantial overlap in how these concepts are defined. Indeed, inspection of the items on these scales suggests that they may really be measuring much of the same thing (e.g. “I feel close to people” vs. “Do you feel you cannot get close to people?”; Cohen et al., 2010; Lee et al., 2001). While much of the relationship may be explained by method invariance, this still raises questions about how schizotypy is conceptualized. Research has characterized this group as behaviorally odd with strange ideas and perceptions and generally apathetic toward others (Cohen, Najolia, Brown, & Minor, 2011; Meehl, 1990; Raine, 1994; Siever & Davis, 2004). A poor connection to the social world is something a bit different.

There are a number of limitations to this study. Though the sample used in this study was chosen based on theoretical considerations, it is limited in several ways. First, this study was not conducted on a clinical sample. Though there are benefits to using a healthy at-risk sample, the results may not generalize to individuals with schizophrenia or other psychotic disorders. Further research will need to determine whether social connectedness is a meaningful construct in clinical samples as well. Second, even as a group at risk for schizophrenia, this sample was

limited. This study employed a psychometric risk sample which identifies a very broad range of individuals purported to have the genetic predisposition for schizophrenia, most of which will never develop clinically significant symptoms of schizophrenia. Studies using other methods of identifying individuals at risk (first degree relatives of individuals with schizophrenia or ultra-high risk samples) would likely include individuals with more severe symptoms and poorer functioning. This study should be replicated in these samples. Finally, this sample included only university students, and even the schizotypy sample was composed of relatively high functioning individuals as evidenced by the unexpected finding of intact neurocognitive performance. Though this has been shown in prior studies (Chun et al., 2013; Jahshan & Sergi, 2007), it raises additional questions of generalizability. Replication of these results in a community-recruited psychometric high risk sample would further support the conclusions drawn here.

Also of note is that most of the measures included in this study (SPQ-BR, BSI, QoL-I, SCS-R) are self-report measures. Self-report measures are always vulnerable to response styles or intentional distortions. While the data were checked in the initial phase for validity as discussed in the method section, it is possible that some of the relationship between self-report data reflects measurement bias rather than true conceptual relationships. Much of the relation between the different constructs is likely due to method invariance. There are, however, reasons to think this may not explain the findings fully. At least one other study has examined this issue in schizotypy, specifically, whether subjective well-being was explained by negative affect as measured by a depression and anxiety scale (Abbott & Byrne, 2012). This study indicated that even among these self-report measures, poorer subjective well-being reflected more than just self-reported negative affect.

Further research employing multiple measurement methods should replicate these findings with non-self-report measures. Social connectedness in particular is conceptualized as a subjective concept and was, therefore, measured via self-report. It is conceivable, however, that one could design measures of social connectedness that do not rely on self-report such as behavioral observations of individuals in a social setting or in a structured interaction with a particular individual, perhaps varying in proximity of relationship (stranger, acquaintance, close friend). Another possibility would be to obtain a data from an individual in close relationship to the schizotypal participant regarding how socially connected that individual is. Examining these data in combination with self-report would enhance our understanding of social connectedness in schizotypal individuals and how it affects functioning.

Another limitation of this study is that only a few variables indicative of functioning and outcome were examined. Given that this is the first examination of social connectedness in schizotypy, it was not completely clear which variables would be most important to examine. In terms of outcome, quality of life seems to be affected by connectedness while performance on social competence measures does not. Further, negative schizotypal traits are more highly related to connectedness than positive or disorganized traits. There are a number of other variables that might be important to examine in schizotypy with relation to social connectedness such as social support, real world social behavior, and social skills. Much more research is needed examining the functional meaning of social connectedness such as how it affects specific social relationships or how it relates to social skills or typical social behavior. This would help determine how significant the concept is to the disorder and to what degree it might influence overall outcome, symptom expression, and symptom severity.

This study has important implications for how schizotypy is conceptualized. Given the data here, social connectedness appears to be a concept central to the definition of schizotypy. Schizotypy predicted social connectedness at a large effect size. While much of this may be due to method invariance, several nuances of this study suggest that this is not the full story. For example, regardless of whether quality of life was subjectively or objectively defined, social connectedness was important in understanding its relationship to schizotypy. Also, as stated above, prior research has indicated that poorer satisfaction with life is not explained by self-reported negative affect (Abbott & Byrne, 2012). Finally, there is evidence that although self-report may be vulnerable to response bias, at least when used in schizotypy samples, self-report captures something beyond what measures of performance in the laboratory capture (Chun et al., 2013). Perhaps the state of the field has not allowed for instruments and methods with enough sensitivity to reveal real deficits in performance or perhaps individuals are reporting something that would not be reflected in laboratory performance. They may be able to perform structured, time-limited tasks in the laboratory even though their real deficits negatively impact functioning in the real world. This may be due to the use of compensatory strategies, lower motivation, role or task ambiguity (see Quirk et al., 2007), or any number of other interferences. Relatedly, studies have found that state affective experience when reported in the lab by individuals with schizotypy differs from their broader appraisal of “trait-like” affectivity (Cohen et al., 2011). It would appear that laboratory studies of in-the-moment performance or experience is missing an integral aspect of the schizotypy phenomena. This integral aspect may be a connectedness-type concept. It is something internal and diffuse that may be difficult to assess in the moment but that clearly impacts functioning.

Meehl's theory of schizotypy and schizophrenia risk (1962; 1990) predicts a type of deficiency like social connectedness. Meehl posited that the genetic diathesis for schizophrenia present in schizotypy gave risk to a central nervous system anomaly which would lead to a diffuse impact on neural functioning, or a "loosening" of cognitive, physiological, and affective systems (1962). It is easy to imagine how a non-specific, diffuse abnormality of the neural system might lead to a broad range of subtle and inconsistent abnormalities in neurocognitive functioning—patterns that might be difficult to delineate in laboratory studies of healthy individuals. These patterns would become clearer as the illness progressed and the already weakened system would suffer large and specific cognitive problems such as those seen in schizophrenia. This same type of effect may operate within the social/emotional system. Meehl's hypothesized "hypokresia" (1962) may be something causing poor functioning within the social attachment system at a very basic level, this very same system that, in social animals, has evolved to process social information apart from non-social information in a more efficient manner. A breakdown in these structures and connections could cause a deficit in the intuitive ability to connect to the social world. When, as Meehl specified, social learning influences came into play (1962), this would mean that the natural developmental progression of connectedness (from companionship to affiliation to connectedness; Lee & Robbins, 1995) did not proceed as normal. As the individual developed, he or she would not learn to intuitively connect and process social information the same way as a non-schizotype. This would result in a deficit that might manifest inconsistently across time and situation. The individual would probably develop strategies to navigate social tasks, but these would not operate in the same manner as a normal individual. Though the deficit may not impact a circumscribed domain or skill in any predictable manner, it would be likely that, overall, functioning would be disrupted. As such, social

connectedness as a risk factor and central feature of schizotypy fits well with Meehl's theory and has the power to explain a number of the deficits seen in these individuals.

The findings in this study have important implications for status monitoring in high risk populations. Given that social connectedness is likely to play an important role in functioning and that it can be reliably measured, clinicians should consider assessing connectedness as part of their assessments with this population. Lower social connectedness could signify greater risk and greater need for intervention. Understanding social connectedness would also be important when designing interventions for both at-risk and decompensated individuals. These results suggest that poor connectedness is a major mechanism through which schizotypal traits are associated with poorer quality of life. In order to improve life quality, therefore, interventions will need to target connectedness deficits rather than simply focusing on schizotypal symptoms. This may result in substantial improvement over targeting symptoms or simply targeting social skills deficits designed to remediate social cognitive or social competence problems. Social connectedness, in contrast to measures of social cognition or social competence, is not performance or skill based. It reflects an intuitive sense of relatedness to the world without a clear functional purpose. As suggested above individuals may have developed compensatory strategies to navigate the social world while still lacking basic connectedness and this lack of connectedness appears to be related to life quality. Interventions targeting connectedness may be more helpful toward changing the developmental and emotional underpinnings of schizotypal traits and symptoms of schizophrenia and may result in further functional improvement. Finally, these findings have implications for interventions in schizotypal individuals. To date, most intervention efforts in at-risk populations have focused on psychotropic medications cognitive behavioral treatment, nutritional supplements, family interventions, and increased

monitoring or early detection (Bird, Premkumar, Kendall, Whittington, Mitchell, & Kuipers, 2010; de Koning, Bloemen, van Amelsvoort, Becker, Nieman, van der Gaag, Linszen, 2009; McFarlane et al., 2012; Morrison et al., 2012; Stafford, Jackson, Mayo-Wilson, Morrison, & Kendall, 2013). If, however, social connectedness deficits underlie much of the dysfunction and contribute to decreased life quality, early intervention efforts would do well to target these problems. This may take the form of treatments that encourage interpersonal connectedness perhaps by increasing attachment or utilizing support groups. Another avenue might be parent training or family therapies for families who are at high risk. These treatments could focus on teaching parents how to encourage healthy attachment to their children or increase healthy, supportive communication and bonding within families. Research examining the efficacy of these types of interventions would also increase the field's knowledge of the implications social connectedness for schizotypy and schizophrenia.

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Appendix A. Schizotypal Personality Questionnaire-Brief Revised Items

Positive Factor

Ideas of Reference/ Suspiciousness

- Do you sometimes feel that people are talking about you?
- Do you sometimes feel that other people are watching you?
- When shopping, do you get the feeling that other people are taking notice of you?
- I often feel that others have it in for me.
- Do you sometimes get concerned that friends or co-workers are not really loyal or trustworthy?
- Do you often have to keep an eye out to stop people from taking advantage of you?

Magical Thinking

- Do you believe in telepathy (mind-reading)?
- Do you believe in clairvoyance (psychic forces, fortune telling)?
- Have you had experiences with astrology, seeing the future, UFO's, ESP, or a sixth sense?
- Have you ever felt that you are communicating with another person telepathically (by mind-reading)?

Unusual Perceptions

- I often hear a voice speaking my thoughts aloud.
- When you look at a person or yourself in a mirror, have you ever seen the face change right before your eyes?
- Are your thoughts sometimes so strong that you can almost hear them?
- Do everyday things seem unusually large or small?

Negative Factor

No Close Friends/ Constricted Affect

- Do you feel that you cannot get "close" to people.
- I find it hard to be emotionally close to other people.
- Do you feel that there is no one you are really close to outside of your immediate family, or people you can confide in or talk to about personal problems?
- I tend to keep my feelings to myself.
- I rarely laugh and smile.
- I am not good at expressing my true feelings by the way I talk and look.

Social Anxiety

- Do you often feel nervous when you are in a group of unfamiliar people?
- I get anxious when meeting people for the first time.
- I feel very uncomfortable in social situations involving unfamiliar people.
- I sometimes avoid going to places where there will be many people because I will get anxious.

Disorganization Factor

Eccentric Behavior

- Other people see me as slightly eccentric (odd).
- I am an odd, unusual person.
- I have some eccentric (odd) habits.

People sometimes comment on my unusual mannerisms and habits.

Odd Speech

I sometimes jump quickly from one topic to another when speaking.

Do you tend to wander off the topic when having a conversation?

I often ramble on too much when speaking.

I sometimes forget what I am trying to say.

Appendix B. Social Connectedness Scale-Revised Items and Scoring

1. I feel distant from people.*
2. I don't feel related to most people.*
3. I feel like an outsider.*
4. I see myself as a loner.*
5. I feel disconnected from the world around me.*
6. I don't feel I participate with anyone or any group.*
7. I feel close to people.
8. Even around people I know, I don't feel that I really belong.*
9. I am able to relate to my peers.
10. I catch myself losing a sense of connectedness with society.*
11. I am able to connect with other people.
12. I feel understood by the people I know.
13. I see people as friendly and approachable.
14. I fit in well in new situations.
15. I have little sense of togetherness with my peers.*
16. My friends feel like family.
17. I find myself actively involved in people's lives.
18. Even among my friends, there is no sense of brother/sisterhood.*
19. I am in tune with the world.
20. I feel comfortable in the presence of strangers.

*reverse scored items

All scores summed to create composite score.

Responses made on a six point Likert scale ranging from 1 *strongly disagree* to 6 *strongly agree*.

Mean scale score = 88.02 standard deviation = 16.82 (Lee, Draper, & Lee, 2001)

Appendix C. Brief Assessment of Cognition in Schizophrenia Description

BACS -- DESCRIPTION OF NEUROCOGNITIVE MEASURES

The following section should be read before test administration. It is crucial that personnel who will test patients practice administering and scoring the entire test battery at their institutions in order to become familiar with the materials. Some tests require simultaneous administration and scoring; in others, additional attention must be paid to scoring details. All the tests require specific and rigorous adherence to instructions to achieve standardization. There are alternate forms for the tests that may be sensitive to practice effect or learning due to previous test administration. Patients should not receive the same forms or versions two times consecutively. The tests must be completed in the order in which they are listed in the BAC manual. The entire battery should last approximately 30 minutes, depending upon patient performance.

VERBAL MEMORY AND LEARNING

Verbal Memory. Patients will be presented with 15 words and then asked to recall as many as possible. This procedure is repeated 5 times.

Measures: verbal recall (number of words)

WORKING MEMORY

Digit Sequencing Task. Patients will be presented auditorily with clusters of numbers (e.g., 936) of increasing length. They are asked to tell the experimenter the numbers in order, from lowest to highest.

Measures: number of correct responses

MOTOR FUNCTION

Token Motor Task. Patients will be given 100 plastic tokens and asked to place them into a container as quickly as possible for 60 seconds.

Measures: number of tokens placed in the container during the 60 seconds

Symbol Coding Task. Patients will receive a key explaining how unique symbols correspond to the individual numerals 1-9. They will be asked to fill in the corresponding number beneath a series of symbols as quickly as possible. There is a 90 second time limit.

Measures: number of correct items

VERBAL FLUENCY

Semantic Fluency. Patients will be given 60 seconds to name as many words as possible within a given category.

Measures: number of words generated

Letter Fluency. In two separate trials, patients will be given 60 seconds to generate as many words as possible.

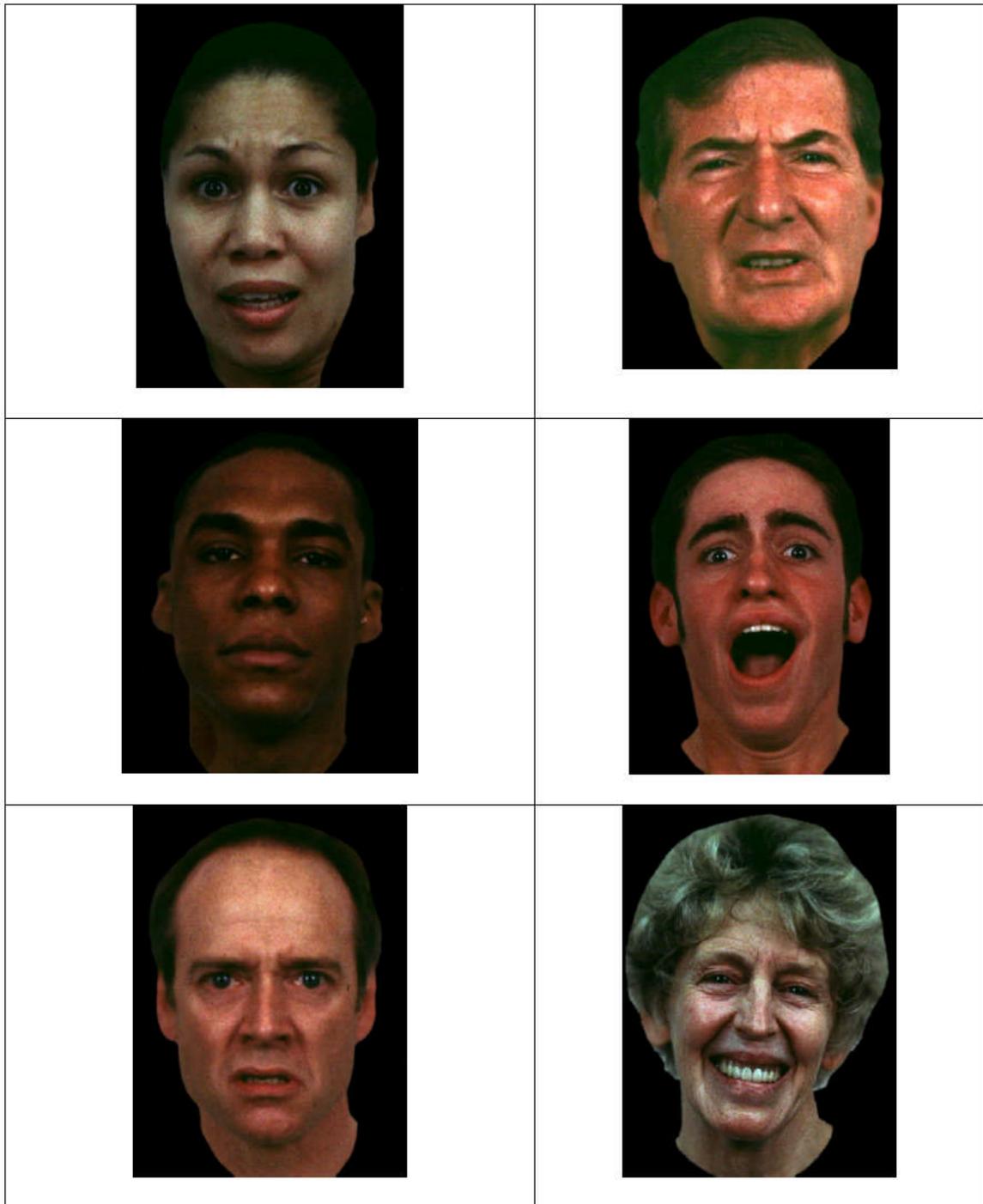
Measures: number of words generated

EXECUTIVE FUNCTION

Tower of London. Patients will look at two pictures simultaneously. Each picture will show three different colored balls arranged on three pegs, but the balls will be in a unique arrangement in each picture. The patient will give the minimum number of times the balls in one picture would have to be moved in order to make the arrangement of balls identical to that of the other, opposing picture.

Measures: number of correct responses

Appendix D. Examples of Stimuli Employed in the Penn Emotion Recognition Test



Appendix E. Lehman's Brief Quality of Life Interview Items

Select the item that best describes how you feel about your life in general.*	Terrible	Unhappy	Mostly Dissatisfied	Mixed	Mostly Satisfied	Pleased	Delighted
Select the item that best describes where you have been living during the past month.	In a house or apartment alone or with a spouse, friend, family or children.	In a house, apartment or boarding home with a part-time mental health professional.	In a treatment program with a full-time mental health professional	In a hospital or nursing home.	In a jail or prison	On the streets or in an emergency shelter for the homeless.	
Select the item that best describes how you feel about the privacy you have where you live.*	Terrible	Unhappy	Mostly Dissatisfied	Mixed	Mostly Satisfied	Pleased	Delighted
During the past month, did you work at a job for pay?	No	1-5 days	6-10 days	11-15 days	16 or more days	.	
During the past month, did you go to school?	No	1-5 days	6-10 days	11-15 days	16 or more days	.	
During the past month, did you do volunteer work?	No	1-5 days	6-10 days	11-15 days	16 or more days	.	
During the past month, did you keep house or take care of children?	No	1-5 days	6-10 days	11-15 days	16 or more days	.	
During the past month, did you go to a day program?	No	1-5 days	6-10 days	11-15 days	16 or more days	.	
Which of these activities did you consider your main activity during the past month?	Working at a job for pay	Going to school	Doing volunteer work	Keeping house, taking care of children	going to a day program	None of these	.
Select the item that best describes how you feel about the amount of fun	Terrible	Unhappy	Mostly Dissatisfied	Mixed	Mostly Satisfied	Pleased	Delighted

you have*

Select the item that best describes how you feel about how you spend your time.*

Terrible Unhappy Mostly Dissatisfied Mixed Mostly Satisfied Pleased Delighted

How often do you talk to a member of your family on the telephone?

Daily Weekly Monthly Less than Monthly Not At All . .

How often do you get together with a member of your family?

Daily Weekly Monthly Less than Monthly Not At All . .

Select the item that best describes how you feel about the way things are in general between you and your family.*

Terrible Unhappy Mostly Dissatisfied Mixed Mostly Satisfied Pleased Delighted

How often do you spend time with a friend who does not live with you?

Daily Weekly Monthly Less than Monthly Not At All . .

How often do you phone a friend who does not live with you?

Daily Weekly Monthly Less than Monthly Not At All . .

How often do you make plans ahead of time to do something with a friend?

Daily Weekly Monthly Less than Monthly Not At All . .

How often do you spend time with someone you consider more than a friend, like a boyfriend, girlfriend or you spouse?

Daily Weekly Monthly Less than Monthly Not At All . .

Select the item that best describes how you feel about the amount of friendship in your life.*

Terrible Unhappy Mostly Dissatisfied Mixed Mostly Satisfied Pleased Delighted

Select the item next to the amount of money you had to spend on yourself during the past month, not counting

Less than \$20 \$20 to \$50 \$51 to \$100 More than \$100 . .

money for room and board (housing and meals)								
In the past month, did you have enough money for food?	Yes	No
In the past month, did you have enough money for clothes?	Yes	No
In the past month, did you have enough money for housing?	Yes	No
In the past month, did you have enough money for transportation?	Yes	No
In the past month, did you have enough money for fun?	Yes	No
Select the item that best describes how you feel about how well off you are in financially.*	Terrible	Unhappy	Mostly Dissatisfied	Mixed	Mostly Satisfied	Pleased	Delighted	
In the past month were you the victim of any violent crime like assault, rape, mugging or robbery?	Yes	No
In the past month were you the victim of any non-violent crime like a theft, burglary or being cheated?	Yes	No
In the past month have you been arrested or picked up for any crime?	Yes	No
Select the item that best describes how you feel about the protection you have against being	Terrible	Unhappy	Mostly Dissatisfied	Mixed	Mostly Satisfied	Pleased	Delighted	

robbed or attacked.*

Overall, how would you rate your health?

Excellent

Very Good

Good

Fair

Poor

.

.

Select the item that best describes how you feel about your health in general.*

Terrible

Unhappy

Mostly Dissatisfied

Mixed

Mostly Satisfied

Pleased

Delighted

*Denotes items subjective quality of life items. All other items objective quality of life.

Appendix F: UCSD Performance-Based Skills Assessment Test-2 Description

The UPSA measures performance in five of domains of everyday functioning through the use of role plays and props. The following are the separate domains and how skills in each are demonstrated.

Financial skills

Individuals are asked to complete a series of change counting tasks with prop currency. Individuals are also given a prop utility bill and asked to identify important features of the bill.

Communication

Individuals are given a prop telephone and asked to make emergency calls, and call directory assistance to request a telephone number. They are then given a medical appointment confirmation letter, asked to call the number, and leave a message to reschedule the appointment.

Organization/planning

Individuals are asked read an advertisement for a theme park and then plan an outing including listing appropriate items to bring.

Transportation

Individuals are asked to read and interpret a bus route map and schedule to answer a list of questions regarding how to use the busses.

Household Management

Participants are given a recipe and asked to examine a number of prop pantry items. They are then asked to prepare a shopping list of recipe items not available in the pantry.

Appendix G: Institutional Review Board Approval

Application for Exemption from Institutional Oversight

Unless qualified as meeting the specific criteria for exemption from Institutional Review Board (IRB) oversight, ALL LSU research/ projects using living humans as subjects, or samples, or data obtained from humans, directly or indirectly, with or without their consent, must be approved or exempted in advance by the LSU IRB. This Form helps the PI determine if a project may be exempted, and is used to request an exemption.



Institutional Review Board
 Dr. Robert Mathews, Chair
 131 David Boyd Hall
 Baton Rouge, LA 70803
 P: 225.578.8692
 F: 225.578.6792
 irb@lsu.edu
 lsu.edu/irb

– Applicant, Please fill out the application in its entirety and include the completed application as well as parts A-E, listed below, when submitting to the IRB. Once the application is completed, please submit two copies of the completed application to the IRB Office or to a member of the Human Subjects Screening Committee. Members of this committee can be found at <http://www.lsu.edu/screeningmembers.shtml>

– A Complete Application Includes All of the Following:

- (A) Two copies of this completed form and two copies of part B thru E.
- (B) A brief project description (adequate to evaluate risks to subjects and to explain your responses to Parts 1&2)
- (C) Copies of all instruments to be used.

*If this proposal is part of a grant proposal, include a copy of the proposal and all recruitment material.

- (D) The consent form that you will use in the study (see part 3 for more information.)
- (E) Certificate of Completion of Human Subjects Protection Training for all personnel involved in the project, including students who are involved with testing or handling data, unless already on file with the IRB. Training link: (<http://phrp.nihtaining.com/users/login.php>)

1) Principal Investigator: Ph: Rank
 Dept: Ph: E-mail:

2) Co Investigator(s): please include department, rank, phone and e-mail for each
 *If student, please identify and name supervising professor in this space

3) Project Title:

IRB# <u>4922</u> LSU Proposal #
<input checked="" type="checkbox"/> Complete Application
<input checked="" type="checkbox"/> Human Subjects Training

4) Proposal? (yes or no) If Yes, LSU Proposal Number

Also, if YES, either
 This application completely matches the scope of work in the grant
 OR
 More IRB Applications will be filed later

Study Exempted By:
 Dr. Robert C. Mathews, Chairman
 Institutional Review Board
 Louisiana State University
 203 B-1 David Boyd Hall
 225-578-8692 | www.lsu.edu/irb
 Exemption Expires: 2-9-2013

5) Subject pool (e.g. Psychology students)

*Circle any "vulnerable populations" to be used: (children <18; the mentally impaired, pregnant women, the aged, other). Projects with incarcerated persons cannot be exempted.

6) PI Signature Date (no per signatures)

** I certify my responses are accurate and complete. If the project scope or design is later changes, I will resubmit for review. I will obtain written approval from the Authorized Representative of all non-LSU institutions in which the study is conducted. I also understand that it is my responsibility to maintain copies of all consent forms at LSU for three years after completion of the study. If I leave LSU before that time the consent forms should be preserved in the Departmental Office.

Screening Committee Action: Exempted <input checked="" type="checkbox"/> Not Exempted	Category/Paragraph <u>4</u>
Reviewer <u>Mathews</u> Signature <u>[Signature]</u> Date <u>2/10/10</u>	

Part 1: Determination of "Research" and Potential For Risk

- This section determines whether the project meets the Department of Health and Human Services (HSS) definition of research involving human subjects, and if not, whether it nevertheless presents more than "minimal risk" to human subjects that makes IRB review prudent and necessary.

Project Report and Continuation Application

(Complete and return to IRB, 131 David Boyd Hall, Direct questions go to IRB Chairman Robert Mathews 578-8692.)



Institutional Review Board
 Dr. Robert Mathews, Chair
 131 David Boyd Hall
 Baton Rouge, LA 70803
 P: 225.578.8692
 F: 225.578.5983
 irb@lsu.edu
 lsu.edu/irb

IRB#: E4922 Current Approval Expires On:
 Review Type: Risk Factor: Exempt
 PI: Alex Cohen Dept: Psychology Phone: 225-578-7017
 Student/Co-Investigator: n/a
 Project Title: Vocal markers of Schizophrenia spectrum disorders
 Number of Subjects Authorized: 500

Please read the entire application. Missing information will delay approval!

IRB Security of Data Agreement: <http://research.lsu.edu/files/item26774.pdf>

I. PROJECT FUNDED BY: n/a LSU Proposal #:

II. PROJECT STATUS: Check the appropriate blank(s) and complete the following:

- 1. Active, subject enrollment continuing; # subjects enrolled: 90
- 2. Active, subject enrollment complete; # subjects enrolled:
- 3. Active, subject enrollment complete; work with subject continues.
- 4. Active, work with subjects complete; data analysis in progress.
- 5. Project start postponed / /
- 7. Project cancelled: no human subjects used.
- 6. Project complete; end date / /

III. PROTOCOL: (Check one).

- Protocol continues as previously approved
- Changes are requested*
--List (on separate sheet) any changes to approved protocol.

IV. UNEXPECTED PROBLEMS: (did anything occur that increased risks to participants);

---State number of events since study inception: 0 since last report: 0
 ---If such events occurred, describe them and how they affect risks in your study, in an attached report
 ---Have there been any previously unreported events? Y/N

V. CONSENT FORM AND RISK/BENEFIT RATIO:

Do new knowledge or adverse events change the risk/benefit ratio? Y/N n/a
 Is a corresponding change in the consent form needed? Y/N n/a

VI. ATTACH A BRIEF, FACTUAL SUMMARY of project progress/results to show continued participation of subjects is justified; or to provide a final report on project findings.

VII. ATTACH CURRENT CONSENT FORM (only if subject enrollment is continuing); and check the appropriate blank;

- 1. Form is unchanged since last approved
- 2. Approval of revision requested herewith: (identify changes)

Signature of Principal Investigator: [Signature] Date: 5-7-12

IRB Action:	<input checked="" type="checkbox"/> Continuation approved;	Approval Expires: <u>5/7/13</u>
	<input type="checkbox"/> Disapproved	
	<input type="checkbox"/> File Closed	
Signed	<u>[Signature]</u>	Date <u>5/8/12</u>

Print Form

CONSENT FORM

Study Exempted By:
Dr. Robert C. Mathews, Chairman
Institutional Review Board
Louisiana State University
203 B-1 David Boyd Hall
225-578-8692 | www.lsu.edu/irb
Exemption Expires: 5/7/2015

Project Title: Identifying the vocal markers of schizophrenia spectrum disorders

Performance Site:
322 Audubon Hall, LSU, Baton Rouge, LA 70803.

Investigator: The following investigator is available for questions Monday-Friday, 9:00 a.m.- 4:30 p.m.
Alex S. Cohen, Ph.D.
Psychology Department, LSU
(225) 578-7017

Purpose of the Study: The purpose of this research project is to understand the relationship between cognition, emotion and social functioning and personality characteristics in college students.

Inclusion Criteria: You are being asked to participate in this study because you are a Louisiana State University undergraduate who is over the age of 18 who showed a scoring pattern of interest on our on-line personality screening measures.

Exclusion Criteria: Individuals showing the scoring pattern of interest on the personality screening measure are eligible to participate. There are no specific exclusion criteria.

Maximum Number of Subjects: The maximum number of participants for this phase will be 500.

Study Procedures/Description of the Study: I am aware that this study will take approximately two hours. I will be asked to fill out a number of questionnaires that assess my emotion, personality, cognitive functions and mental health history. My voice will be recorded during several parts of this study. I also understand the experimenters will measure skin conductance using 2 sensors on my non-dominant fingertips. For participating in this session, I will be compensated \$20 cash.

Benefits: I understand that I will not directly benefit from participating in this study. My participation will help researchers find out more information about mental illnesses.

Risks/Discomforts: I understand that I will be expected to complete the two-hour long session. This may be inconvenient. I also recognize that I will be asked to talk about my mental health history. Other than this discomfort, there are no known risks.

Right to Refuse: Participation in this study is voluntary. I may refuse to answer any questions or discontinue any test I am taking. Further, I can change my mind and withdraw from this study at any time without penalty or loss of any benefit to which I would otherwise be entitled to.

Privacy: All information obtained in this study will be kept confidential unless release is legally compelled. Limits to confidentiality include situations where an individual is at risk of hurting themselves (e.g., suicide) or hurting someone else (e.g., homicide, child abuse). I understand that the investigators are required by law to report any reasonable suspicions.

All records will be kept in a locked laboratory in a secure facility. Electronic data will be entered without identifying information and will be password protected. To ensure confidentiality, I will be assigned a number. All information collected during this study will be linked to this number and kept separate from any identifying information such as my name. Results of the study may be published, but no names or identifying information will be included for publication.

Financial Information: I will receive \$20 cash upon the completion of the session or receive four experimental credits towards a psychology course.

Withdrawal: Participation in this study is voluntary. I may withdraw from this study at any time without penalty or loss of any benefit to which I would otherwise be entitled to.

Signatures:

The study has been discussed with me and all my questions have been answered. I may direct additional questions regarding study specifics to the investigators. If I have questions about subjects' rights or other concerns, I can contact Robert C. Mathews, Chairman, LSU Institutional Review Board, (225)578-8692. I agree to participate in the study described above and acknowledge the researchers' obligation to provide me with a copy of this consent form if signed by me.

Participant Signature

Date

Vita

Laura Brown completed her Bachelor of Science with a major in Psychology and a minor in Latin at Louisiana State University. She went on to pursue graduate training in Psychology with a focus on schizophrenia research and completed her Master of Arts in Clinical Psychology at Louisiana State University. Her master's thesis focused on facial emotion recognition in individuals at risk for schizophrenia. The current research is in partial fulfillment for a Doctorate of Philosophy in Clinical Psychology. She is currently completing her pre-doctoral internship at Northeast Florida State Hospital focusing on forensic psychology and assessment and treatment of severe mental illness. Laura's primary research interest is in risk for schizophrenia and schizophrenia spectrum disorders. Laura's clinical work focuses on diagnostic and forensic assessment and her clinical training has largely occurred in state civil and forensic psychiatric hospitals. In addition to assessment, Laura's has extensive training in evidence based treatment of severe mental illness. Her orientation is primarily cognitive-behavioral with a psychodynamic influence. Clinical interests include assessment, schizophrenia, personality disorders, and forensic issues.