# ABSTRACT

Title of Dissertation: NEUROPSYCHOLOGICAL CHARACTERISTICS OF PUTATIVE SCHIZOTYPES: A COMPARATIVE STUDY

Kari E. Tervo, Doctor of Philosophy, 2004

Dissertation Directed By: Dr. Jack J. Blanchard, Department of Psychology

The development of strategies for the early prediction, detection, and treatment of schizophrenia, a disorder of neural origin, has been a significant aim of schizophrenia research. Understanding and predicting the pathogenesis of schizophrenia is imperative for the early intervention and possible prevention of the myriad negative outcomes associated with the disorder. The Chapman scales are used to identify individuals who show behavioral markers of the disorder, but few studies have examined the neurocognitive characteristics evidenced by those who are designated as schizotypes by those scales. The purpose of this study was to examine two groups designated as putative schizotypes by the Revised Social Anhedonia Scale (RSAS; SocAnh group) and the combined Perceptual Aberration and Magical Ideation Scales (PAS/MIS; PerMag group) in concert with neurocognitive evidence in order to determine whether the RSAS or PAS/MIS is the most valid indicator of the liability for schizophrenia and related disorders. This study was the first to simultaneously examine SocAnh individuals and PerMag individuals on a variety of neurocognitive indices in a community sample. Results indicate that while there do not appear to be neurocognitive differences between the groups with respect to

attention, working memory, and general memory indices, SocAnh individuals had more schizophrenia-spectrum characteristics and diagnoses, as well as lower global functioning, than did PerMag individuals or controls. Null neurocognitive results may have occurred for a variety of reasons. First, the measures in this study are designed to detect gross impairment rather than the attenuated impairment that schizotypes may have relative to schizophrenia patients. Gross neurocognitive decline may not occur until after the expression of schizophrenia symptoms. Second, this study used pure schizotype samples, which proved to be instrumental in demonstrating that a combination of schizotypy characteristics may be a more reliable marker of schizophrenia liability. Finally, this community sample may be a better representation of schizotype functioning than are college samples. Future directions are discussed.

# NEUROPSYCHOLOGICAL CHARACTERISTICS OF PUTATIVE SCHIZOTYPES: A COMPARATIVE STUDY

By

Kari Ellen Tervo

Dissertation submitted to the Faculty of the Graduate School of the
University of Maryland, College Park, in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy
2004

Advisory Committee:
Dr. Jack Blanchard, Chair
Dr. Ellen Fabian, Dean's Representative
Dr. Robert Coursey
Dr. James Gold
Dr. Barry Smith

# Dedication

This dissertation is dedicated to my grandmother, Helen Tomasic. In completing high school at the age of 65, she instilled in me the intrinsic value of an education. She has also fortified me with support and wisdom along the way. Grammy, you rock in so many ways, and I love you.

Acknowledgements

I would like to acknowledge the following people for their support, in a variety of incarnations, while I was completing this dissertation:

*My mentor, Jack Blanchard. He has influenced my critical thinking skills and my desire to be a strong schizophrenia researcher.

*My dissertation committee. I found that my proposal meeting was a very pleasant exchange of thought-provoking ideas. Your feedback was instrumental in the development of this manuscript.

*Ed McCann. In service of my last vestiges of academic sanity, he reviewed my work, accurately assisted with tedious transcriptions, and several times (without complaint), got up in the middle of the night to coax an obstreperous computer into behaving. He also pretended to be interested in working memory.

*Claire Murray. Whenever I became stressed about writing my dissertation, she would shrug and state in a reassuring tone, “Just think of it as a really long paper.” Somehow, that worked.

*Officer Derrick Thompson of the UMCP Police Department. He came to the rescue when a librarian locked me into McKeldin Library, and, with good humor, let me into the stacks to copy the article I absolutely needed to get any work done that night.

Table of Contents

List of Tables

List of Figures

# CHAPTER ONE: INTRODUCTION AND LITERATURE REVIEW

The development of strategies for the early prediction, detection, and treatment of schizophrenia has been a significant aim of schizophrenia research in recent years (e.g., Yung et al., 1998; Tsuang, Stone, & Faraone, 2000; McGorry et al., 2001). This line of inquiry rings with a particular urgency and importance. Even though a relatively small percentage of the adult population presents with schizophrenia (approximately 1%; American Psychiatric Association, 1994), the various symptoms and sequelae of the disorder require a significant amount of time, money, and other resources to be dedicated towards treating those with schizophrenia (Meltzer, 1999).

Beyond considerations of the use of resources, schizophrenia takes a personal toll on those afflicted. Individuals with schizophrenia are less likely to marry than those who do not have schizophrenia, manifest impairments in work-related functioning, and sometimes have difficulties independently addressing life needs (Zaluska, 1998). Moreover, left untreated, psychosis is thought to have a toxic effect on the brain such that further episodes of psychosis are more likely to manifest (Wyatt, 1995). Fortunately, early identification and treatment of schizophrenia may result in both neuroprotection and the mitigation of social sequelae of the disorder (Tsuang, Stone, & Faraone, 2000); however, some negative consequences may still be expected once the diathesis is expressed. Clearly, understanding and predicting the pathogenesis of schizophrenia is imperative for the early intervention and possible prevention of the myriad negative outcomes associated with the disorder. Such prediction could prevent disturbances in many areas, including the social, interpersonal, cognitive, and affective domains (Tsuang

et al., 2000). In response to this aim, several strategies have been put forth that attempt to identify the behavioral, genetic, and developmental markers of schizophrenia.

The purpose of the proposed study is to examine two of the scales that represent one of these strategies (the psychometric detection paradigm) in concert with neurocognitive evidence in order to determine which scale is the most valid indicator of the liability for schizophrenia and related disorders. This has possible implications in terms of early intervention. This chapter will review the related literature. First, methods of identifying individuals who are possibly psychosis-prone will be discussed. Next, cross-sectional and longitudinal data as they relate to two scales of the psychometric detection paradigm (the combined Perceptual Aberration/Magical Ideation scales and the Revised Social Anhedonia Scale) will be presented in order to establish the current state of the field as it relates to schizophrenia prediction using psychometric means. It will become apparent from this discussion that using this method alone can yield conflicting findings. Thus, a strategy of examining the neurocognitive "signatures" of individuals who have elevated scores on either scale will be suggested in order to strengthen the approach and to determine which scale is more likely to have validity in the prediction of schizophrenia and related disorders.

Predicting the pathogenesis of schizophrenia is not a task for the faint of heart or the easily discouraged. It is difficult to predict something that has not been precisely defined; the heterogeneity of schizophrenia presents one stumbling block on the path to prediction. Various manifestations of schizophrenia exist, and it is unclear whether these presentations reflect one shared pathophysiology or several different pathophysiologies (Buchanan & Carpenter, 1994). To further complicate the picture, of individuals who

carry the genetic liability for schizophrenia, some will develop the disorder and some will be spared, implicating developmental and environmental antecedents. Myriad factors have been implicated, and it is difficult to determine which variables, and how much of them, combine to push the individual over the threshold into expressed schizophrenia. Meehl (1962, 1990) proposed that one way to predict the presence of the liability for schizophrenia is to examine certain personality characteristics that are likely to be present in those who carry the liability for schizophrenia and have experienced certain environmental situations that potentiate the development of a schizotypal personality organization.

Schizotaxia: An Approach for Understanding the Pathogenesis of Schizophrenia

Meehl postulated that schizophrenia and associated disorders arise from a heritable neural aberration that is ubiquitous in the central nervous system, present at "all levels, from the sacral cord to the frontal lobes" (Meehl, 1992, p. 936), which likely manifests at a biological level (Grove, 1982). The presence of this neural defect, which Meehl termed "schizotaxia," renders one vulnerable to social learning processes (including stress and family conflict) that potentiate the development of the phenotypic expression of schizotaxia, or schizotypy, which is represented by molar behavioral characteristics, including social anhedonia, magical ideation, and other factors (Grove, 1982). Thus, in this view of schizophrenia liability, genetic transmission is not the necessary and sufficient mechanism for the expression of the disorder; rather, environmental factors must be considered in concert with the genetic liability.

Schizotypy, the phenotypic expression of schizotaxia, is a personality organization that represents a latent liability for schizophrenia, and is characterized by

subtle thought disorder ("cognitive slippage"), ambivalence, anhedonia, and interpersonal aversiveness (Meehl, 1962), four core traits that develop as a result of the schizotype's social learning history. Meehl emphasized, however, that despite the potential for the schizotype to develop schizophrenia or related manifestations of schizotaxia, individual difference factors, such as an ability to resist stress, could provide a buffer between genetic predisposition and the environment, resulting in more favorable outcomes. Although about 10% of schizotypes are predicted to decompensate into schizophrenia (Meehl, 1990), others may develop milder manifestations of schizotaxia, such as paranoid personality disorder, while still others may not evidence any clinical characteristics.

## Identifying Schizotypes

How are schizotypes identified? Three main approaches serve to provide a means to study this phenomenon. One strategy is to examine individuals who meet the diagnostic criteria for the Diagnostic and Statistical Manual (DSM; American Psychiatric Association, 1994)-defined schizophrenia spectrum personality disorders. The conceptualization of these disorders emerged from observations that some individuals evidenced ways of thinking, perceiving, and behaving that were associated with the genetic liability for schizophrenia. However, this approach is limited because most schizotypes do not present at clinics (Lenzenweger, 1998), and the schizophrenia-spectrum disorders are not always necessarily related to schizotaxia; paranoid personality disorder is elevated in relatives of patients with unipolar depression (Maier et al., 1994). Moreover, not all schizotypes will necessarily meet the diagnostic criteria for those disorders even though they may evidence subthreshold behavioral markers or a certain neurocognitive profile. Although some schizotypes may develop DSM-defined

schizotypal personality disorder (STPD), the distinction between Meehl's schizotypy and STPD is an important one. STPD is not necessarily related to the schizophrenia diathesis; in some studies, the positive-like symptoms of schizotypal personality disorder did not appear to be associated with genetic transmission (Torgersen, 1985), while in others (Faraone et al., 2001), it was associated with a familial history of affective disorders. Although STPD is certainly found in the relatives of individuals with schizophrenia, its presentation in this group differs from that found in relatives of affective disorder patients; STPD relatives from families with a history of schizophrenia tend to show more of the negative-like symptoms of the disorder, while those from families with a history of affective disorders display more of the positive-like symptoms of STPD (Faraone et al., 2001). Thus, it is apparent that schizotaxia is not best identified via the diagnosis of schizophrenia-spectrum personality disorders.

Another strategy is to examine family members of individuals with schizophrenia, who presumably carry the same liability for schizophrenia as their ill family members and thus are considered to be "genetic" schizotypes (Lenzenweger, 1998). Meehl originally proposed that a single gene, or "schizogene," was responsible for the presence of schizotaxia. The preponderance of subsequent research, however, suggests that the genetic risk for schizophrenia is polygenic (Gottesman, 1991). Nonetheless, family studies of schizophrenia support Meehl's assertion that genetic transmission underlies the etiology of schizophrenia. As early as 1909, Emil Kraeplin noted that the family members of those with schizophrenia had a tendency to evidence anomalies such as eccentric personalities, what might now be diagnosed as a schizophrenia-spectrum personality disorder. Presaging Meehl, Kraeplin surmised that these family members had

an unexpressed latent liability for schizophrenia (Lenzenweger, 1998). A large corpus of studies examining the family members of individuals with schizophrenia provides support for the idea that the latent liability for schizophrenia has a genetic basis. Many studies have demonstrated that the relatives of individuals with schizophrenia show increased rates of schizophrenia and schizophrenia-spectrum personality disorders as compared to relatives of control probands (e.g., Kendler, 1988, Parnas et al., 1993; Asarnow et al., 2001), and greater levels of shared genes are associated with a greater risk for schizophrenia (Gottesman, 1991). Gottesman & Shields (1972) found that 64% of monozygotic twins raised apart were concordant for schizophrenia, providing a compelling example of the role that genetics plays in the transmission of schizophrenia. Overall, schizophrenia, schizotypal personality disorder, and non-affective psychoses appear to be strongly aggregated in the relatives of schizophrenia probands (Kendler & Gardner, 1997). Family studies of schizophrenia demonstrate that there does indeed appear to be a genetic link in the pathogenesis of schizophrenia. Family members of individuals with schizophrenia are more likely to develop the disorder or related maladies themselves; studies of twins raised apart provide especially compelling evidence for this link. This evidence converges to suggest that a genetic process is at work in the transmission of schizophrenia and schizophrenia-spectrum disorders.

Although family studies are compelling in their illustration of the genetic transmission of schizophrenia and related disorders, the approach suffers from some limitations. First, not all biological relatives of individuals with schizophrenia are likely to be schizotaxic (Lenzenweger, 1998); approximately 80% of individuals with schizophrenia do not have a relative with schizophrenia (Gottesman, 1991), although this

may merely reflect a lack of penetrance of the disease process. Many individuals at risk for schizophrenia may not have a positive family history for the disorder, thus, an identification strategy that includes only relatives of schizophrenia patients may neglect a large portion of individuals who are at risk for the disorder. Second, only a small subset of individuals who are at risk for the disorder are likely to display measurable markers of their liability. Clearly, the role of genetic factors in the development of schizophrenia and related disorders is strong, yet not wholly deterministic (Gottesman & Erlenmeyer-Kimling, 2001).

The Psychometric Detection Paradigm

In order to cast a wider net in the identification of schizotypes than the schizophrenia-spectrum or genetic approaches can offer, the psychometric detection paradigm was developed. This third approach to identifying schizotypes is a questionnaire approach that reflects a promising development in schizophrenia-proneness research. Meehl (1962) postulated that schizotypic individuals display behavioral markers that indicate one's liability for schizophrenia. To review, he proposed that four core symptoms marked such a status: cognitive slippage, interpersonal aversiveness, anhedonia, and ambivalence to environmental cues and stimuli. The psychometric paradigm for the detection of schizotypy capitalizes on the probability that these indices actually are fallible markers of the schizotypic phenotype and can be used to identify carriers of the schizophrenia diathesis. How are these indicators measured? The psychometric approach utilizes paper and pencil self-report questionnaires with items that tap the core domains of schizotypy. This approach, besides being efficient, offers several advantages over other methods of identifying individuals at risk for schizophrenia. One

advantage is that given the need to screen large numbers of individuals to detect a low base-rate phenomenon, this method is an economical means for such screening. Another advantage to this approach in detecting psychosis-proneness is that it taps personality characteristics that may reflect the latent liability for schizophrenia in absence of the clinical syndrome; active symptoms are not required. Related to this, individuals can be studied free of the confounds of a regimen of neuroleptic medication. Moreover, individuals identified by this method can be examined longitudinally in order to follow the development of schizophrenia symptoms, if any. A fifth advantage is that the scales are not derived from the DSM classification of schizophrenia and schizophrenia-spectrum disorders; rather it is theoretically driven, that is, based on Meehl's (1962) concept of schizotypy. Thus, there is a higher probability that one will be identified as a schizotype even if one falls into the subthreshold range based on DSM-IV criteria of schizophrenia-spectrum disorders. Finally, given enough empirical evidence to support this method, it is possible that it may someday be used for early identification and intervention strategies. For example, if one could state with some certainty that certain individuals were going to develop schizophrenia, further research could focus on using prophylactic strategies to aid in the cessation of that development, such as the administration of low doses of neuroleptic medications.

A host of measures employing this approach have been developed since the 1970s, including Eysenck and Eysenck's (1976) Psychoticism Scale, Golden and Meehl's (1979) Schizoida Scale, and Raine's (1991) Schizotypal Personality Questionnaire. However, the most promising and most commonly used questionnaires for assessing schizotypy are scales developed by Chapman, Chapman and colleagues (referred to here

as “the Chapman scales”), which represent a translation of Meehl’s theory of schizotypy into a psychometric instrument. These scales include the Perceptual Aberration Scale (PAS, Chapman, Chapman & Raulin, 1978), which taps distortions in the perception of one’s body and the environment, the Magical Ideation Scale (MIS; Eckblad & Chapman, 1983), the items of which represent a belief in magical forms of causation, and the revised Social Anhedonia Scale (RSAS; Eckblad, Chapman, Chapman, & Mishlove, 1982), which taps schizoid asociality. At least one study has established that the PAS and MIS are highly intercorrelated, with Pearson correlation coefficients of .68 in men and .70 in women (Chapman, Chapman, Kwapil, Eckblad, & Zinser, 1994). Thus, many studies have employed mixed samples that include individuals with elevations on one or both of these scales. However, some studies employ “pure” groups in which individuals are elevated on only one scale or the other, while others only utilize a single scale to identify putative schizotypes. In the following sections, samples in studies that used a combined extreme group will be referred to as high “PerMag” scorers, as they scored high on either or both of the Perceptual Aberration Scale and the Magical Ideation Scale. Samples that were identified based on elevations on only one scale will be indicated as such.

## The Chapman Scales: Validity and Controversy

Importantly, individuals with schizophrenia themselves display aberrant scores on the Chapman scales (Katsanis, Iacono, & Beiser, 1990; Laurent et al., 2000), as do individuals with schizophrenia-spectrum personality disorders (Lyons, Toomey, Faraone, Kremen, Yeung, & Tsuang, 1995). Scores on these scales, however, may only be related to psychosis in general; they do not differentiate between affective psychosis and

individuals with psychosis related to schizophrenia (Katsanis et al., 1990), but this may be an artifact. As will be discussed more in depth later, the PAS and MIS appear to identify individuals with an array of psychopathological symptoms. This presents a problem: Both the PAS/MIS and RSAS are presented as instruments that identify putative schizotypes; however, the findings regarding the two are inconsistent with the conjecture that they are both tapping schizotypy. While PAS/MIS elevations are associated with general psychopathology, the RSAS appears to be associated more specifically with schizophrenia-related pathology. In addition, none of the scales alone appears to be a particularly strong predictor of actual schizophrenia. This review of the Chapman scale validity literature will lead to the suggestion that another strategy, that is, an examination of the neurocognitive signatures of the two groups in concert with scale elevations, will likely help in determining which of the two scales is the most valid predictor of schizophrenia.

A literature on the validity of each of the Chapman scales has emerged since their inception. Broadly, this literature indicates that individuals with aberrant scores on the Chapman scales evidence clinical and cognitive deficits similar to those with schizophrenia. Moreover, genetic transmission appears to come into play in this expression of schizotypy. The following sections will expand on this literature with regard to individuals with elevations on the Perceptual Aberration and Magical Ideation Scales, as well as the Revised Social Anhedonia Scale. These sections will focus on general validity data; studies that have neurological and neurocognitive foci will be discussed later as a focus of the present study.

One caveat to consider in the interpretation of this evidence is that reports are not always clear as to how groups considered elevated on the Chapman scales were selected. Some studies may have included samples that were not "pure" groups; that is, while they were elevated on the scale(s) of interest, they may also concurrently have had elevations on one or more of the other scales, which could influence the results.

The Perceptual Aberration and Magical Ideation Scales

In his original treatise on schizotypy, Meehl (1960) attached importance to body image and perceptual distortions, and reaffirmed this in later writings (Meehl, 1990); the PAS and MIS were developed to tap such experiences. The scales have been shown in one study to have good convergent and discriminant validity (Bailey, West, Widiger, & Freiman, 1993), and individuals with schizophrenia have elevations on the scales, providing some evidence of the scales' construct validity (Chapman et al., 1978; Laurent et al., 2000). However, much of the evidence of the scales' predictive and convergent validity is mixed. As will be detailed in this section, the PAS and MIS, while associated with schizophrenia-spectrum characteristics in some cases, are also associated with the facets of other disorders. Overall, it appears that a wide net is cast when PAS and MIS elevations are considered; although true schizotypes may be included in the group captured, individuals with other forms of psychopathology are subsumed as well.

Evidence concerning familial elevations on the PAS does not show a clear pattern. In one study, relatives of schizophrenia patients showed elevations on the PAS (Laurent et al., 2000), yet in others, relatives of schizophrenia patients did not evidence these same elevations on the PAS or the MIS (Katsanis et al., 1990; Franke et al., 1994). Relatives of high PAS scorers, however, tend to have family members who are also

elevated on the PAS (Battaglia, Gasperini, Sciuto, Scherillo, Diaferia, & Bellodi, 1991; Berenbaum & McGrew, 1993, Lenzenweger & Loranger, 1989a). In one study (Lenzenweger & Loranger, 1989a), individuals with elevations on the PAS were more likely to have relatives with schizophrenia than were control subjects. This evidence converges to suggest that the same genetic process is responsible for both schizophrenia and high PAS scores, but one limitation of this study must be considered. Lenzenweger and Loranger (1989a) employed a chart review method of determining if the relatives had schizophrenia; however, this method is often not sensitive to the presence of other disorders (Lenzenweger & Loranger, 1989a), thus, relatives of high PAS scorers may also have had a variety of other disorders. This would be consistent with data that shows an increased risk for affective disorders in individuals with PAS elevations (e.g., Chapman et al., 1994).

PAS/MIS Elevations and Similarities to Schizophrenia

Research suggests that individuals who score high on the PAS have clinical characteristics similar to individuals with schizophrenia, but these similarities may be related to other disorders as well. High PAS scorers manifest schizophrenia spectrum personality characteristics (Blanchard & Brown, 1999) and report frequent psychotic-like experiences (Chapman et al., 1980; Allen, Chapman, Chapman, Vucehtich, & Frost, 1987), as do high PerMag scorers (Kwapil, Chapman, & Chapman, 1999; Tallent & Gooding, 1999). Eighty-five percent of individuals who scored high on the PAS had psychotic and psychotic-like experiences in one study (Allen et al., 1987), which appears to offer striking proof of the PAS in predicting such experiences. However, these individuals also scored high on the Depression subscale of the General Behavior

Inventory, suggesting that a process related to affective psychosis could account for these findings.

The Minnesota Multiphasic Personality Disorder (MMPI) profiles of high PAS scorers are similar to those of schizophrenia patients (Lenzenweger, 1991); schizophrenia-related point codes (e.g., the 2-7-8 profile) were five times as prevalent among high PAS scorers than among controls. This study, however, did not examine the prevalence of non-schizophrenia related point codes among this group, thus revealing a confirmatory bias. Another investigation (Fujioka & Chapman, 1984) provides a clearer picture of the relation of MMPI elevations to Chapman scale scores. While PerMags and individuals with a 2-7-8 MMPI point code profile did not differ on the basis of psychotic and psychotic-like experiences, PerMags showed elevations on hypomania, while the 2-7-8 group did not, providing further evidence that the PAS/MIS may tap individuals with general psychopathology rather than true schizotypes. High PerMag scorers were also elevated in terms of schizotypal experiences while 2-7-8 elevators were not, but these experiences, including feeling different from others, extreme anger, and depersonalization, are also related to disorders such as borderline personality disorder and are not specific to the schizotypal personality disorder diagnosis. Indeed, borderline personality disorder diagnoses explain a significant amount of the variance in MIS and PAS scores (Lyons, Toomey, Faraone, Kremen, Yeung, & Tsuang, 1995). Offering more proof that the PAS and MIS capture a class of individuals that includes more than schizotypes alone, the 2-7-8 profile and the PerMag classification select different groups of individuals, with little overlap.

Communication Deviance

A small literature suggests that high PAS scorers show some of the same communication anomalies as schizophrenia patients. With regard to general communication deviance, individuals with high scores on the PAS have been observed to display more socially inappropriate behaviors, including hostile behaviors, than were controls, although unlike schizophrenia patients, they did not display any specific social skill deficits relative to controls (Numbers & Chapman, 1982), nor did they show skill deficits in a similar investigation (Haberman, Chapman, Numbers, & McFall, 1979).

There is also some evidence that high PAS and MIS scorers display the bizarre and unusual verbalizations that are one of the hallmarks of schizophrenia, bearing out Meehl's (1962) conjecture that schizotypes will display cognitive slippage, or attenuated thought disorder. PerMags gave unusual idiosyncratic responses in an unstructured response task (Miller & Chapman, 1983); these results were confirmed in a study of high PAS scorers (Coleman, Levy, Lenzenweger, & Holzman, 1996), in which such individuals also used peculiar language in their responses. Miller and Chapman (1983), however, used a sample that also had elevations on the Impulsive Nonconformity Scale along with the PAS/MIS elevations; such individuals may give unusual responses consistent with their proclivity to appear different from others; thus, it is unclear whether that tendency or processes related to PAS elevations account for these results.

When presented with unfamiliar proverbs, PerMag individuals tend to offer bizarre and idiosyncratic interpretations of them, along with evidencing an element of concreteness in their responses (Allen & Schuldberg, 1989), although their responses do not differ from those of controls when responding to familiar proverbs. Allen, Chapman,

& Chapman (1987) present more evidence of possible cognitive slippage in PerMags; in their study, PerMags were more deviant than controls on a word association task, offering bizarre responses that were not obviously related to the target word. It is important to consider, however, that PerMag participants who were also high in depressive symptoms were the most deviant; individuals who were low in depressive symptoms did not differ from control participants on the word association task. This could indicate that PAS/MIS elevations are associated with affective psychosis rather than schizophrenia-related symptoms; mild cognitive slippage has also been found in individuals with affective psychosis (Miller & Chapman, 1983). However, higher scores on the depression scale could also be associated with symptom severity and distress related to perceptual aberrations and magical ideation, obfuscating a more exact interpretation of this evidence.

Other cross-sectional evidence converges to suggest that PAS and MIS elevations are associated with various anomalies. Believers in paranormal phenomena have high MIS scores (Pizzagalli, Lehman, Gianotti, Koenig, Tanaka, Wackerman, & Brugger, 2000), and PerMag individuals tend to be ambidextrous (Chapman & Chapman, 1987). This information is of potential importance because of handedness research in schizophrenia that shows that many schizophrenia patients are left-handed, suggesting a degree of developmental instability (Yeo et al., 1997). Perhaps the same holds true for PerMags.

These cross-sectional data indicate that high PAS and MIS scorers display characteristics similar to individuals with schizophrenia, but are the scales actually predictive of a later expression of schizophrenia? One 10-year longitudinal study

(Chapman, Chapman, Kwapil, Eckblad, & Zinser, 1994) found that individuals classified as PerMag reported more psychotic-like experiences than did control subjects; however, the group did not significantly differ from control subjects on the basis of schizophrenia diagnoses. Rather, PAS and MIS elevations predicted the development of general psychopathology. For instance, individuals classified as PerMag demonstrated a significant rate of psychological disorders such as depression, mania, substance abuse, and borderline personality disorder relative to controls.

In line with this, a host of other studies provides evidence that PAS/MIS elevations actually capture a group of individuals with pathology that is not specific to schizophrenia-related disorders. Kwapil (1998) suggests that the PAS identifies those at risk for psychoses, mood disorders, and substance use, but not specifically schizophrenia-spectrum disorders. Lenzenweger & Loranger (1989b) report that in a sample of psychiatric patients with no history of psychosis or bipolar disorder, high PAS scores were associated with symptoms of anxiety disorders, depression, avoidant personality disorder, and obsessive compulsive personality disorder, along with symptoms of the schizophrenia-spectrum personality disorders. Park and colleagues (1995) circumstantiate this evidence with their finding that PAS elevations were associated with higher levels of symptoms of anxiety and depression. Other data suggest that PAS/MIS elevations are associated with depressive symptoms and hypomanic episodes (Fujioka & Chapman, 1984).

Biological data support the conjecture that the PAS and MIS identify a group at risk for general pathology. Male PerMags have a pattern of abnormal platelet monoamine oxidase activity that is associated with a wide range of psychiatric illnesses

(Yehuda, Edell, & Meyer, 1986). These data point to the possibility that the distinction of scoring high on these scales lacks specificity for indicating the schizophrenia diathesis.

To sum, individuals who score high on the PAS, MIS and the two scales combined do demonstrate high rates of psychotic-like experiences and schizophrenia-spectrum characteristics. However, the scales appear to also identify individuals at risk for a host of disorders that may or may not result in psychosis, while they do not appear to adequately predict which individuals will later present with the schizophrenia diagnosis. That high PAS, MIS, and PerMag scorers do present with schizophrenia-spectrum characteristics, though, perhaps indicates that the scales are somewhat sensitive to the characteristics of individuals who perhaps manifest only milder manifestations of schizophrenia-like characteristics. The following section will detail cross sectional and longitudinal research on the Revised Social Anhedonia Scale, which appears to have stronger predictive validity than the PAS or MIS.

The Revised Social Anhedonia Scale

Social anhedonia, or a schizoid form of social withdrawal that reflects indifference to social interactions with others, was referred to by Meehl in his earlier writings as a "quasi-pathognomonic sign" (Meehl, 1962, p. 892) of schizotypy. He later tempered this view (Meehl, 1990), however, subsequent research has proved the presence of social anhedonia to be one of the most promising core features of schizotypy (Kwapil, 1998).

In order to tap this feature of schizotypy, Chapman, Chapman, and Raulin (1976) developed the original 48-item Social Anhedonia Scale in order to measure feelings and behaviors that reflected a lack of pleasure in social interactions, as well as social anxiety.

This scale, however, proved not to be an effective predictor of psychotic-like experiences. Therefore, Eckblad, Chapman, Chapman, and Mishlove (1982) revised the Social Anhedonia Scale to include more items that measured schizoid withdrawal and indifference to others, while jettisoning the eight items that reflected social anxiety, surmising that such items may actually be tapping other personality characteristics not central to schizotypy (Kwapil, 1998). The new scale that emerged from this restructuring, the Revised Social Anhedonia Scale, or RSAS, has proved to have good psychometric properties, including a coefficient alpha of .79 for both males and females in one sample (Mishlove & Chapman, 1985) and good test-retest reliability (Blanchard, Mueser, & Bellack, 1998) in another. The scale proves to have good convergent validity with scales of attitudes towards others, and modest discriminant validity when compared with scales of pessimism and anxiety that do not have a social component (Leak, 1991). Elevations on the RSAS are also associated with poor social adjustment and a proclivity towards social withdrawal (Mishlove & Chapman, 1985), providing further evidence for the convergent validity of the scale.

Considerable research suggests that hedonic capacity, as measured by the RSAS, is a promising indicator of the liability for schizophrenia. Social anhedonia is an enduring individual difference factor in schizophrenia; higher levels of social anhedonia are associated with poorer social functioning and higher trait negative affect (Blanchard, Mueser, & Bellack, 1998). Compared to controls, individuals with schizophrenia report more schizoid withdrawal (Blanchard, Mueser, & Bellack, 1998). There is some controversy as to whether social anhedonia in schizophrenia is primary or secondary to other factors such as psychotic symptoms or medication side effects; however, social

anhedonia in individuals with schizophrenia has been shown in at least two studies to be independent of symptom status (Blanchard, Bellack, & Mueser, 1994; Blanchard, Horan, & Brown, 2001).

The specificity of social anhedonia to schizophrenia has also been called into question; social anhedonia does not always discriminate between individuals with schizophrenia and those with major depression (Blanchard, 1998). Social anhedonia associated with major depression, however, tends to ameliorate with the cessation of depressive symptoms, while it has been shown to remain a stable and enduring feature in schizophrenia over a one-year period (Blanchard, Horan, & Brown, 2001). Moreover, social anhedonia appears to be specific to disorders in the schizophrenia spectrum; individuals with schizophrenia and schizoaffective disorder display elevations in social anhedonia, but individuals with bipolar disorder do not manifest such elevations (Blanchard, Bellack, & Mueser, 1994). This suggests that social anhedonia in the schizophrenia spectrum is a trait, while as it is related to other disorders it is a state-related variable (Blanchard, 1998). To further differentiate social anhedonia as it is related to schizophrenia from that related to other disorders, social anhedonia is related to poor premorbid adjustment in first-episode schizophrenia, but is unrelated to premorbid adjustment in patients with psychotic affective disorders (Katsanis et al., 1992).

Converging lines of evidence support the notion that social anhedonia, as measured by the RSAS, can be used to identify individuals who are at risk for schizophrenia. The presence of social anhedonia appears to follow the same genetic pathways as schizophrenia; social anhedonia scores are elevated in the first degree relatives of schizophrenia patients (Katsanis et al., 1990; Katsanis et al., 1992), and

RSAS scores are significantly associated with the presence of schizophrenia-spectrum personality disorder characteristics in the relatives of schizophrenia patients as well (Lyons et al., 1995). RSAS scores were also associated with the presence of avoidant personality disorder in that sample, however, suggesting that the RSAS may tap the social sphere-related characteristics of that disorder. As further evidence for the validity of the social anhedonia construct in identifying individuals who may be at risk for schizophrenia, high RSAS scores are associated with schizoid and schizotypal personality disorder traits in inpatients with personality disorders (Bailey et al., 1993), as well as in a college sample (Mishlove & Chapman, 1985), in which individuals with elevated RSAS scores also reported more psychotic-like experiences than did control participants. Furthermore, high RSAS scorers have Minnesota Multiphasic Personality Inventory profiles similar to individuals who have schizophrenia spectrum disorders (Merritt, Balogh, & DeVinney, 1993). Social anhedonia may also be a potentiator of psychopathology in individuals who experience perceptual aberrations; males with elevations on both the RSAS and PAS have increased schizotypal personality disorder characteristics and psychotic-like experiences (Mishlove & Chapman, 1985), possibly owing to a compounding of neural abnormalities associated with the two types of elevations, as detailed in later sections.

Such cross-sectional evidence provides compelling reasons to believe that elevated scores on the RSAS may indicate the presence of the schizophrenia diathesis. Longitudinal studies echo these results and provide validation for the predictive power of the RSAS; in one particularly striking example (Kwapil, 1998), 24% of individuals who had elevated scores on the RSAS were diagnosed with schizophrenia-spectrum disorders

10 years after completing the measure, compared to only 1% of control participants. Moreover, individuals with high RSAS scores exceeded control participants in terms of psychotic-like experiences. Another 10-year follow-up study utilizing the Kwapil (1998) data found that social anhedonia was a significant predictor of schizotypal personality disorder dimensional scores and psychotic-like experiences among those with elevations on the MIS and/or PAS (Chapman et al., 1994). Those who score high on both the RSAS and MIS appear to be especially psychosis-prone; 21% of individuals with elevations on both scales met criteria for some form of clinical psychosis (Chapman et al., 1994). A replication of this study (Kwapil, Miller, Zinser, Chapman, & Chapman, 1997) using a combined MIS/RSAS elevation group confirmed these results.

Disentangling the independent effects of the MIS and RSAS elevations is important in evaluating the predictive validity of the scales. RSAS elevations appear to be a stronger predictor of, and have higher correlations with, schizophrenia-spectrum characteristics than are MIS elevations. Elevated RSAS scores are independently associated with higher schizotypal personality disorder dimensional ratings (Kwapil et al., 1997) as well as more schizoid and paranoid personality disorder features, while MIS scores are not (Blanchard & Brown, 1999).

To sum, the Revised Social Anhedonia Scale has proved to be a particularly promising indicator of schizotypy, both in cross-sectional correlational studies and longitudinal predictive studies. Both cross-sectional and longitudinal data provide evidence that elevated social anhedonia is associated with schizophrenia-related characteristics, and sometimes, the development of schizophrenia itself. RSAS elevations appear to be specific to schizophrenia-related characteristics, unlike the PAS

and MIS, and thus are likely to be a stronger indicator of the latent liability for schizophrenia than are elevations on the other two scales.

However strong the scales may be in identifying individuals with schizotypy-related personality characteristics, they do not come without their criticisms. Individuals putatively at risk for schizophrenia based on their Chapman scale scores in a ten-year longitudinal study did not, in fact, develop schizophrenia (Chapman et al., 1994). PAS/MIS scores were particularly weak predictors. In addition, while DSM-III-R criteria and the Chapman scales identified mostly the same individuals in one study (Thaker, Moran, Adami, & Cassady, 1993), scores on the scales were not associated with the reliable identification of the schizophrenia-spectrum disordered relatives of schizophrenia patients, suggesting that the scales are not sensitive to schizophrenia-related psychopathology in the relatives of schizophrenia patients.

Drawbacks to the psychometric method in general must also be considered. For instance, one must take into consideration the heterogeneous nature of the syndrome of schizophrenia. There may not be one unitary pathophysiology that can be indicated by the behavioral markers included in these scales. Related to this, individuals may present an array of markers, which can preclude reliably identifying the markers that actually indicate the schizophrenia diathesis. That is, some markers may be associated with general psychopathology, and may not be specifically associated with schizophrenia. Finally, schizophrenia proneness may not necessitate displaying measurable behavioral markers at all; individuals may be schizotaxic and thus vulnerable to developing schizophrenia but may evidence only normal-range behaviors (Grove, 1982).

## Do the PAS and RSAS Identify the Same Group of At-Risk Individuals?

Despite the wealth of literature that provides support for the construct validity of both the RSAS and PAS, there is considerable controversy as to whether the measures both tap the same a group of individuals carrying an unexpressed characteristic, that is, individuals who carry the liability for schizophrenia and schizophrenia-spectrum disorders. The cross-sectional data for both are promising (although this is more true for the RSAS), but the findings concerning the two scales are conflicting and uncertain. As outlined earlier, while elevations on the RSAS seem to be specific to schizophrenia-spectrum characteristics, elevations on the PAS can be indicative of more general psychopathology, from schizophrenia-related psychosis to affective psychosis to mood disorders. Social anhedonia is elevated in the relatives of individuals with schizophrenia (Katsanis et al., 1990), yet the relatives' PAS scores are similar to those of control participants (Franke et al., 1994). Still, individuals with high PAS scores report psychotic-like experiences. This contradictory evidence poses a conundrum: Do the RSAS and PAS both identify individuals who may be schizophrenia prone?

Some believe that the RSAS and PAS identify the same group because social anhedonia manifests secondary to PAS-related traits (Meehl, 2001). Social anhedonia has been found to form a taxon (a statistically-identified normal distribution of individuals who share a characteristic, found within the population distribution) with a base rate of approximately .10 (Blanchard, Gangestad, Brown, & Horan, 2000). Meehl's (1990) revised theory of schizotypy, however, refers not to anhedonia, or the complete absence of the experience of pleasure, but to hypohedonia, a non-taxonic normal range individual differences variable. According to Meehl (2001), hypohedonia can take two forms.

Primary hypohedonia is a latent endophenotypic characteristic which reflects a pleasure deficit as a result of genetic aberrations in the limbic system, while secondary hypohedonia is a manifest characteristic, which is the result of the experience of negative affect secondary to other traits or symptoms (Meehl, 2001). Meehl suggests that measurable social hypohedonia, rather than playing a primary role in the expression of schizotypy, is actually a non-taxonic mediating factor (Meehl, 2001) that is manifested as a result of aversive drift secondary to other symptoms of schizotypy, such as perceptual aberrations. Secondary hypohedonia *appears* taxonic, according to Meehl, because the schizotypy characteristics to which it is secondary form taxa themselves; thus, the characteristic of hypohedonia is "dragged" into the natural taxon as a function of aversive drift. For example, perceptual aberrations may form a taxon, and a lack of desire to participate in social interactions may be secondary to odd perceptual experiences during those perceptual aberrations; that anhedonia may then appear to form a taxon. In response to this, Horan and colleagues (2002) analyzed responses to the RSAS, MIS, and PAS and discovered that jointly, the three scales do not capture the same group; the taxon of individuals identified as socially anhedonic was independent of the constructs of the PAS and MIS. That the taxonicity of social anhedonia is not related to unexpressed factors associated with the PAS and MIS contradicts Meehl's notion that anhedonia is secondary to positive schizotypy traits and aversive drift.

However, in a schizophrenia-spectrum analysis (Suhr & Spitznagel, 2001), PAS and MIS scores fell into positive symptom-related and negative symptom-related factors, with the positive cluster including ideas of reference, magical ideation, and unusual

perceptual experiences, and the negative factor consisting of social anxiety, restricted affect, and a lack of close friends.

The RSAS, PAS, and MIS are all widely-used scales thought to contain fallible indicators of schizotypy. On the basis of extant research, it appears doubtful that all of these measures capture the same group of individuals at risk for schizophrenia-related disorders. Cross-sectional data provide support for the idea that the RSAS and PAS/MIS are useful tools for the identification of individuals at risk for developing schizophrenia, although longitudinal evidence suggests that individuals with elevations on the RSAS and PAS/MIS have different longitudinal outcomes. Moreover, clinical characteristics are rather distal from the pathogenic genotype, and for the purposes of schizophrenia prevention research, clinical phenotypic characteristics indicating the propensity for schizophrenia may be expressed too late for prophylactic measures to be taken. Thus, there is question as to how people who evidence schizotypy can be reliably identified, as well as uncertainty as to whether both the RSAS and PAS/MIS are tapping schizotypy. One domain that could be investigated is neuropsychology, as neuropsychological characteristics are closer to the endophenotype (a set of biological characteristics proximal to the genotype) than are clinical characteristics.

Neuropsychology appears to be a promising domain in this line of research, as it represents a group of endophenotypic markers that are presumably more proximal to genetic causes than are clinical markers and is less prone to error. Moreover, Meehl proposed that schizotaxia is reflective of a central nervous system abnormality, the presence of which could be assessed indirectly via neurocognitive assessment. A broader assessment of neuropsychological functioning in these two groups is indicated. The

purpose of this study is to examine the neurocognitive profiles of the two groups (people with RSAS elevations and people with PAS/MIS elevations) and to determine on the basis of those results which scale is most likely the most valid indicator of schizotypy, or if both are representative of the group of individuals who carry that latent liability for schizophrenia.

## Neurocognitive Assessment: An Endophenotypic Approach to Psychosis-Proneness Classification

One way to further this line of inquiry is to examine the endophenotypic (neurophysiological) characteristics of individuals in the Social Anhedonia and Perceptual Aberrations groups. The endophenotype is presumably closer to the pathogenic genotype than is the clinical phenotype. In addition, symptoms that arise from pathology in brain mechanisms may be more sensitive to genetic variations (Michie et al., 2000). Assuming that such characteristics reflect the integrated neural defect that is schizotaxia, one would expect that individuals who carried the liability for schizophrenia would evidence endophenotypic features similar to individuals with manifest schizophrenia. Following from this, if high RSAS and PAS/MIS scorers identify the same group, then both groups should evince similar neurocognitive profiles. Currently, there is a dearth of direct neurocognitive comparisons of the two groups. First, it is necessary to better understand how neurocognitive data can provide validity for the use of the Chapman scales in predicting the pathogenesis of schizophrenia. Thus, the literature as it relates to the neurocognitive profile of schizophrenia itself will be discussed. Then, the current evidence regarding the neurocognitive profiles of psychometrically-identified schizotypes will be presented. This will make it clear that

there is a need for studies which directly compare such profiles of individuals with RSAS and PAS elevations. This will help to determine which group actually shares the endophenotypic aberrations of schizophrenia, regardless of clinical presentation.

Since schizophrenia is primarily a neurocognitive disorder (Goldman-Rakic, 1997), one's neurocognitive profile can represent a latent trait marker in high-risk research. Neurocognitive deficits may appear without the presentation of clinical symptoms. Such a profile can represent a more reliable and valid marker than the behavioral and clinical characteristics that are often examined, as it is closer to its genetic or biological etiology than are clinical symptoms (Tsuang et al. 2001). Tsuang and colleagues (2001) point out that "clinical symptoms often reflect a remote consequence of the events that gave rise to them. . .there are numerous levels of biological and environmental modulation that can weaken the connection between genetic determinants and behavioral outcome" (p. 520). That is, one may not be able to reliably discern from where symptoms arise, as there are several "layers" of influencing factors that may result in heterogeneous clinical outcomes. Furthermore, there is often a temporal disjunction between the onset of neuropathology and its expression; neurocognitive markers can help in the early identification of those who are at risk for schizophrenia and related disorders while clinical symptoms may not yet be expressed (Walker, Diforio, & Baum, 1999) or reliably assessed (Michie et al., 2000). This approach can be a strong one; neurocognitive symptoms tend to remain stable (Faraone et al., 1999), while clinical symptoms may fluctuate. With regard to schizotaxia, Faraone, Green, Seidman, & Tsuang (2001) call for a neurocognitive approach for the identification of schizotypes, believing that schizotaxia is not adequately represented by only clinical signs. In an

illustration of this approach, Sponheim and colleagues (2001) examined the utility of using biological and neurocognitive indices to identify the relatives of individuals with schizophrenia, and found that the biological deviance of the relatives varied as a function of the probands' biological deviance. For instance, those probands with oculomotor dysfunction had relatives with oculomotor dysfunction. Importantly, there was better classification sensitivity and specificity when individuals were grouped by biological deviance cluster than when grouped by proband DSM diagnosis.

Biobehavioral functions are also important to consider because of their relation to everyday global functioning. Better social functioning in schizophrenia patients is associated with an increased resting arousal, lower stress reactivity, and an increased response to orienting stimuli (Brekke et al., 1997). In addition, better work functioning in this population is linked with good visuospatial processing, while the ability for independent living is associated with visuomotor and verbal processing skills. Moreover, awareness, empathy, self-monitoring, and the ongoing awareness of social circumstances are all associated with intact frontal lobe activity (Gualtieri, 1995). Cognitive deficits can also influence symptom presentation. In schizotypal personality disorder, impairments in cognitive and perceptual organization are associated with social isolation, guardedness, and detachment, likely due to an altered environmental representation and interpretation (Trestman, et al., 1995). Furthermore, neurometabolic activity, particularly hypodopaminergic (attenuated dopamine) activity in the ventral tegmental area and frontal lobes, is speculated to be associated with the symptom presentation of social anhedonia (Blanchard, 1998).

Schizophrenia as a Disorder of Neural Origin

Consistent with the notion that schizophrenia is a disorder of genetic origin, myriad abnormalities of brain structure and function exist in schizophrenia, for which there is a host of empirical evidence. In light of evidence revealing neurocognitive and neurological deficits and abnormalities in schizophrenia, it is now considered proximally to be a disorder of neurodevelopmental origin (influenced, of course, by genetic and other factors). Neurocognitive abnormalities are present early on in schizophrenia, are present in multiple cognitive domains, and predate the onset of the illness (Davidson, Reichenberg, Rabinowitz, Weiser, Kaplan, and Mark, 1999). These deficits also found in the non-ill relatives of schizophrenia patients (e.g., Erlenmeyer-Kimling et al., 2000)

Neuropsychological testing has been a rich source of information about possible neural dysfunction in schizophrenia. Although it is a somewhat more indirect method for assessing such dysfunction than imaging studies, neuropsychological testing nonetheless provides information about possible brain regions implicated in the disorder. Moreover, such data may allow us to speculate how brain dysfunction is manifested in the real-life experiences of individuals with schizophrenia and the environmental and social consequences of neural dysfunction.

Bryson and colleagues (2001) provide comment on how brain dysfunction may lead to difficulties processing social exchanges, theorizing that "a failure in understanding and organizing information from the external world may lead patients to reduce or abandon attempts to make interpersonal connections and engage in activities" (p. 35). Indeed, neurocognitive abnormalities associated with deficit schizophrenia in

one study (Buchanan et al., 1990), including sensory integration and short term memory problems, are associated with withdrawal and blunted affect.

The neurocognitive approach to determining schizophrenia risk status is a promising, though imperfect, method. In an investigation conducted by Davidson, Reichenberg, Rabinowitz, Weiser, Kaplan, and Mark (1999), healthy males who were later hospitalized for schizophrenia had lower scores on tests of arithmetic, verbal abstraction, nonverbal abstract reasoning, and verbal comprehension than did males who did not develop schizophrenia. In fact, one of the best predictors of the development of schizophrenia appears to be poor intellectual functioning (Davidson et al., 1999).

While it has its strengths, the neurocognitive approach is associated with false positive rates. Although Gottesman and Erlenmeyer-Kimling (2001) showed that the offspring of patients with schizophrenia had impairments in attention, short term verbal memory, and gross motor skills, among those identified to be at risk for developing schizophrenia, such impairments failed to predict the development of the disorder in the offspring at the rate of 18% (attention), 28% (verbal memory), and 27% (motor skills). Thus, although an endophenotypic/neurocognitive approach to the prediction of schizophrenia has significant strengths (such as being closer to the genetic influences on schizophrenia), neurocognitive abnormalities appear to be a fallible indicator, and will not always predict schizophrenia-related pathology.

Schizophrenia is associated with a wide range of neurological abnormalities. These abnormalities and deficits are pervasive, appearing in myriad brain regions and influencing performance on neurocognitive tasks. Post-mortem studies of schizophrenia patients have revealed subtle cellular anomalies, an imbalance in afferent neuron

connections, cortical degeneration, and neuronal loss (Goldman-Rakic & Selemon, 1997), while positron emission tomography studies in living patients have unveiled hypofrontality (e.g., Andreasen et al., 1992, Tamminga et al., 1992). These structural and metabolic abnormalities possibly give rise to functional deficits. Although some investigators have attempted to localize neurocognitive dysfunction in schizophrenia, the pattern of deficits appears to be generalized, encompassing domains as distinct as motor, sensory, and perceptual functioning, memory (Blanchard & Neale, 1994), executive functioning (Randolph et al., 1993), verbal fluency (Gruzelier et al., 1988; Hoff et al., 1992), and strategic planning (Andreasen et al., 1992). The pattern of dysfunction in the relatives of schizophrenia patients is just as generalized; they have co-occuring deficits in abstraction, verbal memory, and auditory attention, whereas control participants' performances on the different tasks do not correlate with one another, suggesting that this pattern of generalized deficits is unique to individuals putatively at risk for schizophrenia (Toomey et al., 1998).

Given the heterogeneity of schizophrenia, it follows that individuals with different types of symptoms may have different neurocognitive profiles. Basso and colleagues (1998) found that those with a negative cluster of symptoms (anhedonia, alogia, and affective flattening) show deficits in executive functioning, sustained attention, and sensory motor functioning, while the thought disorder and bizarre behaviors of the disorganized subtype are associated with attention span and sensory motor functioning. Surprisingly, hallucinations and delusions do not appear to be associated with any particular neurocognitive deficit.

Relatives of schizophrenia patients and individuals with schizophrenia-spectrum personality characteristics also demonstrate neurocognitive anomalies. In relatives of schizophrenia patients, executive functioning, visual and verbal memory, and auditory attention are risk indicators for the schizophrenia genotype, deficits that remained stable over four years (Faraone et al., 1999). Eye tracking dysfunction, allusive thinking, and soft neurologic signs are found in the adult relatives of schizophrenia patients as well (Faraone et al., 2001), and poor short-term memory, impaired attentional vigilance, and communication deviance are observed in both child and adult relatives (Faraone, et al., 2001). The relatives of schizophrenia patients perform worse than do those of affective psychosis patients on measures of reading and verbal fluency (Gilvarry et al., 2001). Although relatives in both groups had high levels of schizophrenia-spectrum traits, the neurocognitive deficits were specific to the relatives of schizophrenia patients. Even though scores on neurocognitive tasks are multidetermined, these results support the idea that "neuropsychological dysfunction among relatives of patients with schizophrenia is a stable trait caused by one or more genes that also increase the predisposition to schizophrenia" (Faraone et al., 1999, p. 179).

The spectrum traits themselves are associated with neurocognitive dysfunction and neuroanatomical anomalies. Paranoid traits are negatively correlated with reading performance, schizoid traits are negatively correlated with flexible attention, and schizotypal traits are negatively associated with verbal fluency (Gilvarry et al., 2001). In individuals with schizotypal personality disorder, the posterior of the corpus callosum is smaller than that of controls, but larger than those of schizophrenia patients (Downhill et al., 2000). This is consistent with a hypothesis of decreased hemispheric connectivity in

schizophrenia-related disorders; if activity is not perceived by both hemispheres of the brain, it may be perceived by the individual as not part of the self, and could be associated with a misanalysis of causal roles (Downhill et al., 2000). Schizotypal personality disorder is also associated with diminished P300 amplitudes, indicative of a lack of frontal lobe inhibitory capacity (Keefe et al., 1997) similar to that found in schizophrenia.

When considering the results of neurocognitive investigations, it is important to consider some limitations. Neuropsychological testing may more reflect differential performance on tasks of varying difficulty as compared to actual abilities, as outlined by Chapman and Chapman (1978). That is, although schizophrenia patients may perform worse than controls on tests of executive functioning as compared to tests of attention, it could be that the executive functioning measures are simply more difficult than the attention measures.

Frontal Lobe Dysfunction in Schizophrenia and Related Disorders: Attention and Working Memory

In general, although neurocognitive deficits in schizophrenia appear to be pervasive and generalized, there is particularly strong evidence for the involvement of prefrontal deficits in schizophrenia. Similarities in the performance and characteristics of schizophrenia patients and frontal lobe-damaged patients abound (Goldman-Rakic & Selemon, 1997), and many studies point to a pathological change in the dorsolateral prefrontal region in schizophrenia (Goldman-Rakic, 1996). In fact, there is diminished metabolic activity in the dorsolateral prefrontal cortex (DLPC) in schizophrenia patients,

as evidenced by the functional magnetic resonance imaging technique (Goldman-Rakic, 1996; Perlstein, Carter, Noll, & Cohen, 2001).

Studies of eye-tracking and executive functioning have revealed evidence which suggests apparent hypofrontality in schizophrenia. These domains will not be examined in the proposed study, but they deserve to be mentioned here. Abnormalities in smooth pursuit eye movement (SPEM) and other measures of eye tracking, including saccadic suppression, have been found in individuals with schizophrenia, schizophrenia spectrum personality disorders, and in relatives of schizophrenic patients (Kinney et al., 1998). Abnormal functioning in this domain is associated with disrupted functioning and decreased metabolism in the frontal cortex (Ross et al., 1997), particularly the DLPC (Evdokimidis et al., 1996), reflecting the diminished capacity of the frontal lobes to inhibit reflexive responses. With regard to executive functioning, several studies have concluded that there is no specific executive functioning deficit in schizophrenia (e.g., Goldberg et al., 1990). Other investigations have found the opposite pattern of results (Scarone et al., 1993; Goldman-Rakic, 1996; Laurent et al., 2000), particularly with regard to the "categories completed" index, which is sensitive to prefrontal functioning (Laurent et al., 2000). The relatives of schizophrenia patients also exhibit executive functioning deficits (Franke et al., 1992). Examinations of working memory and attention, which will be focused on in depth here, have also pointed to dysfunction in the frontal lobes.

Attention

Attention is a neurocognitive domain that has received particular focus in the literature as it relates to schizophrenia. Attentional deficits have been shown to be a promising predictor of schizophrenia (Gottesman & Erlenmeyer-Kimling, 2001); a significant number of investigations have found sustained attention deficits in schizophrenia, relatives of schizophrenia patients, and psychometrically identified schizotypes, although fewer studies have focused on the latter. Attention is a prefrontal function (Maier et al., 1992); the frontal lobes are associated with attention as the endpoint of an axis that includes the thalamus and reticular activating system (Gualtieri, 1995). Emphasizing the connection between the frontal lobes and schizophrenia, both frontal lobe and schizophrenia patients are impaired on measures of sustained attention (Buchsbaum et al., 1990).

Myriad studies have documented the occurrence of impaired sustained attention in schizophrenia patients as compared to control participants (e.g., Braff, 1993; Cornblatt & Keilp, 1994; Roitman et al., 1997). A low hit rate and a high rate of false alarms on various versions of the continuous performance task characterizes the sustained attention profile of the schizophrenia patient (Michie et al., 2000). Such a deficit appears to be a stable trait marker rather than a state characteristic; it occurs independent of the expression of clinical symptoms (Cornblatt, Lenzenweger, & Erlenmeyer-Kimling, 1989; Michie et al., 2000) or the decline of other indices such as IQ (Weickert et al., 2000). This deficit also appears independent of the chronicity or severity of symptoms (Orzack & Kornetsky, 1971). Neuroleptic treatment shows promise in ameliorating the attention deficit; a sample of neuroleptic-medicated schizophrenia patients performed equally as

well as control participants in one study (Jones, Cardno, Sanders, Owen, & Williams, 2001), although these results could be related to the negative symptom profile of the sample. A meta-analysis (Niuewenstein, Aleman, & deHaan, 2001) and an examination of patients with the deficit syndrome of schizophrenia (chararacterized by primary, chronic negative symptoms; Buchanan, Strauss, Breier, Kirkpatrick, & Carpenter, 1997) both show that negative symptoms are associated with particularly pronounced sustained attention deficits.

As in other neurocognitive domains, the biological relatives of schizophrenia patients evince deficits in sustained attention as measured by d', or sensitivity to the presence of a target (Maier et al., 1992; Keefe et al., 1997; Laurent et al., 1999; Erlenmeyer-Kimling et al., 2000), although they tend to perform better on tests of sustained attention than do schizophrenia patients themselves (Chen et al., 1998; Laurent et al., 1999). Errors of omission are associated with positive schizotypal personality disorder symptoms both in the relatives of schizophrenia probands and in control participants (Keefe et al., 1997).

What is the sustained attention profile of other individuals putatively at risk for schizophrenia? A small amount of research advances the idea that sustained attention deficits are a vulnerability marker for schizophrenia, as the picture of sustained attention in this group largely matches that of schizophrenia patients. Individuals with schizotypal personality disorder perform worse than do control participants and individuals with other non-schizophrenia-spectrum personality disorders on the continuous performance task-identical pairs version (Roitman et al., 1997). Interestingly, the performance of the non-schizophrenia-spectrum personality disordered individuals did not differ from that of

the control participants, lending credence to the notion that sustained attention impairments have specificity for the schizophrenia spectrum.

Deficits in sustained attention are not specific to the schizophrenia diagnosis. Such deficits have also been observed in individuals with bipolar disorder (Clark, Iverson, & Goodwin, 2002; Ferrier & Thompson, 2002) and other affective disorders (Cornblatt, Lenzenweger, & Erlenmeyer-Kimling, 1989). Upon closer examination, however, it appears that the nature of sustained attention deficit in affective patients is different from that of schizophrenia patients; while affective patients display a high rate of false alarms, the performance of schizophrenia patients is characterized by a low hit rate coupled with a high rate of false alarms (Cornblatt, Lenzenweger, & Erlenmeyer-Kimling, 1989). Overall, then, the literature suggests that sustained attention, while associated with schizophrenia related disorders, is not a definitive endophenotypic marker but is instead a fallible indicator of schizophrenia liability.

Individuals with schizotypal personality disorder symptoms demonstrate deficiencies in focused attention (Moritz, Andreasen, Naber, Krausz, & Probstein, 1999). Studies of event-related potentials (ERPs), one measure of early attentional processing, show that schizophrenia patients evidence latencies in the presentation of several ERP indices (Strandburg et al.,1994), as do the relatives of schizophrenia patients (Faraone et al., 1995), suggesting that there is a deficiency in early attentional allocation in individuals who carry the liability for schizophrenia. Given that ERPs are a stable trait marker (Nuchpongsai et al., 1999) this area of inquiry shows promise in schizophrenia proneness research. Related to this, individuals with schizotypal personality disorder symptoms show an attenuation of latent inhibition, which is the ability to suppress

attentional allocation to a previously encountered stimulus. This process could account for the occurrence of P300 and P50 ERPs encountered in individuals with schizophrenia and related disorders.

Schizophrenia patients display a wide range of attentional difficulties which appear without regard to the presentation of clinical symptoms, and are characterized by a lack of sensitivity to the presence of a target, as well as a high rate of false alarms. These difficulties could be related to symptom manifestations (e.g., social awkwardness) and predict functional outcomes (Michie et al., 2000). In addition to attention deficits serving as a potential valuable indicator of schizophrenia liability, working memory is another domain that could be promising in that respect.

Working Memory

Another neurocognitive domain that has received considerable attention in the literature is that of working memory. Working memory is a system for temporarily maintaining and manipulating information during the performance of a range of cognitive tasks, including comprehension, learning, and reasoning (Baddeley, 1986). Considerable research has supported the idea that the working memory comprises three domains: the central executive, which is an attentional control system; the phonological loop, which holds auditory information via rehearsal; and the visuospatial sketchpad, which maintains and manipulates visuospatial images (Baddeley, 1986). These three areas provide a computational area for holding items of information "on-line" as they are recalled, manipulated, and associated with ideas and incoming information (Goldman-Rakic & Selemon, 1997). Working memory is a particularly important construct in the domain of neurocognitive testing; most neuropsychological tests require working memory if

performance is not dependent on information not immediately present in the environment at the time of response and if it requires the updating of information on a moment-to-moment basis (Goldman-Rakic, 1987), such as when registering and remembering feedback on the Wisconsin Card Sorting Test.

The working memory is an index of prefrontal functioning; the maintenance of contextual information allows for the inhibition of dominant response tendencies, such as impulsive responses (Goldman-Rakic, 1987). As inhibition is a property of the prefrontal cortex, it is also a function of the working memory. In particular, working memory is associated with the dorsolateral prefrontal cortex (DLPC). Deficits in verbal, visual, and spatial working memory are all correlated with volume loss in the DLPC (Goldman-Rakic & Selemon, 1997), and studies of non-human primates show that lesions in this area affect working memory performance on a variety of tasks, including oculomotor delayed response tasks (Goldman-Rakic, 1987). Similarly, in humans, neurocognitive (Baddeley, 1998), lesion, and functional magnetic resonance imaging studies (Braver et al., 1997; Cohen et al., 1997) have implicated the DLPC as well.

Growing evidence supports the hypothesis that the behavioral disruption observed in schizophrenia arises from deficits in the working memory (Goldman-Rakic, 1991). Negative symptoms are associated with working memory failures in the DLPC, and working memory dysfunction may be a fundamental deficit underlying the cognitive features of schizophrenia. Goldman-Rakic & Selmon (1997) hold that "the disorganized thought process in schizophrenia patients that manifests itself in idiosyncratic content may be reducible to an impairment of neural mechanisms by which symbolic representations are both retrieved from the long-term memory and held in mind to guide

behavior" (p. 437-438). This assertion is supported by research that demonstrates that working memory impairments interact with interpersonal criticism to predict the emergence of psychotic thinking (Rosenfarb et el., 2000), showing that the behavior guidance properties of working memory are impaired in schizophrenia.

Given the impaired performance of schizophrenia patients on a range of working memory tasks, it is assumed that schizophrenia patients have functional and/or structural deficits in the DLPC. Following from this, it is likely that putative schizotypes should show deficits in this area as well. From the standpoint of psychosis prediction, working memory can be viewed as a particularly reliable endophenotypic marker; there is a significant and stable association between schizophrenia and memory impairment independent of age, medication, illness duration, severity of psychopathology, or positive symptoms, suggesting that such impairment is a trait rather than a state marker (Alemon et al., 1999; Kurtz et al., 2001).

Considerable evidence shows that individuals with schizophrenia have impaired working memory on a variety of tasks. Spatial working memory is one area in which schizophrenia patients show deficits; Carter and colleagues (1996) found spatial working memory deficits in a group of schizophrenia patients, and Park and Holzman (1992) determined that schizophrenia is associated with spatial working memory impairments. Another study (Park & Holzman, 1995) employing similar methods confirmed these results.

Although deficits were found in spatial working memory, Park & Holzman (1992) did not find auditory working memory deficits in their sample. They concluded on the basis of these results that the working memory deficits of schizophrenia are domain-

specific, that is, related only to visuospatial processes rather than verbal or auditory ones. However, other research has supported the view that these deficits are pervasive and not limited to one domain. Gold, Randolph, Carpenter, Goldberg, & Weinberger (1997) found that schizophrenia patients do exhibit impaired auditory working memory, utilizing a letter number sequencing task that required the storage, manipulation, and recall of a series of letters and numbers presented aurally. In a study examining each of the domains of working memory, Perry and colleagues (2001) provide more evidence that the working memory deficit in schizophrenia is pervasive; schizophrenia patients performed worse than a standardization sample on tests of auditory and visuospatial working memory. Moreover, in the schizophrenia group, performance on these measures was correlated, providing evidence for a pervasive deficit.

Unlike other neurocognitive deficits, working memory deficits appear to be specific to the schizophrenia diagnosis. Schizophrenia patients, but not bipolar patients, exhibit impaired performance on an oculomotor delayed response task (a task in which individuals must respond motorically after manipulating and/or holding visual information in the visuospatial sketchpad of working memory; Park & Holzman, 1992); in fact, bipolar patients did not show impaired performance on any spatial working memory task. Although they did not exhibit significantly different performance from each other, bipolar patients did not perform worse than controls on a spatial working memory task while schizophrenia patients did (Gooding & Tallent, 2001), suggesting incrementally worse performance for the schizophrenia group. Moreover, working memory makes a unique contribution to antisaccade task performance in schizophrenia patients, while it does not in bipolar patients (Gooding & Tallent, 2001). Even siblings of

schizophrenia patients can be discriminated from siblings of bipolar patients on the basis of visuospatial working memory (Keri, Kelemen, Benedek, & Janks, 2001). Providing even more convincing evidence for the specificity of working memory deficits as an endophenotypic marker in schizophrenia, working memory dysfunction appears to be a state marker of abnormality in bipolar disorder rather than a trait indicator; in that disorder, working memory performance is inversely associated with the number of manic episodes experienced (Cavanagh et al., 2002). Conversely, in schizophrenia, working memory abnormalities exist independent of clinical state.

Working memory deficits in the unaffected relatives of schizophrenia patients mimic those found in the patients themselves. As in schizophrenia patients, working memory deficits in the relatives of schizophrenia patients exist across a number of domains. Conklin and colleagues (2000) determined that relatives of schizophrenia patients exhibit deficits on measures of verbal working memory similar to that of schizophrenia patients, and Park and colleagues (1995) found that relatives of schizophrenia patients display deficits on the oculomotor delayed response task, a measure of visuospatial working memory. Deficits in spatial working memory were also found by Park, Holzman, and Goldman-Rakic (1995). Although the literature in this area is sparse, it is apparent that a genetic predisposition to schizophrenia renders one susceptible to working memory impairment, providing support for the idea that working memory is an endophenotypic marker that can be of use in the prediction of psychosis.

Providing further support for this assertion, working memory impairment is also apparent in schizotypal personality disorder. Working memory deficits may be a trait characteristic in this realm as well as in schizophrenia (Gooding & Tallent, 2002).

Individuals with schizotypal personality disorder display working memory impairments on a variety of tasks, including the backward masking continuous performance task, a measure of visual working memory (Farmer et al., 2000; Ratey, 1995). Also, those with schizotypal personality disorder display significantly lower accuracy than controls on a delayed response spatial working memory task (Park et al., 1995), as do those with schizotypal characteristics (Park & McTigue, 1992). These deficits appear to be specific to schizotypal personality disorder and are not related to the non-odd personality disorders (Keefe et al., 1997). Raine, Benishay, Lencz, & Scarpa (1997) claim to find evidence for working memory dysfunction in their skin conductance orienting paradigm; individuals with schizotypal traits failed to habituate to an auditory stimulus, which may indicate a deficit in preattentive template matching. In preattentive template matching, one stimulus is held in the working memory while the other is presented, the two are then compared, and then if matched, one will respond with normal skin conductance orienting. That the schizotypes in this sample failed to do so may indicate a failure in template matching at the level of holding the first stimulus in the working memory.

Working memory deficits in schizophrenia and in individuals putatively at risk for schizophrenia are pervasive, encompassing visual, verbal, and auditory domains. Overall, it appears that although working memory deficits appear to be specific to schizophrenia, their relation to individuals at risk for schizophrenia remains unclear.

It is apparent from this discussion that neurocognitive deficits are pervasive in schizophrenia and schizophrenia-spectrum disorders and predate the expression of the illness itself. It follows, then, that individuals putatively at risk for schizophrenia would likely evidence some of these genetically-based neurocognitive deficits themselves.

Related to this, it is likely that the same genetic factors that give rise to neurocognitive deficits also give rise to the personality characteristics seen in individuals at risk for schizophrenia, or that the neurocognitive deficits engender environmental experiences that foster the development of those personality characteristics, such as those described by Meehl (1962) in his theory of schizotypy. Identifying individuals with schizotypic personality characteristics should result in the identification of a group with specific neurocognitive abnormalities, who thus are likely to be prone to developing schizophrenia. How can such individuals be identified? One promising method is to examine the neurocognitive characteristics of individuals identified by the psychometric detection paradigm. This method of jointly examining both neurocognitive and personality characteristics should result in information about the validity of the psychometric detection paradigm, particularly with regard to which of the Chapman scales is likely to be a more valid indicator of schizotypy. With the preceding review of neurocognitive deficits in schizophrenia in mind, this introduction will now examine the neurocognitive profile of individuals identified by the Chapman scales. The extant literature is somewhat sparse, but provides a starting point for the current study, which examined the neurocognitive characteristics of individuals with elevations on the RSAS versus those with elevations on the PAS.

## Neurocognitive Deficits in Putative Schizotypes

A small literature has examined the neurocognitive correlates of psychometrically-designated schizotypy. Socially anhedonic individuals display right hemisphere underactivation on chimeric emotion face tasks while PerMags and controls do not (Luh & Gooding, 1999), consistent with hypotheses that social withdrawal

behaviors are associated with right hemisphere underactivation. On the contrary, PAS/MIS elevations are associated with left hemisphere overactivation (Overby, 1992; Luh & Gooding, 1999). PerMags are markedly sensitive to the affective components of words, while socially anhedonic individuals tend to process affective information in an affectively shallow manner, as evidenced by semantic priming tasks (Kerns & Berenbaum, 2000). Moreover, electroencephalography shows that PerMags demonstrate sustained negativity of the O wave in response to stimuli. This demonstrates that PerMag individuals continue to process stimuli long after presentation, a proclivity which could result in misinterpretation of those stimuli. It should be noted that Chapman scale scores do not appear to be correlated with neurocognitive measures in relatives of schizophrenia patients (Laurent et al., 2000), but this study did not examine whether individuals with scale elevations performed more poorly on the tasks than relatives without elevations.

Sustained Attention

Although there is plenty of research which has identified sustained attention deficits in schizophrenia, other research supports the idea that sustained attention is a marker of general pathology and is not specific to schizophrenia related disorders. Using individuals with poor continuous performance task scores as index subjects, Obiols and colleagues (1999) compared this group with a group of control participants on the Chapman scales. No differences emerged between the groups with regard to the RSAS or PAS, suggesting that individuals with a propensity for other disorders were included in the index group as well. This sample, however, consisted of adolescents, who may not yet have manifested measurable schizotypic traits.

The sustained attention literature as it relates to psychometrically identified schizotypy is mixed. To date, only one study has examined the sustained attention of individuals with RSAS elevations; that study found sustained attention deficits in that group (Dickerson, Diaz, & Kwapil, 2002). With regard to PAS elevations, Lenzenweger, Cornblatt, & Putnick (1991) found that those with PAS elevations perform significantly worse than do controls. These results are confirmed by those of Lenzenweger (1998), who found that PAS-elevators have fewer correct hits than do controls. These data are in line with prior investigations of schizophrenia patients which show a significantly lower hit rate than that of controls (e.g., Cornblatt et al., 1989). However, these data diverge from previous studies of schizophrenia patients, as the performance of PAS elevators is not marked by a high false alarm rate. Another study of PAS elevators (Lenzenweger, 2001) met with negative results. The author attributed this to insufficient statistical power, but it is possible that the positive schizotypal signs represented by the PAS are not associated with sustained attention deficits. Recall that negative symptoms in schizophrenia are associated with pronounced attention deficits (Niuewenstein, Aleman, & deHaan, 2001); perhaps Lenzenweger's sample did not include individuals with negative schizotypy symptoms.

Although attentional difficulties do not appear to be specific to schizophrenia related characteristics, a low hit rate in measures of sustained attention distinguishes the attention deficits of putative schizotypes from those of other disorders. Such evidence provides the impetus for the current study to examine differences in sustained attention in individuals with elevations on the RSAS versus those with elevations on the PAS.

Studies of event-related potentials (ERPs), one measure of early attentional processing, show that as a group, psychometrically identified schizotypes evidence latencies in the presentation of several ERP indices (Nuchpongsai, Arakaki, Langaman, & Ogura, 1999). However, no studies to date have compared those with PAS/MIS elevations and those with RSAS elevations. This lacuna deserves to be rectified.

Working Memory

With regard to psychometrically identified schizotypes and their working memory characteristics, the literature is sparse and equivocal. Visuospatial working memory deficits are not always found in psychometrically defined schizotypes on object alternation tasks (Faraone et al., 2001), although two studies (Balogh & Merritt, 1985; Park, Holzman, & Lenzenweger, 1995) did find such deficits on a visual masking task (Balogh & Merritt, 1985). Lenzenweger & Gold (2001) failed to find auditory working memory deficits in a sample of individuals who were elevated on the PAS; they suggest that auditory working memory deficits may only surface after the presentation of clinical illness.

To date, there have been no studies which have examined the relation of working memory deficits to social anhedonia alone. Park & McTigue (1997) found that not having any close friends was related to spatial working memory, but a lack of close friends could be related to any number of proximal causes, including schizotypal or paranoid personality traits, which are associated with more positive-type traits. It is interesting, though, as pointed out by Park and McTigue (1997), that both working memory and social reward pathways are associated with the dorsolateral prefrontal

cortex; perhaps deficits in this area inhibit feelings of reward from social relations and are manifested in working memory abnormalities.

Only one study has simultaneously examined individuals with PAS/MIS elevations and individuals with elevations on the RSAS (Tallent & Gooding, 1999). This investigation found that individuals in both groups were worse than controls on a task of spatial working memory but were not different from each other. Although social anhedonics exhibited increased reaction times as compared to controls, they did not differ from people with PAS/MIS elevations on this index. Despite this lack of difference, Tallent & Gooding (1999) propose that working memory deficits in the two groups emerge as a function of disparate processes. That is, they propose that people with PAS/MIS elevations exhibit working memory difficulties secondary to deficits in attention, while individuals with social anhedonia display impaired working memory due to less efficient storage and maintenance of information, that is, an actual working memory deficit. In addition, given the non-specificity of the PAS for schizophrenia proneness and its relation to some forms of working memory deficits, it is unclear that working memory deficits are specific to those at risk for schizophrenia. These results may also have occurred because of the use of a combined PAS/MIS elevation group. Recall that MIS elevations in social anhedonics appear to potentiate the development of psychosis (Chapman et al., 1994); thus, it is possible that although scores on the PAS and MIS are highly correlated, the elevations interact in such a way that spurious patterns of results occur.

Other Neurocognitive Deficits in Psychometrically Identified Schizotypes

Though not the focus of the proposed study, psychometrically identified schizotypes also evidence deficits in other areas of neurocognitive functioning, which deserve to be mentioned here. This section will review evidence concerning eye movement dysfunction and executive functioning performance.

Similar to schizophrenia patients, both individuals with PAS/MIS elevations (Lenzenweger & Gold, 2000) and socially anhedonic individuals (Gooding, 1999) show aberrations in antisaccade performance. Two direct comparisons of individuals with PAS/MIS elevations and socially anhedonic individuals show that the two groups do not differ from each other in terms of SPEM (Gooding, Miller & Kwapil, 2000) or antisaccade performance (Gooding, 1999); both perform worse than controls. This is not necessarily evidence for a shared pathophysiology of dysfunction, however. Gooding (1999) conjectures that the performance of socially anhedonic individuals is aberrant because of frontal lobe deficits, as negative symptoms (of which social anhedonia is comprised) are associated with the frontal cortex. On the other hand, she hypothesizes that individuals with PAS/MIS elevations display aberrant performance due to attentional interference spurred on by psychotic-like symptoms.

That the two groups did not differ in terms of SPEM and antisaccade performance should not be construed as providing support for the assertion that the PAS and MIS tap true schizotypy. Taken together with the fact that bipolar disorder patients also exhibit eye movement dysfunction, these results support the hypothesis that such dysfunction is a marker for a liability to general psychopathology. Notably, three of the PerMag

participants in the Gooding, Miller, & Kwapil (2000) investigation met criteria for bipolar disorder.

Executive functioning is another area that has been explored in psychometrically identified schizotypes. Currently, there is a paucity of literature on executive functioning deficits in this population. The literature that does exist shows that individuals with PAS elevations have significant problems with maintaining set (Park et al., 1995; Lenzenweger & Korfine, 1994). Those PAS high-scorers with predominantly negative symptoms show deficits on all the indices of the WCST, while surprisingly, positive symptoms are associated with WCST performance that is significantly better than that of control participants (Suhr & Spitznagel, 2001). Socially anhedonic individuals also evidence WCST deficits as compared to controls (Gooding, Kwapil, & Tallent, 1999), but individuals with PAS/MIS elevations and socially anhedonic individuals do not differ from each other (Gooding, Kwapil, & Tallent, 1999; Gooding, Tallent, & Hiegyi, 2001) on the basis of WCST performance. Social anhedonia combined with cognitive slippage, however, renders one particularly susceptible to committing WCST perseverative errors (Gooding, Tallent, & Hegyi, 2001).

It is clear that although some studies have been done to examine the neurocognitive signatures of psychometrically-defined schizotypes, few studies have simultaneously examined those with PAS/MIS elevations (PerMag group) and those with RSAS elevations (SocAnh group). Such examinations are necessary to establish which scale is most likely to identify individuals at risk for schizophrenia. This work would have relevance for early identification and early intervention attempts. Given that neurocognitive endophenotypical markers are more proximal to genetic causes than

clinical characteristics, they are likely less prone to error. Such a strategy would therefore be useful in determining the differences between and the validity of the RSAS and PAS/MIS.

# CHAPTER TWO: THE CURRENT STUDY

## Overview

Meehl's (1962, 1990) theory of schizotaxia and schizotypy proposes that individuals who are at risk for developing schizophrenia and related disorders will evidence certain behavioral characteristics without necessarily crossing the line into frank psychosis. Thus, one would surmise that there is a group of individuals who evidence these characteristics, and that these characteristics are measurable by the use of paper-and-pencil questionnaires. At issue is if measures such as the Chapman scales can adequately identify individuals at risk for psychosis, and if so, which measure(s) most accurately identify this class. The PAS, MIS, and RSAS have been used extensively to identify individuals putatively at risk for psychosis, but it is unclear that they are tapping the same group of individuals. PAS and MIS elevations appear to be associated with a wide range of pathology, while one can more confidently associate RSAS elevations with disorders and characteristics of the schizophrenia spectrum, as evidenced by interview and longitudinal investigations.

One useful strategy to determine whether the PAS/MIS and RSAS are tapping the same group is to examine endophenotypic (i.e., neurophysiological) markers as represented by neurocognitive indices. Given that individuals at known genetic risk for schizophrenia evidence the same neurocognitive abnormalities as those with schizophrenia, although to a less marked degree, it follows that those designated as at risk for schizophrenia related disorders on the basis of psychometric elevations will evidence those same abnormalities. Should they not, it is possible that the scales do not measure

true schizotypy. A small amount of literature (as detailed in the previous chapter) suggests that individuals psychometrically designated as at-risk for schizophrenia do evidence the same neurocognitive abnormalities as schizophrenia patients. However, other studies, particularly those involving the PAS/MIS, have conclusions to the contrary. Thus, it is important to determine if the RSAS and PAS/MIS can be used jointly with endophenotypic indicators to confer risk status for schizophrenia. Very few studies have simultaneously compared PAS/MIS and RSAS elevators to see if one group evidences worse neurocognitive abnormalities than the other.

Besides this limitation of the literature, it deserves to be mentioned that most studies that have employed the Chapman scales have utilized college samples. Though convenient, college samples are not necessarily representative of the population as a whole. Individuals who attend college appear to be higher functioning than those who do not (Newman, Moffitt, Caspi, & Silva, 1998). Furthermore, while 100% of the studies that have examined neurocognitive correlates of the Chapman scales have used college samples, fully 76% of the United States population does not attain a Bachelor's degree (U.S. Census Bureau, 2002). It is likely that even a higher percentage of individuals with schizophrenia do not go on to college; most schizophrenic individuals only complete high school (Lewine, Haden, Caudle, & Shurett, 1997). In addition, a recent study (Fuller et al., 2002) revealed that individuals with schizophrenia show cognitive deficits as early as elementary school; these deficits became more pronounced as the individuals entered high school. Such deficits are inconsistent with college attendance. Thus, psychometrically-identified schizotypes who go on to enter college may be largely different, and probably higher functioning, than those who do not go to college. The

"college sophomore problem" seems to be a serious impediment in the prediction of schizophrenia related disorders. Thus, to truly harness the powers of the Chapman scales and neurocognitive indicators in the prediction of schizophrenia related disorders, it is necessary to examine a heterogeneous community sample.

This study sought to address these issues by simultaneously examining individuals with PAS/MIS and RSAS elevations from a community-based sample on the basis of neurocognitive indices. This was be achieved by examining a large community sample of 18 year olds. The RSAS and PAS/MIS were used to identify social anhedonia, perceptual aberrations and magical ideations, and control groups. A combined PAS/MIS elevation group was used because of evidence that the scales are highly correlated (r = .70; Gooding, 2000). Following subject selection based on self-report results, subjects were recruited to participate in a full diagnostic and neuropsychological battery. Group differences in neurocognitive performance and diagnostic status were examined among the groups. The current study was the first investigation to simultaneously examine individuals with PAS/MIS and RSAS elevations on neurocognitive indices in a community sample. In addition, this study employed a broader battery of neuropsychological functioning than has been used in any study to date, including measures of verbal and spatial working memory, verbal memory, and sustained attention.

## Hypotheses

This study examined the following hypotheses:

1) The neurocognitive performance of individuals with elevations on the RSAS (SocAnh group) will differ from that of individuals with elevations on the PAS/MIS (PerMag group). Specifically, the SocAnh group will perform worse than the PerMag group

on measures of general intellectual ability (Wechsler Adult Intelligence Scale-III [WAIS-III] Block Design and Vocabulary subtests) spatial working memory (Wechsler Memory Scale-III [WMS-III] Spatial Span), auditory working memory (WMS-III Letter-Number Sequencing), and sustained attention discriminability sensitivity (WMS-III Degraded Stimulus Continuous Performance Task, d'). Those in the PerMag group will perform better than those in the SocAnh group and equal to controls on these measures.

2) Diagnostic interviews will identify greater schizophrenia spectrum pathology in the SocAnh group than in those in the PerMag group. The latter will have less schizophrenia-spectrum pathology than social anhedonics, but more than will controls.

# CHAPTER THREE: METHODS

## Participants

Participants were recruited by the University of Maryland Survey Research Center. Using random digit dial methods, telephone numbers that were within a 15-mile radius of the university were incorporated into a database; those numbers were randomly dialed to recruit participants. A total of 3,494 18 year-olds in Washington D.C., Arlington County, VA, Prince George's County, MD, and Montgomery County, MD were identified. They were mailed a consent form, a screening questionnaire that included measures of social anhedonia, perceptual aberrations, and magical ideations, as well as five dollars as partial compensation for their participation. They were mailed an additional ten dollars upon receipt of their completed survey. Selection and recruitment of participants was independent of race, educational attainment, and socioeconomic status. This method yielded 1,483 complete questionnaires from individuals who consented to participate in the study.

Participants for the control, Social Anhedonia, and Perceptual Aberration/Magical Ideation groups were drawn from the initial screening participants who returned their surveys. Following from selection procedures used in previous studies (e.g. Chapman et al., 1994, Kwapil et al., 1998), subjects with extreme scores on the Revised Social Anhedonia Scale (RSAS) and the Perceptual Aberration/Magical Ideation Scales (PAS/MIS) were selected for the Social Anhedonia (SocAnh) and Perceptual Aberration/Magical Ideation (PerMag) groups. "Extreme" was defined as being at least 1.80 standard deviations above the mean of the entire sample. Groups were "pure," that is, participants who had extreme scores on the RSAS or PAS were not selected to be in

the SocAnh or PerMag groups if they also had extreme scores on the PAS/MIS or RSAS, respectively. Participants whose scores were lower than .5 SD above the mean were chosen as control participants. The sample consisted of 33 individuals with RSAS elevations, 28 individuals with PAS/MIS elevations, and 89 control participants.

With regard to group sample sizes, the 33 SocAnh individuals selected for the "pure" SocAnh group came from a larger sample that is being used in a larger study (of which the current data are a part). The larger SocAnh sample consisted of 87 individuals with RSAS elevations who were selected without regard for PAS/MIS elevations. Given that the current control group was matched to the SocAnh group in that larger study, the number of current control participants is higher than the number of SocAnh or PerMag participants. PerMag individuals were recruited solely for the current study and are not a part of a larger sample unselected for RSAS elevations.

Sample Demographic Characteristics

Race and sex data may be viewed in Table One. The three groups did not differ with regard to participant sex, $\chi^2$ [2] (N = 148) = .948, ns. There were significant differences among the groups with regard to participant race, $\chi^2$ [6] (N = 148) = 15.14, p = .019. There were significantly more Caucasian participants in the PAS/MIS group than in the other two groups, fewer African-Americans in the PAS/MIS group than in the other two groups, and fewer participants describing themselves as "Another Race" in the RSAS group.

Following the subject selection process described above (see also Instruments section), potential participants identified as being in the SocAnh, PerMag, and control groups were contacted by telephone, e-mail, or written letter and were invited to

participate in the proposed project. Each subject was informed of the study procedures, length (3-5 hours), and compensation ($100), and was instructed to refrain from the use of drugs and alcohol in the 24 hours prior to study participation.

## Informed Consent

All procedures were fully explained to the participants during informed consent procedures. They were debriefed regarding more specific aims of the study following their completion of the study procedures.

This proposal was submitted to the University of Maryland at College Park Institutional Review Board, and received approval. This study was part of a larger IRB-approved study funded by a grant from the National Institutes of Health.

## Instruments

### Instruments Used to Identify Participants

*Assessment of Social Anhedonia* The Revised Social Anhedonia Scale (RSAS) is a 40 item true/false inventory designed to tap schizoid asociality. Social anhedonia was assessed based on participants' responses to such items as "Just being with my friends can make me feel really good" (keyed false) and "Having close friends is not as important as many people say" (keyed true). The RSAS has been shown to be internally consistent (e.g., Blanchard, Mueser, & Bellack, 1998; Mishlove & Chapman, 1985) and has demonstrated high test-retest reliability over a 90-day period (Blanchard et al., 1998). Validation of the RSAS as a measure of social anhedonia comes from findings that high scores on this scale are related to interview-based reports of current social withdrawal and isolation (but not loneliness) and reports of less enjoyment from and need for social

contact (Mishlove & Chapman, 1985). Data supporting the validity of the RSAS as a measure of schizotypy come from studies showing elevated SocAnh in individuals with schizophrenia (Blanchard, Mueser, & Bellack, 1998) and their family members (Katsanis et al., 1990; Kendler et al., 1996), cross-sectional studies showing elevated schizophrenia-spectrum disorder dimensional scores in social anhedonics (Brown, Blanchard, & Horan, 1998), and longitudinal studies of the development of schizophrenia-spectrum disorders in social anhedonics (Kwapil, 1998).

*Assessment of Perceptual Aberrations/Magical Ideations* The Perceptual Aberration Scale (PAS) is a 35 item true-false questionnaire that measures transient aberrations in the perception of one's own body and other perceptual aberrations. Perceptual aberrations were assessed based on participants' responses to items such as "Occasionally it has seemed as if my body had taken on the appearance of another person's body" (keyed true) and "My hands and feet have never seemed far away" (keyed false). Individuals identified by this scale appear to be psychosis-prone (Chapman et al., 1994). The Magical Ideation Scale (MIS) is a 30 item true/false questionnaire that measures belief in forms of causation that are regarded as invalid and magical. Magical ideation was assessed based on participants' responses to items such as "I have sometimes had the momentary feeling that someone's place has been taken by a look-alike" (keyed true) and "I almost never dream about things before they happen" (keyed false). The PAS and MIS are highly correlated ($r = .70$) and are often combined to select a deviant group (Gooding, 2000).

*Screening of Random or Invalid Responding* During initial screening, the Infrequency scale (Chapman et al., 1976) was used to identify random or invalid

responding. The 17-item Infrequency scale includes items that almost everyone answers in one direction, for example, "I visited Easter Island last year." High scores (a criterion of three or more items endorsed) on the infrequency scale suggest invalid responding in general, thus, scores on this scale were used as an invalidity index for all measures. Subjects scoring high on this scale were excluded from the study.

Participants who were selected from the pool of initial screening participants and were invited to participate in the study took part in diagnostic interviews, and were tested on several neurocognitive measures.

Diagnostic Interviews

*Assessment of Axis I Disorders* Psychiatric diagnoses were evaluated using the Structured Clinical Interview for DSM-IV Axis I Disorders, Patient Edition-Research Edition (SCID; First et al., 1996), including the mood, psychotic, and substance use disorders sections. This is a widely used instrument in other studies of psychosis proneness (e.g., Asarnow, et al., 2001), and prior versions of the SCID have shown good inter-rater agreement, with kappas greater than .60 (Williams et al., 1992). All clinical interviews were conducted by doctoral students who have been SCID-trained by a licensed clinical psychologist (Dr. Jack Blanchard).

*Assessment of Schizophrenia Spectrum Disorders* The International Personality Disorder Examination (IPDE; Loranger, et al., 1995) is a semi-structured interview designed to assess personality disorders in both the DSM-IV and the International Classification of Diseases-10 (ICD-10) classification systems. The IPDE interview surveys behavior and life experiences relevant to the criteria and can be used to determine DSM-IV categorical diagnoses and dimensional scores of personality disorders. The

present study examined schizophrenia-spectrum characteristics, that is, the characteristics of schizoid, schizotypal, and paranoid personality disorders. The questions tapping characteristics of these disorders include items concerning unusual thinking or beliefs, unusual perceptual experiences, suspicions and paranoid ideation, inappropriate or constricted affect, odd/eccentric behavior or appearance, relationships with others, and social anxiety. Reports of interrater reliability in joint interviews have demonstrated an overall weighted kappa for individual personality disorders to be .57 for the DSM-III-R and .77 for the ICD-10. Interrater reliability was higher for dimensional scores with ICCs ranging from .79 to .94 for the DSM-III-R and .86 to .93 for the ICD-10. The IPDE has been successfully used in several studies of schizophrenia-spectrum disorders in putatively psychosis-prone subjects (e.g., Blanchard & Brown, 1999).

*Assessment of Functioning* The Global Assessment of Functioning scale (GAF; American Psychiatric Association, 1994) was used to measure participants' overall functioning. The GAF score provides a rating of overall adjustment ranging from marked psychopathology at the low end to superior functioning at the high end.

All diagnostic interviews were videotaped for supervision purposes.

Measures of Neurocognitive Functioning

When selecting measures to be used in this study, measures were chosen that looked promising with regard to identifying deficits that may be related to the liability for schizophrenia, as demonstrated by studies that have found such deficits in groups of individuals with expressed schizophrenia (see literature review). The domains assessed in this study tapped those relating to those already reviewed, including sustained

attention, working memory, other indices of memory, and general intellectual functioning.

*Assessment of Attention* Participants took part in the Degraded Stimulus Continuous Performance Task, which evaluates the ability to sustain attention over a period of time during a rapidly paced visual discrimination task. The DSCPT has identified deficits in attention in relatives of schizophrenics (Cannon et al., 1994; Cornblatt & Erlenmeyer-Kimling, 1985; Grove et al., 1991) and in symptomatic as well as stabilized schizophrenics (Nuechterlein, Dawson, Ventura, Fogelson, Gitlin, & Mintz, 1990; Nuechterlein, Edell, Norris, & Dawson, 1986). The DSCPT was administered on an IBM-compatible computer. It requires subjects to discriminate highly-blurred zeroes from other highly-blurred digits during an eight-minute period in which single digits are presented very briefly (40 milliseconds) at a rate of one per second. The index of discriminability sensitivity, d', was used in this study as a measure of sustained attention.

*Assessment of Working Memory* Three subtests from the Wechsler Memory Scales-III were used to assess working memory. These include Digit Span (Forward and Backward), Letter-Number Sequencing, and Spatial Span (Forward and Backward). These tests have all been found to assess working memory abilities (Wechsler, 1997).

Digit Span This is a task of auditory working memory. In the Digit Span Forward test, the examiner reads a series of numbers; the participant is then asked to repeat these numbers back to the examiner in the same order. Similarly, in the Digit Span Backwards test, a series of numbers are read and the participants are asked to repeat them back in the reverse order of which they were given. For both tests, the number of digits in each

sequence ranges from two to nine. The Digit Span tests have excellent internal reliability (.91 in the 18-19 year old age group; The Psychological Corporation, 1997).

Spatial Span This is a task of visuospatial working memory. In the Spatial Span Forward test, the examiner points to a series of blocks on a three-dimensional boards in a specified sequence. The participant must point to the same blocks in the same order. In the Spatial Span Backward test, the participant must point to the blocks in the reverse order of which the examiner presented them. Spatial Span also has good internal reliability (.83 in the 18-19 year old age group; The Psychological Corporation).

Letter-Number Sequencing This is a task of auditory working memory. The examiner reads a sequence of letters and numbers to the participant, who must then repeat the sequence with the numbers first, in ascending order, and then the letters in alphabetical order. The length of the string ranges from 2 to 8 items. The Letter-Number Sequencing task has good reliability (.88; The Psychological Corporation, 1997).

*Assessment of General Memory Functioning* Four subtests from the Wechsler Memory Scales-III were used to assess general memory. These include Logical Memory I and II and Visual Reproduction I and II. These tests have all been found to assess general memory abilities (Wechsler, 1997).

Logical Memory The Logical Memory I and II subtests of the WMS-III assess short and long-term memory, respectively. Participants are asked to recall two short stories immediately after an examiner reads them and then 25-35 minutes after they are read. Intervening tasks do not engage verbal memory in order to ensure the absence of a confound. After the delayed recall, subjects answer 15 yes/no recognition questions about the stories. Logical Memory I has good reliability (.86; The Psychological

Corporation, 1997), as does Logical Memory II (.73; The Psychological Corporation, 1997).

Visual Reproduction The Visual Reproduction I and II subtests of the WMS-III assess nonverbal short- and long-term visual memory, respectively. Participants are shown five designs and are asked to draw them from memory immediately following presentation of the stimulus. Twenty-five to 35 minutes later, they are asked to draw all the designs again from memory. Both subtests have shown excellent interrater reliability (.97, Wechsler, 1987).

*Assessment of General Intelligence*

The Vocabulary and Block Design subtests of the Wechsler Adult Intelligence Scales-III (WAIS-III) were used to assess participants' general cognitive abilities. Both scales have been shown to have good reliability and a high correlation with the Full Scale IQ (Jeyakumar, Warriner, Raval, & Ahmad, 2004). This two-subtest short form version of the WAIS-III has been used in other schizophrenia research (e.g. Gooding et al., 1999).

Vocabulary On this subtest of the WAIS-III Verbal Index, subjects were asked to define up to 33 words of increasing difficulty. Their answers received a score of 0, 1, or 2 depending on the complexity and accuracy of the response.

Block Design On this subscale of the WAIS-III Performance Index, subjects were asked to construct with colored blocks 14 designs that are presented on stimulus cards. They received scores ranging from 0 to 7 for each item depending on the accuracy and speed with which they completed the designs.

# CHAPTER FOUR: RESULTS

## Overview

This study examined the neuropsychological and clinical characteristics of individuals with RSAS elevations, individuals with PAS/MIS elevations, and control participants. Specifically, with regard to neuropsychological characteristics, the study examined sustained attention, working memory, general memory functioning, and general intellectual ability. With regard to clinical characteristics, the study examined schizophrenia-spectrum characteristics and lifetime Axis I diagnoses. Neurocognitive test scores and interviewer-obtained clinical ratings were compared among the three groups.

## Neurocognitive Index Scores

In order to determine if the groups differed with regard to sustained attention, working memory, general memory ability, and general intellectual ability, MANOVAs were performed based on the thematic relationship of the variables. Summary data for these analyses may be found in Table Two.

A MANOVA was performed on the estimate of general intellect variables, which include WAIS-III standard scores for Block Design and Vocabulary. The two variables were significantly correlated at the $\alpha = .05$ level, $r = .35$. The omnibus MANOVA was non-significant, $F\ (4, 286) = 2.08$, ns, although it did approach significance, with a $p$ value of .08. The SocAnh group demonstrated a lower, though non-significant, Block Design score than did the other two groups. The effect size was small, $\eta^2 = .03$, and observed power was .62.

A MANOVA was performed on the WMS-III standard scores of the general memory variables, which include Logical Memory I Theme, Logical Memory II Theme, Visual Reproduction I, Visual Reproduction II, and Visual Reproduction Recognition. All were significantly correlated with one another at the $\alpha = .05$ level. The range was $r = .15$ to $r = .72$, $p < .05$, with the smallest correlation between Logical Memory II Theme and Visual Reproduction Recognition, and the highest between Logical Memory I Theme and Logical Memory II Theme. The omnibus MANOVA was non-significant, $F$ (10, 266) = .86, ns. The effect size was small, $\eta^2 = .03$, and observed power was .45.

A MANOVA was performed on the WMS-III standard scores of the working memory variables, which include Digit Span, Spatial Span, and Letter Number Sequencing. All were significantly correlated $\alpha = .05$ level. The range was $r = .23$ to $r = .58$, $p < .05$, with the smallest correlation between Spatial Span and Letter Number Sequencing, and the highest between Letter Number Sequencing and Digit Span. The omnibus MANOVA was non-significant, $F$ (10, 280) = .63, ns. The effect size was small, $\eta^2 = .02$, and observed power was .34.

Finally, a one-way ANOVA was performed on the sustained attention variable, DSCPT d'. The test was non-significant; $F$ (2) = 1.61, ns. The effect size was small, $\eta^2 = .02$, and observed power was .34.

The above group findings raised a question about the cognitive characteristics of this community sample and how representative these cognitive profiles were of normative performance in other samples. That is, the lack of difference between putative at-risk groups and the control group may indicate that this sample is particularly high-functioning with respect to general intellect. One approach to exploring this question is

to use available standardized data, such as that provided in the WAIS-III standardization sample for participants aged 18-19 (Psychological Corporation, 1997). The WAIS-III is an acceptably reliable index of general cognitive functioning. The standardization sample provides a large group for comparison and is tied to functioning in the general population, stratified for sex and ethnicity. Thus, Z-scores were computed using the WAIS-III standard score mean of 10 and standard deviation of three as the expected population mean and variance (Psychological Corporation, 1997).

SocAnh group Z-scores on the Vocabulary subtest were somewhat higher than standardization sample scores, Vocabulary $\underline{Z} = 1.11$. PerMag group scores were similar; Z-scores above 1 were also Vocabulary $\underline{Z} = 1.08$, the control group was comparable, with a Z-score greater than 1 on Vocabulary, $\underline{Z} = 1.08$. Scores on the Block Design subtest were comparable for all groups. Thus, it appears that the entire sample was largely comparable to the WAIS-III standardization sample and fell within the normative range, and actually performed somewhat better than that sample on certain tests. Individuals in all groups, in addition to not significantly differing from one another, also did not differ from a nation-wide sample.

In addition to exploring general intellect as indexed by WAIS-III norms, it was thought to be illustrative to examine the cognitive abilities of college samples in studies of putative schizotypes that have reported group differences. Such an examination allows for a better understanding of whether these current null results are due to the characteristics of a heterogeneous community sample. That is, there was a possibility that this community sample had lower general intellectual functioning, as defined by the full scale IQ, than that of student samples obtained at universities. If so, null findings

might be expected given the possibility that individuals with lower general intellectual ability, regardless of schizotype status, would perform more poorly on measures of neurocognitive functioning, demonstrating a type of floor effect relative to higher functioning samples. Thus, an estimated WAIS-III full scale IQ was obtained using a conversion table for the Vocabulary and Block Design WAIS-III sum of scaled scores (Jeyakumar et al., 2004). This short form approach has an internal consistency of .94 and a part-whole validity correlation of .90 (Jeyakumar et al. 2004). This method yielded estimated full scale IQ scores for the control (M = 112.87, SD = 14.48), social anhedonia (M = 109.12, SD = 15.17), and PerMag groups (M = 115.89, SD = 14.14). It should be noted, however, that caution must be used in interpreting these scores, as WAIS-III estimated Full Scale IQ scores that are based on Vocabulary and Block Design tend to slightly overestimate the Full Scale IQ score (Jeyakumar et al., 2004).

These obtained estimates of full scale IQ were compared with those of Gooding and colleagues (2002), who utilized a sample of Chapman scale-identified schizotypes and control participants at the University of Wisconsin-Madison. In that study, for which no PerMag group was identified, the social anhedonia and control groups had mean estimated full scale IQs of 114.28 and 117.49, respectively. These means were used as test values in one-sample t-tests. These analyses revealed that the control subjects in the University of Wisconsin sample had a significantly higher mean full scale IQ than did those in the community sample in the current study, $t$ (85) = -2.96, $p < .05$. The test approached significance for the social anhedonia group, with the University of Wisconsin sample demonstrating a higher, though not statistically significant, full scale IQ than the community sample, $t$ (32) =- 1.95, ns. The lack of comparison data for the PerMag group

not withstanding, it appears that university samples may demonstrate better general intellectual ability than do community samples.

In addition to the estimate of intellectual ability obtained in this study as a broad index of overall cognitive functioning, sustained attention was also examined. Sustained attention is a potent cognitive marker of schizophrenia liability, and differences between control participants and schizophrenia patients in this domain have been well-documented (e.g., Braff, 1993; Cornblatt & Keilp, 1994; Roitman et al., 1997). Given this, it was thought that our sustained attention measure, the DSCPT, would be the most sensitive to deficits, as even non-ill biological family members of schizophrenia patients perform less well than do controls on sustained attention tasks (Maier et al., 1992; Keefe et al., 1997; Laurent et al., 1999; Erlenmeyer-Kimling et al., 2000). However, in this study, this was not the case, as group differences were not found. Thus, I sought to examine whether the current sample was comparable to others in relation to performance on the DSCPT.

To accomplish this, the d' mean for the current control group was compared to that of a control sample in another study (mean age = 24; Nuechterlein et al., 1998). A one-sample t-test, using the 1998 Nuechterlein and colleagues DSCPT d' control mean of 2.75 as a control value, revealed that the control subjects in the current sample had a significantly lower DSCPT d' score than did the control subjects in the Nuechterlein and colleagues (1998) study, $t$ (85) = -4.64, $p < .05$. Thus, the current sample may not be comparable to other samples. This may be relevant in considering the current non-significant results. The Nuechterlein and colleagues (1998) sample performed much

better on the DSCPT than did the current sample, which may indicate that the current control sample is better matched to the target groups with regard to cognitive functioning.

To further explore these null neurocognitive results, I sought to examine whether the occurrence of current major depressive disorder may have led to false identification of individuals in the SocAnh group. Given that depression may be either a prodromal indicator of schizophrenia risk or could lead to erroneous identification of individuals as socially anhedonic (because current depression can lead to transient increases in anhedonia), these analyses were performed again excluding individuals who met current criteria for major depressive disorder. When repeating these analyses excluding currently depressed individuals, the prior results were replicated. Results were non-significant for the general intellect variables, $F(4, 276) = 1.98$, $\eta^2 = .028$, the general memory variables, $F(8, 268) = .49$, $\eta^2 = .014$, the working memory variables, $F(10, 270) = .65$, $\eta^2 = .02$, and the sustained attention variable, DSCPT d', $F(2, 1.330) = 1.49$, $\eta^2 = .021$. Observed power for the general intellect, general memory, working memory, and sustained attention variables was .59, .23, .34, and .31, respectively. Thus, depression was not a factor in the current results.

The possibility of a main effect of race on these variables was also explored. MANOVAs using race as an index variable were performed. Analysis revealed a main effect of race with respect to the general intellect variables, $F(6, 260) = 7.21$, $p < .05$, $\eta^2 = .143$, general memory variables, $F(12, 346.86) = 1.88$, $p < .05$, $\eta^2 = .054$, working memory variables, $F(12, 352.18) = 2.48$, $p < .05$, $\eta^2 = .07$, and the sustained attention variable, $F(1.90, 2.26) = 5.71$, $p < .05$, $\eta^2 = .05$. There were no significant race by group interactions in any domain. Post-hoc analyses used the Tukey's Least Significant

Difference test. A summary of significant comparisons follows in the paragraphs below. Racial group differences are indicated in superscripts in Table Two.

With respect to the general intellect variables, on the Vocabulary subtest, Caucasian individuals scored a mean of 2 standard points higher than African-American individuals and a mean of 2.25 standard points higher than individuals identifying themselves as of another race. On the Block Design subtest, Caucasian individuals scored a mean of 3.25 standard points higher than African-American individuals and a mean of 2.80 standard points higher than individuals identifying themselves as of another race.

With respect to general memory variables, post-hoc analysis of the Logical Memory I and II Theme standard scores revealed that Caucasian individuals scored a mean of 1.46 and 1.43 standard points higher than African-American individuals, respectively. On the Visual Reproduction I subtest, Caucasian individuals scored a mean of 2.42 standard points higher than African-American individuals and a mean of 2.36 standard points higher than individuals identifying themselves as of another race. Visual Reproduction II met with similar results; Caucasian individuals scored a mean of 2.23 standard points higher than African-American individuals and a mean of 2.60 points higher than individuals identifying themselves as of another race. Finally, Caucasian individuals scored a mean of 1.17 standard points higher than African-American individuals on the Visual Reproduction Recognition subtest.

Post-hoc analysis of the working memory variables showed a slightly different pattern. Caucasian individuals scored a mean of 2.78 standard points higher than African-American individuals on the Spatial Span Forward subtest. African-Americans

also scored a mean of 4.85 standard points lower than individuals of Asian descent and a mean of 1.83 standard points lower than Caucasian individuals on the Spatial Span Backwards subtest. In addition, individuals of Asian descent scored a mean of 3.5 standard points higher than those identifying themselves as of another race and 3.02 standard points higher than Caucasian individuals. On Letter-Number Sequencing, Caucasian individuals scored a mean of 1.83 points higher than those identifying themselves as of another race. There were no racial group differences on the Digit Span subtest.

On the sustained attention variable, DSCPT d', Caucasian individuals demonstrated a mean of .43 units more than African-Americans. There were no racial group differences with regard to d' between other racial groups.

One further exploratory analysis was warranted. Despite the fact that there were no between-group differences when taking into account the data for the entire sample, a question remained as to whether the SocAnh group performed worse than did the control group with regard to neurocognition. This question was informed by prior studies that have demonstrated that social anhedonic individuals perform worse than do controls. Independent samples t-tests revealed that the SocAnh group performed worse than did controls on Logical Memory I (SocAnh, $\underline{M} = 8.94$, $\underline{SD} = 2.96$; Control, $\underline{M} = 10.26$, $\underline{SD} = 3.12$), $\underline{t}\ (117) = 2.09$, $\underline{p} < .05$. In addition, the SocAnh group performed worse than did controls on Block Design ((SocAnh, $\underline{M} = 9.85$, $\underline{SD} = 2.93$; Control, $\underline{M} = 11.22$, $\underline{SD} = 2.90$), $\underline{t}\ (117) = 2.30$, $\underline{p} < .05$. All other between-group differences were non-significant. These significant results are consistent with prior research, but they are interpreted with caution given that these are post-hoc analyses.

In summary, there were no group differences with regard to the neurocognitive variables. All groups were comparable to the WAIS-III standardization sample with respect to general intellect, but the control and SocAnh groups exhibited lower aptitude than that of a college sample (i.e., that of Gooding et al., 2002). In addition, the current control group performed significantly worse than did another control group (i.e., that of Nuechterlein and colleagues, 1998) on a measure of sustained attention, perhaps indicating that the current control sample was better matched to the target groups than was the other sample in this regard. There was a main effect of race, with racial group differences demonstrated on most of the neurocognitive measures, but there was no race-by-group interaction.

## Clinical Pathology Characteristics

Characteristics of clinical pathology (Axis I disorders) were compared across groups. A summary of Axis I diagnoses may be found in Table Three. To assess potential differences in the prevalence of lifetime Axis I diagnoses among the three groups, the occurrence of all diagnoses were compared with a chi-square test.

Analysis of these diagnoses revealed that there was a similar number of Axis I diagnoses across the three groups (SocAnh group, 12 diagnoses [36%]; PerMag group, 12 diagnoses [43%]; control group, 23 diagnoses [26%]), $\chi^2$ [8] ($\underline{N}$ = 148), 14.49, $\underline{ns}$. The effect size of the prevalence of lifetime diagnoses was small ($\underline{\eta^2}$ = .01). Thus, as indicated by the overall prevalence of diagnoses, the groups were comparable.

Exploratory analyses were performed with regard to specific Axis I diagnoses. Major depressive disorder and substance use disorders were most prevalent in the sample, as can be viewed in Table Three. The next most frequent disorder (dysthymia) occurred

only four times, and all other diagnoses occurred only once, if at all. Thus, it was appropriate to more closely examine the contribution of these disorders to group differences. In addition, it has been suggested that PAS/MIS elevations are more of a marker for general psychopathology than they are for schizophrenia-spectrum characteristics, particularly major depressive disorder and substance use disorders (Kwapil, 1998). Furthermore, it was necessary to demonstrate that social anhedonia scores were independent of a lifetime occurrence of major depressive disorder, as social anhedonia is associated with that disorder (Blanchard, 1998; Blanchard et al., 2001). These analyses, however, revealed no differences among the groups with regard to the lifetime occurrence of major depressive disorder, $\chi^2$ [2] = 5.91, ns, and no differences with regard to the lifetime occurrence of substance use disorders, $\chi^2$ [8] = 5.39, ns. Effect sizes related to these diagnoses were small (major depressive disorder, $\eta^2$ = .001; substance use disorders, $\eta^2$ = .01), as was observed power (major depressive disorder, .06; substance use disorders, .17).

Figure One presents a visual representation of GAF scores for each group. Group differences were found with regard to general functioning as measured by the GAF score, $F$ (2) = 5.74, $p < .05$. Subsequent post-hoc analysis using Tukey's Least Significant Difference test indicated that SocAnh participants had significantly lower GAF scores than did participants in the other two groups, $p < .05$. PerMag participants did not differ from SocAnh or control members on this index. The proportion of variance attributable to group membership, however, was small, $\eta^2$ = .07.

Given group differences in racial composition, it became a question as to whether observed statistics for prevalence of Axis I disorders and GAF score could be accounted

for by such differences. In order to explore this, a MANOVA was performed with both race and group as independent variables. The main effect of race was non-significant for the prevalence of Axis I scores, $F$ (3) = 2.39, ns. The main effect of race was significant for the GAF score, $F$ (3) = 2.73, $p < .05$, with African-American individuals scoring a mean of 14.97 fewer points on the GAF scale than those of Asian descent, $p < .05$, $\eta^2$ = .05. There was no group by race interaction.

## Schizophrenia-Spectrum Characteristics

Summary data for schizophrenia-spectrum personality disorder diagnoses may be found in Table Four. The occurrence of schizophrenia-spectrum diagnoses was analyzed among groups with a chi-square test. Analysis revealed that individuals with RSAS elevations had significantly more schizoid personality disorder diagnoses, n = 6, $\chi^2$ [2] = 13.64, $p < .05$, and more paranoid personality disorder diagnoses, n = 6, $\chi^2$ [2] = 11.21, $p < .05$ than did the other two groups. There were no significant differences among groups with respect to schizotypal personality disorder diagnoses, $\chi^2$ [2] = 1.10, ns.

Means and standard deviations for IPDE dimensional score data may be found in Table Five. Given that many participants received positive scores on the IPDE subscales even if they did not meet full criteria for a disorder, it was also deemed appropriate to perform a one-way ANOVA on dimensional scores in order to obtain a more thorough representation of pathology among the groups. This strategy has been used in other studies (e.g., Kwapil, Crump, & Pickup, 2002). A one-way ANOVA revealed significant differences among the groups with regard to IPDE dimensional scores. The SocAnh group demonstrated more pathology than the PerMag group and the control group on schizoid personality disorder dimensional scores, $F$ (2) = 18.02, $p < .05$, $\eta^2$ = .20,

schizotypal personality disorder dimensional scores, F (2) = 6.69, p < .05, $\eta^2$ = .09, and paranoid personality disorder dimensional scores, F (2) = 5.05, p < .05, $\eta^2$ = .06. Moreover, the social anhedonia group had more schizophrenia-spectrum personality characteristics overall than did the other two groups, F (2) = 15.42, p < .05, $\eta^2$ = .18. There were no group differences between the PerMag group and controls with regard to the schizophrenia-spectrum variables.

In order to determine if schizophrenia-spectrum personality characteristics were associated with lower global functioning as indicated by the GAF score, exploratory Pearson's correlations were performed within at-risk groups. Within the SocAnh group, schizophrenia-spectrum personality characteristics were significantly negatively associated with GAF scores for schizoid personality disorder, r = -.53, p < .05, paranoid personality disorder, r = -.55, p < .05, schizotypal personality disorder, r = -.62, p < .05, and total schizophrenia-spectrum personality pathology, r = -.70, p < .05.

Within the PerMag group, results did not follow the same pattern. With the exception of greater paranoid characteristics being significantly associated with lower GAF scores, r = -.40, p < .05, no other correlation reached the level of significance. Non-significant correlations were observed for schizoid personality disorder, r = .148, ns, schizotypal personality disorder, r = -.11, ns, and total schizophrenia-spectrum characteristics, r = -.18, ns. In summary, while greater schizophrenia-spectrum pathology (overall and within diagnostic category) was associated with lower global functioning in the SocAnh group, only paranoid characteristics were associated with lower global functioning in the PerMag group.

With respect to individuals who met criteria for a schizophrenia-spectrum disorder, the results remained the same when individuals with current major depressive disorder were excluded (as was done with the neurocognitive data). Non-depressed individuals with RSAS elevations had significantly more schizoid personality disorder diagnoses, n = 6, $\chi^2$ [2] = 11.41, $p < .05$, and more paranoid personality disorder diagnoses, n = 6, $\chi^2$ [2] = 9.17, $p < .05$, than did the other two groups. There were no significant differences among groups with respect to schizotypal personality disorder diagnoses, $\chi^2$ [2] = 1.21, ns.

With respect to schizophrenia-spectrum personality characteristics, the results remained the same when individuals with current major depressive disorder were excluded. Individuals in the social anhedonia group demonstrated more pathology than the PerMag group and the control group on schizoid personality disorder dimensional scores, $F$ (2) = 15.91, $p < .05$, schizotypal personality disorder dimensional scores, $F$ (2) = 5.62, $p < .05$, and paranoid personality disorder dimensional scores, $F$ (2) = 4.70, $p < .05$. Moreover, the social anhedonia group had more schizophrenia-spectrum personality characteristics overall than did the other two groups, $F$ (2) = 12.75, $p < .05$.

Again, group differences in racial composition were a consideration in the evaluation of these data. A MANOVA using race as an index variable to examine both diagnoses and IPDE dimensional scores indicated that there was no main effect of race for any dependent variable, $F$ (6) = 1.44, ns.

Although there were no group differences in cognitive functioning, exploratory correlational analyses were conducted to examine the hypothesis that greater neurocognitive impairment would be associated with greater schizophrenia-spectrum

pathology in the putative schizotype groups. Zero-order correlations were performed within the high-risk groups to examine this. See Tables Six and Seven for a summary of correlations regarding the linear relationship between neurocognitive scores and IPDE dimensional scores in the SocAnh and PerMag groups, respectively. These correlations were computed within each putative at-risk group. In the social anhedonia group, schizotypal dimensional scores were negatively correlated with Visual Reproduction II Recognition, $r = -.42$, $p < .05$. That is, poorer visual recognition was associated with greater schizotypal personality pathology. Schizoid, paranoid, and total schizophrenia-spectrum IPDE dimensional scores in the social anhedonia group were not significantly correlated with any neurocognitive variable at the $p = .05$ level.

In the PerMag group, paranoid dimensional scores were negatively correlated with Visual Reproduction I total scores, $r = -.47$, $p < .05$ and Visual Reproduction II total scores, $r = -.51$, $p < .05$. Thus, poorer visual memory was associated with greater paranoid personality pathology. Schizoid, schizotypal, and total schizophrenia-spectrum personality pathology in the PerMag group were not significantly correlated with any neurocognitive variable at the $p = .05$ level. To sum, poor visual recognition was associated with greater schizotypal scores in the SocAnh group, and poor visual recall was associated with greater paranoid scores in the PerMag group.

Given that several individuals in the SocAnh group crossed the threshold for meeting the diagnostic criteria for a schizophrenia-spectrum disorder, exploratory analyses was performed to determine if these individuals had a different neurocognitive profile than did SocAnh participants who did not meet criteria for such a disorder. Independent samples t-tests demonstrated that SocAnh individuals who met criteria for a

schizophrenia-spectrum disorder did not differ from SocAnh individuals without a schizophrenia-spectrum disorder on the basis of any neurocognitive variable.

# CHAPTER FIVE: DISCUSSION

## Overview

The psychometric detection paradigm has been used extensively to identify individuals putatively at risk for schizophrenia and related disorders. The most promising and widely used self-report measures are the Revised Social Anhedonia Scale and the combined Perceptual Aberration/Magical Ideation scales. Based on a review of the extant literature, however, it is unclear whether each of the Chapman scales truly identifies schizotypy. Data have demonstrated different clinical outcomes for each group (e.g., Kwapil, 1998). Unfortunately, there has been a lack of direct comparison between the SocAnh and PerMag groups. "Pure" samples that exclude individuals with elevations on another Chapman scale are infrequently identified, rendering attributions regarding the contribution of each scale's characteristics difficult. Moreover, existing studies have been performed in college samples, the data from which may not be generalizable to the population as a whole. In an attempt to address some of these problems, this study examined the neurocognitive characteristics of psychometrically-identified schizotypes in a community sample.

Schizophrenia patients evince a wide range of cognitive deficits. Drawing from investigations which show that family members of schizophrenia patients show some of those deficits, it would be expected that those putatively at risk for the disorder would exhibit some of those same deficits, even if in attenuated form. Thus, the purpose of this study was to examine memory, attention, and intellect in those psychometrically defined as at risk in an attempt to determine if the RSAS and PAS/MIS capture distinctly different groups. In addition, schizophrenia-spectrum personality characteristics were

investigated. This study was the first to simultaneously examine individuals with RSAS and PAS/MIS elevations from a community-based sample on the basis of neurocognitive indices. Further, each putatively at-risk group was "pure," that is, no member of one group had an elevation on the scale that formed the basis of identification for the other group. The main hypothesis, that is, that SocAnh individuals would demonstrate more neurocognitive impairment than would PerMag members or controls, found no support. However, clinical data (i.e., schizophrenia-spectrum characteristics) demonstrated that the SocAnh group had more schizophrenia-spectrum pathology than did the PerMag group or control participants.

## Neurocognitive Performance

The first aim of this study was to examine the neurocognitive profile of psychometrically-identified putative schizotypes. Specifically, it was hypothesized that individuals with RSAS elevations would perform more poorly on measures of working memory, verbal and visual memory, and sustained attention than would individuals with PAS/MIS elevations or control participants. The results of this investigation are inconsistent with the idea that psychometrically-identified schizotypes have poorer neurocognitive functioning than those not at risk for schizophrenia; putative schizotypes did not differ from controls in any neurocognitive domain. No neurocognitive "signature" was demonstrated in either group. This was one of the first studies (along with Tallent & Gooding, 1999 and Gooding, 1999) to examine the neurocognitive and clinical characteristics of individuals with RSAS or PAS/MIS elevations concurrently, which is key in determining whether possible deficits are unique to a particular trait.

Below, the current results as they compare to existing literature will be summarized. Then, theoretical and methodological interpretations of the data will be discussed.

Attention and Memory

Attentional deficits have been shown to be a promising predictor of schizophrenia (Gottesman & Erlenmeyer-Kimling, 2001). Not only do schizophrenia patients differ from controls, but unaffected family members also perform more poorly than controls in this domain (e.g., Gottesman & Erlenmeyer-Kimling, 2001; Conklin, Curtis,Katsanis & Iacono, 2001), which was thus proposed as an area of study in the current investigation. This was only the second study to examine sustained attention deficits in individuals with RSAS elevations; the other (Kwapil & Diaz, 2000) met with positive results. Several studies have examined sustained attention deficits in individuals with PAS elevations, two (Lenzenweger, Cornblatt, & Putnick, 1991; Lenzenweger, 1998) determined that such individuals do have sustained attention deficits, and the other (Lenzenweger, 2001) did not, although the author of this study proposed that these findings were due to low power. The findings from these studies, however, may not be as interpretable as the findings from the current study, as the measure used in the current study utilized a task that is specific to sustained attention and early visual processing (the DSCPT), while the others used tasks that also recruited the working memory (e.g., the CPT-Identical Pairs task).

The current study found that neither putatively at-risk group demonstrated sustained attention deficits as compared to controls. Comparability with prior studies is limited, however, given that this is the first study of this kind to use the DSCPT as a measure of sustained attention. However, comparison with a control sample of

individuals with a mean age in their mid-twenties (Nuechterlein et al., 1998) indicated that the control sample in the current study performed more poorly on the DSCPT than did those in the Nuechterlein and colleagues (1998) study. It is difficult to make a conclusion about this on the basis of one comparison study, but two conjectures may emerge. First, it is possible that the functioning of this control sample with regard to sustained attention may have been worse than in the general population, which would have precluded significant results. Alternatively, consider that perhaps the Nuechterlein and colleagues control sample comprised individuals who were at a level of functioning higher than that of the general population. This could occur by defining "normal" as optimal functioning, which may be rare in the general population. If this were the case, these "super-normals" would be expected to demonstrate sustained attention capacity that is higher than expected for the general population. A sample of control participants not pre-screened for pathology, as was obtained in the current study, may be more representative of the sustained attention capacity of the general population. If differences between controls selected for not falling in the extremes of schizotypy characteristics and putative schizotypes exist, they may be too subtle for detection by the DSCPT.

Working memory dysfunction, present during all levels of symptom presentation in schizophrenia, is considered a trait, rather than a state, marker of the disorder (Alemon et al., 1999; Kurtz et al., 2001). Furthermore, working memory dysfunction is considered to be specific to the schizophrenia diagnosis (e.g., Park & Holzman, 1992). Thus, it is a reasonable prediction that if the psychometric detection paradigm is a valid predictor of future schizophrenic decompensation, working memory function would be a robust differentiator between putative at-risk and non-at-risk groups. However, this was not

borne out in the current study; neither putatively at-risk group differed from controls on measures of spatial or auditory working memory. Moreover, groups did not differ on measures of verbal or visual recall and retention, although there is no existing literature with which to compare these data.

These results add to the uncertain nature of the existing working memory in schizotypy literature, which is comprised by conflicting results. Of course, no study of this nature thus far has used Letter-Number Sequencing, Digit Span, and Spatial Span together as measures of working memory, and this does limit comparison among studies. Of studies examining putative at-risk groups alone or mixed together, Gooding and Tallent (2003) reported that individuals with elevated social anhedonia have deficits in spatial working memory, and two studies have reported impaired visual working memory in schizotype groups that had elevations on one or more of the Chapman scales (Balogh & Merritt, 1985; Park, Holzman, & Lenzenweger, 1995). Those results were not replicated in this study, but the current data are somewhat in line with the results of Tallent and Gooding (1999), who found in the only other tandem investigation of individuals with RSAS or PAS/MIS elevations that the two groups did not differ on a measure of spatial working memory. However, in that study, the groups differed from the control group in that domain, which was not the case in the current study.

With regard to auditory working memory, Lenzenweger and Gold (2000) reported that individuals with PAS elevations did not differ from controls on an auditory measure, which was the case in the current investigation. However, a previously published paper (Gold, Carpenter, Randolph, Goldberg, and Weinberger, 1997) reported that such schizotypes demonstrated deficits on a measure of spatial working memory and another

visual task that recruits the working memory (the Wisconsin Card Sorting Test). Taken together with the results of Tallent & Gooding (1999), Balogh & Merritt (1985) and Park, Holzman, & Lenzenweger (1995), this may indicate that visual, rather than auditory, working memory should be the domain of interest. It is clear that more study is needed before drawing a strong conclusion regarding the nature of working memory in psychometrically-identified schizotypes.

Neurocognition and Schizophrenia-Spectrum Dimensional Scores

As an exploratory analysis, correlations between neurocognitive index scores and schizophrenia-spectrum dimensional scores were computed in the putatively at-risk groups. While three significant correlations indicated a negative association between schizophrenia-spectrum characteristics and visual memory in both at-risk groups, the majority of correlations did not reach the level of significance or were in the unexpected direction. This could possibly be accounted for by the fact that, while clinical pathology may emerge around the age at which these participants were evaluated, deterioration in neurocognitive functioning may not surface until a later age, if at all. This possibility will be discussed further in the sections below.

Significant correlations indicated that worse visual memory was associated with greater schizophrenia-spectrum pathology in both at-risk groups. In the SocAnh group, poorer visual recognition was associated with greater schizotypal dimensional scores, and in the PerMag group, poorer visual learning and recall were associated with greater paranoid dimensional scores. Prior data show that visual measures of working memory are a differentiator of the SocAnh and PerMag groups (e.g., Tallent & Gooding, 1999; Balogh & Merritt, 1985; Park, Holzman, & Lenzenweger, 1995; Lenzenweger & Gold,

2000), which was not the case in this study. However, this may be accounted for by the current study's group selection procedure (i.e., selecting only individuals with "pure" elevations); that is, it may be the case that pure elevations on one scale alone do not account for neurocognitive differences. Taking together past studies and the current data, it seems that visual processing may be a prime domain of interest for future studies.

Main Effect of Race

Given that the groups differed on the basis of race, exploratory analyses examined whether there was a main effect of race on the neurocognitive variables. This proved to be the case, with Caucasians and Asians generally performing better on neurocognitive indices than other race groups. It is clear that these racial differences did not contribute to the current results, as there was no race-by-group interaction. These results, however, call to attention the issue of racial representation in studies of putative schizotypes. Most prior studies (e.g., Kwapil, 1998; Gooding, 2000) of schizotypes have utilized samples that were largely white, which is likely related to the fact that these were college samples in areas where minorities were under-represented. These results indicate that other racial groups may demonstrate cognitive functioning different from that of Caucasians, and bear upon the interpretability and generalizability of data gathered from largely white samples.

Accounting for Current Depressiona

Given that depression could lead to erroneous identification of individuals as socially anhedonic (considering the association of transient anhedonia with depression; Blanchard et al., 2001), analyses were performed a second time excluding individuals

with a current diagnosis of major depressive disorder. The exclusion of such individuals from the analyses had no impact on the neurocognitive findings.

### What Accounts For These Findings? Theoretical and Methodological Considerations

In this study, we theorized that putatively at-risk individuals would show a pattern of neurocognitive deficits comparable to individuals with schizophrenia. The question was whether individuals with RSAS elevations and individuals with PAS/MIS elevations represent the same group of individuals at risk for developing psychopathology. In this context, what accounts for the current data? This section explores reasons that may be central for the current pattern of results.

First, certain of the neurocognitive measures chosen for this study (i.e., the WAIS-III and WMS-III subtests) are designed to be measures of general cognitive ability. While they may be sensitive to large decrements in functioning relative to controls in schizophrenia patients, they may not have enough discriminating power to detect the presumably smaller deficits in individuals who, though thought to be at risk for schizophrenia, have not yet developed the disorder. Consider that Lenzenweger and Gold (2000) did not find differences in auditory working memory when comparing individuals with PAS elevations and controls.

That not every individual in the SocAnh group demonstrated clinical characteristics raises the possibility that the sample may have contained participants who were falsely identified as schizotypes. If certain individuals were experiencing depressive symptoms, which can lead to transient increases in social anhedonia (Blanchard et al., 2001), at the time that they completed the Chapman scales, some false-positives may have been identified. Thus, determining the long-term temporal stability of

social anhedonia is key in determining true schizotypy status. Such an assessment is included in the longitudinal study of which this investigation is a part. In addition, other markers of schizotypy (e.g., perceptual aberrations) or pathological characteristics (e.g., an anxiety disorder) may influence the development of social anhedonia secondary to the expression of that characteristic. In Meehl's (2001) conceptualization of secondary hypohedonia, anhedonia may manifest as, for example, a response to avoiding social situations due to perceptual aberrations. Following this conjecture, individuals may have been falsely identified as socially anhedonic in the current sample due to secondary anhedonia. Anhedonia may be one of the final expressed characteristics of disorders that were not examined in the current study, which might lead to falsely identified schizotypes and null results.

Besides the contribution of depression and other characteristics to false-positive status, there is a large false-positive identification rate that has been reported in prior studies using the Chapman scales. Kwapil's (1998) longitudinal study of putative schizotypes demonstrated that 24% of social anhedonic participants met criteria for schizophrenia spectrum disorders at the 10-year follow-up. This is striking, but it then follows that 76% of those individuals did not develop clinically significant schizophrenia-related pathology, and no individual in the sample developed schizophrenia (Chapman et al., 1994). Using the current data based on the joint phenotype/endophenotype (behavioral/neurocognitive) strategy, it appears that behavioral markers such as those on the RSAS and PAS/MIS may not be sufficient to indicate a schizophrenia-related pathophysiology. This raises questions regarding the validity of the Chapman scales in identifying individuals who are actually at-risk for schizophrenia.

Perhaps this method does not tap into more specific markers of schizophrenia risk that would decrease the possibility of false positives. It should be noted, however, that the individuals in that long-term study had not passed through the entire high-risk period for developing schizophrenia at the time of the follow-up study.

While neurocognitive functioning did not differentiate the groups, substantial clinical and functional differences emerged. The SocAnh group demonstrated significantly more schizophrenia-spectrum pathology than did the other groups, which supports the idea that individuals in the SocAnh group comprise a unique class of individuals that may be more liable to develop schizophrenia than those in the PerMag group. Moreover, the SocAnh group demonstrated significantly worse global functioning. This will be discussed further in a later section.

May It Be Too Difficult to Measure Neurocognition Premorbidly?

A somewhat controversial issue is whether neurocognitive deficits are apparent in the premorbid stage of schizophrenia, or if neurocognitive performance deteriorates over time after the appearance of the first psychotic episode. If the deterioration hypothesis holds true, it may be that this study's snapshot of neurocognition took part while the at-risk participants, presumably still in a premorbid stage, were too young and too psychologically healthy to demonstrate impairment on neurocognitive indices.

Several studies have demonstrated that such deterioration does occur over time, and that poor premorbid neurocognitive functioning predicts the development of schizophrenia. For instance, Ang and Tan (2004) demonstrated that deterioration in math ability and poor educational outcome characterized first-episode schizophrenia patients.

Conflicting evidence, however, exists. Children thought to be at-risk for schizophrenia demonstrated extremely low performance on a measure of sustained attention as children as opposed to those who did not (i.e., decrements are present from childhood, rather than representing a decline; Cornblatt & Erlenmeyer-Kimling, 1986). A high IQ in childhood has been deemed to be a protective factor in individuals who were considered to be at-risk for schizophrenia as children (Erlenmeyer-Kimling & Cornblatt, 1987), suggesting that if cognitive abilities are intact from the start, they will remain intact. However, Amminger, Edwards, Brewer, Harringan, and McGorry (2002) have shown that higher premorbid IQ is an independent predictor of cognitive *deterioration* in schizophrenia patients, a non-intuitive result. At the same time, Swanson, Gur, Bilker, Petty, and Gur (1998) determined that a higher level of education predicted fewer symptoms (especially negative symptoms), better premorbid adjustment in a variety of domains, and importantly, a better neurocognitive profile. That education is related to clinical presentation does not necessarily suggest that education is a protective factor for neurocognition, as those individuals destined to have a worse presentation may simply have had poorer premorbid academic ability, but it nonetheless represents an index of predictive utility.

Certain of the above studies suggest that deterioration over time is to be expected in schizophrenia and that one may not be able to demonstrate impairments in neurocognition while putatively at-risk participants are in the premorbid stage. Other researchers, however, disagree, and conclude that cognitive ability remains stable over time. Russell, Munro, Jones, and Hemsley (1997) refute the "myth of intellectual decline" in schizophrenia with their data, which showed no significant differences

between child and adult full-scale IQs in schizophrenia patients. IQ scores were greater than one standard deviation lower than the general population's at both time points, suggesting, the authors say, that children who later develop schizophrenia have lower than average intellectual ability to begin with.

Other authors, however, offer a rebuttal to this claim and critique the methods of the study by Russell and colleagues (1997). For example, Gold (1998) counters that the WAIS-R (which was used by Russell, et al.) likely overestimates the full-scale IQ of adult schizophrenia patients, as that version of the WAIS does not include certain subtests, such as Letter-Number Sequencing, on which poor performance is common in schizophrenia patients. Given that working memory (which Letter-Number Sequencing measures) is considered a reliable marker of schizophrenia risk (Alemon et al., 1999; Kurtz et al., 2001), this is a particularly compelling argument. Gold (1998) also points out that WAIS-R scores tend to be higher than WISC-R scores, so the fact that there were no differences between child and adult full-scale IQs may actually represent a decline in cognitive functioning.

Whether or not there is cognitive decline, performance in young adulthood, as was measured in the current study, may not be a good predictor of clinical outcome. In one study (Stirling, White, Lewis, Hopkins, Tantam, Huddy, & Montague, 2003), neurocognitive performance in schizophrenia patients at the 10-year follow-up point was negatively correlated with clinical pathology, while performance at baseline was not. Therefore, the data gathered in this study may not be a reliable predictor of later psychopathology, even if the at-risk individuals in the current sample later do show cognitive decline.

Despite evidence of cognitive decline, certain indices may not show decline even while clinically significant symptoms are present, or deficits may be trait-related. While verbal learning deterioration has been shown to be a state-related correlate of a schizophrenia episode, other indices, such as semantic memory, may be trait-related and not subject to pathology-related decrements in functioning (Albus, Hubmann, Scherer, Dreikorn, Hecht, Sobizack, & Mohr, 2002). In addition, there does not seem to be progressive neurocognitive deterioration in the first few years after schizophrenia onset (Albus et al., 2002). Two conclusions may be drawn from these data. First, certain trait-related domains, such as semantic memory, may only differentiate non-symptomatic at-risk individuals from those who are not at-risk if they already have a pre-existing deficit in that domain. Individuals whose semantic memory has been spared may not appear different, even if they are true schizotypes. Second, it appears that it may be premature to make conclusions about the risk for schizophrenia on the basis of even the state-related neurocognitive profiles of individuals in their late adolescence.

Existing evidence regarding deterioration is conflicting. Taken together, it suggests that while certain deficits may exist pre-morbidly, those deficits can be expected to show further deterioration over time by the presence of active schizophrenia symptoms. Certain of the above findings suggest that impairment may not be present prior to illness, and the current null results should be interpreted in that context.

Methodological Considerations

The above are theoretical interpretations for the data, but methodological issues need to be considered as well. Overall, neurocognitive measures are fallible in the detection of a pathological endophenotype, and the presence or absence of deficits does

not always reliably predict the development of schizophrenia (Gottesman & Erlenmeyer-Kimling, 2001). Thus, individuals with Chapman scale elevations may in fact develop schizophrenia-related pathology over the course of the next several years despite a lack of definitive neurocognitive deficits.

In addition, consider that while this study used pure groups that did not include individuals with elevations on more than one scale, other studies did not examine pure groups. Such a method may have revealed intact functioning in the pure groups that was hidden in the other studies by virtue of the fact that those groups may not have been entirely distinct from one another (i.e., displayed overlap on Chapman scale items).

It should be noted that in the Kwapil and colleagues (1998) study that found striking schizophrenia-spectrum symptom outcome differences between individuals with RSAS elevations and individuals with PAS/MIS elevations, it was the individuals who demonstrated elevations on both the RSAS and the MIS who later developed the greatest pathology. Thus, it appears that selecting homogenous groups in this study provides evidence that a combination of positive and negative schizotypy symptoms confers schizophrenia risk more than does a single scale elevation. Consider also that Gooding and colleagues (2000) used pure at-risk groups in a smooth pursuit eye-tracking paradigm and did not find significant differences between groups, although both were different from the control group. This raises the likelihood of the hypothesis that some combination of positive and negative schizotypy is more predictive of a pathological outcome.

Given this, the current study is elucidating in that it clarifies whether the various characteristics of schizotypy independently contribute to neurocognitive impairment.

The results of this study suggest that pure elevations on the RSAS or PAS/MIS are not independently associated with neurocognitive impairment. The co-occurrence of schizotypy characteristics may be particularly important in predicting schizophrenia risk, as evidenced by the worse outcomes of individuals in the Kwapil and colleagues (1998) study who had both social anhedonia and magical ideation.

Another explanation is that certain individuals, represented in all groups, were perhaps at risk for other disorders that were not yet expressed. Recall that the sustained attention deficit, while consistently found in schizophrenia, is a fallible marker of liability and is not specific to the schizophrenia diagnosis. Schizophrenia shares the deficit with bipolar disorder (Clark, Iverson, & Goodwin, 2002; Ferrier & Thompson, 2002) and other affective disorders (Cornblatt, Lenzenweger, & Erlenmeyer-Kimling, 1989). Thus, individuals who were liable for the development of bipolar disorder may have been scattered among the comparison groups, which would have precluded significant results, at least with regard to sustained attention. It is apparent from these analyses, however, that major depression did not contribute to the results.

In addition, the current results could be interpreted in light of the fact that this study utilized a heterogeneous community sample rather than a college sample, which had not been done to this point. Students attending the same college comprised participant groups in other studies (e.g., Park, Holzman, & Lenzenweger, 1995; Dickerson, Diaz, & Kwapil, 2000; Gooding & Tallent, 2001). To be at the same college raises the point that those students are presumably at a similar level of functioning, and would thus exhibit a restricted range of scores on cognitive tasks. Thus, any deviation from this norm may become statistically significant. That is, even if the difference in

performance in the schizotypy group is small, it will become apparent in the context of such restriction. In this sense, the college recruitment strategy may be more sensitive to small incremental differences in cognitive functioning. These differences may not be clinically significant, but have potential for the identification of schizophrenia risk.

Consider that Gooding and colleagues (2002) had a relatively high-functioning university sample with regard to general intellectual ability. In contrast, the current sample demonstrated full-scale IQs in the average range, with considerable variability, and demonstrated lower (though not statistically lower in the case of the SocAnh group) full-scale IQs than putative schizotypes in another study (Gooding, 2002). Thus, the variability in aptitude of individuals in a heterogeneous sample may be more diffuse, and therefore fail to show statistical significance. In this study, standard deviations across groups were similar and relatively large, pointing to the degree of variability in this sample.

With regard to an estimate of general intellectual functioning, groups did not differ from each other on verbal or performance aptitude measures, and each group scored within the average or above-average range in those domains with regard to standard WAIS-III qualitative descriptors (Psychological Corporation, 1997). Indeed, Z-score comparisons showed that the groups were at a level of functioning similar to, or higher than, the WAIS-III standardization sample for 18-19 year-olds (Psychological Corporation, 1997). One study (Fuller et al., 2002) revealed that individuals with schizophrenia show cognitive deficits as early as elementary school and that deficits become more pronounced in mid-adolescence. Overall, then, the samples were at a level of intellectual functioning higher than one would expect of individuals at risk for

developing schizophrenia. This is a context in which to interpret the neurocognitive data from this study. If one of the best predictors of the development of schizophrenia appears to be poor intellectual functioning (Davidson et al., 1999), the captured sample does not appear to be representative of those at risk for schizophrenia.

The fact that this study employed the WAIS-III and the WMS-III as primary measures of neurocognitive functioning, however, raises an additional question. The tasks that comprise these batteries may simply be more sensitive to gross impairments in cognitive functioning rather than subtle ones. Thus, given that individuals in the at-risk groups have not developed schizophrenia, these tests may not have been entirely appropriate in detecting what may be more subtle premorbid neurocognitive deficits.

In addition, putative at-risk group sample sizes were relatively small. Recruitment difficulties precluded reaching a goal of 40 individuals per group, the adequacy of which was determined by power analysis. Although there were no significant differences on the neurocognitive tests, most of the results were in the expected direction, with individuals in the SocAnh group demonstrating poorer performance on most of the cognitive tasks, albeit non-significantly, than those in the other groups. The lower aptitude scores of the SocAnh group approached significance. However, post-hoc power analyses indicated that effect sizes were negligible for all analyses. The associated low power suggests that were there as many participants in each group as was originally intended, there still would not have been sufficient power to detect significant differences on these indices.

Finally, another note on the nature of the current sample is warranted. Of 3,494 individuals who agreed by phone to complete and return the Chapman scales, 1,493

returned them. Of those who returned them and were identified as potential participants in the laboratory assessment, a smaller percentage agreed to present for the study. It is not possible to determine on the basis of the current data why some individuals chose not to return the measures or chose not to participate in the laboratory assessment once contacted. However, this raises the issue of response bias. Individuals who chose not to participate could have made that decision for any number of reasons, including disorganization, forgetfulness, or symptom occurrence (such as depression or paranoia) that may have discouraged them from wanting to participate. This may have resulted in an obtained sample that was neurocognitively and/or clinically higher-functioning than the identified population of possible participants. Such a sample could produce data that are not representative of the actual neurocognitive and clinical functioning of the target groups.

To sum, no identifying pattern of neurocognitive results was observed in either the SocAnh or the PerMag group in the domains of sustained attention, working memory, and general memory. Neither group differed from the control group. While these data may indicate that the Chapman scales fail to reliably identify individuals at risk for schizophrenia on the basis of endophenotypical characteristics and that the two putatively at-risk groups do not form distinct groups, methodological limitations must be considered in the interpretation of the data.

## Clinical Characteristics

The second aim of this study was to determine whether individuals with RSAS elevations demonstrate greater schizophrenia-spectrum pathology than would individuals with PAS/MIS elevations or controls. Specifically, it was hypothesized that social

anhedonic individuals would have higher schizophrenia-spectrum personality characteristics than individuals in the other groups. Results were consistent with that prediction. Individuals with RSAS elevations showed more schizoid, schizotypal, paranoid, and total schizophrenia-spectrum characteristics than did individuals in the other two groups, which did not differ. With regard to secondary analyses, groups did not differ with regard to Axis I diagnoses, and the SocAnh group demonstrated lower global functioning as measured by the GAF score.

That groups did not differ with regard to clinical pathology is important in two main respects. First, on the basis of this cross-sectional data, the PAS and MIS did not identify a group that is more liable to exhibit current psychopathology than was the control group. With regard to identifying objectively different groups on the basis of current psychopathology, the Chapman scales were not useful with regard to Axis I characteristics. Second, these results establish that reported social anhedonia in the SocAnh group was independent of the presence of major depression and that negative neurocognitive results were likely thus not the result of depression.

That SocAnh group members demonstrated lower global functioning may reflect that attenuated negative symptomatology had a deleterious effect on social, interpersonal, affective, and occupational functioning, even without any apparent decrease in cognitive functioning. Anhedonia has been demonstrated to be correlated with poor social functioning (for a review, see Blanchard & Panzarella, 1998). This study supports the notion that social anhedonia has such an effect on functioning even in the absence of a schizophrenia diagnosis, suggesting that it is important to identify regardless of schizophrenia risk status. Indeed, within the SocAnh group, higher schizophrenia-

spectrum personality dimensional scores were associated with lower GAF scores for each of the personality disorders and for total personality pathology.

At the same time, it must be acknowledged that while the SocAnh group did have a statistically significantly lower GAF score than did the other two groups, their mean score (74.88) reflects a qualitatively modest level of impairment, as reflected by the DSM-IV GAF descriptors. That is, the SocAnh mean GAF score falls in the 71-80 range, indicating only transient symptoms or slight impairment (American Psychiatric Association, 1994). This could perhaps be addressed in light of the fact that individuals in the sample were in late adolescence; many were living at home and were not required to work or do other activities with which negative symptomatology could interfere. Long-term follow-up may find that this mean GAF score demonstrates downward drift as the individuals age and life demands more of them.

It is notable that greater schizophrenia-spectrum pathology was associated with lower GAF scores in the SocAnh group, but that such an association only occurred with regard to paranoid characteristics in the PerMag group. Although it cannot be empirically investigated with the current data, it could be that, given the SocAnh individual's distaste for social interaction, operating in a world that requires regular interaction with others renders them more vulnerable to distress and/or dysfunction.

As for schizophrenia-spectrum clinical characteristics and diagnoses, the SocAnh group demonstrated more of these than did the other two groups, while the PerMag group and the control group did not differ. It was somewhat surprising that the PerMag group did not differ from controls in this respect given previous research which has found elevated schizophrenia-spectrum characteristics in such samples (e.g., Blanchard &

Brown, 1999, Tallent & Gooding, 1999). That the SocAnh group evidenced more of such pathology was consistent with the hypothesis and serves as a replication of prior studies (e.g., Kwapil, 1998; Blanchard & Brown, 1999, Tallent & Gooding, 1999). It is interesting, however, that while SocAnh individuals evidenced more schizoid and paranoid personality disorder diagnoses, no between-group differences were found with regard to schizotypal personality disorder diagnoses. It is possible that this occurred due to the sampling method used in this study, which selected SocAnh participants who did not have elevations on the PAS/MIS. In light of this, the current results are not surprising given that PerMag characteristics are more closely associated with schizotypal personality characteristics, and the current SocAnh group did not have elevations of such characteristics.

Given that schizophrenia is associated with such a wide range of brain dysfunction, including structural and chemical anomalies, it follows that schizophrenia-spectrum pathology in supposedly premorbid individuals would be associated with neurocognitive dysfunction. If not neuropathology, from where are such symptoms arising? Ultimately, these clinical data suggest that SocAnh individuals are distinct from individuals in the PerMag group, while the neurocognitive data, which are presumably a closer representation of the endophenotype, were non-significant among groups. One explanation is related to the fact that, as mentioned earlier, the neurocognitive measures used in the current study were not designed to detect subtle differences in cognitive functioning between groups in research studies, but to detect clinically significant differences in the general population. While a variety of studies have determined that schizophrenia patients demonstrate cognitive deficits as related to normal controls, it was

expected that the individuals in this study's at-risk groups would only exhibit an attenuated profile of cognitive deficits. If these measures are not sensitive to subtle differences, it would follow that the at-risk individuals in this study would not appear different from control participants. Based on this explanation, no firm conclusion can be made regarding the origin of the clinical symptoms.

Evidence exists to support this conjecture; several studies have failed to find differences between control and psychometrically-defined schizotypes on the basis of neurocognitive measures. For instance, Obiols and colleagues (1999) found that, when used as an index variable, sustained attention deficits do not reliably separate samples into individuals with PAS and RSAS elevations. With regard to working memory, measures of auditory working memory (Lenzenweger and Gold, 2001) and visuospatial working memory (Faraone et al., 2001) have failed to elicit significant differences between at-risk groups and control participants, leading Lenzenweger and Gold (2001) to speculate that working memory deficits may surface only after the presentation of clinical illness. Alternatively, perhaps those deficits do exist, but were too subtle to be detected in their measurement paradigm.

If one assumes, however, that the at-risk groups simply were not cognitively different from the control group and that scores on these neurocognitive measures adequately represent the neurocognitive functioning of all groups, a different explanation for between-group differences on clinical indices must be sought. It could be that biological and environmental determinants influenced schizophrenia-spectrum pathology outcome in this sample; clinical symptoms are not consistently related to the

endophenotype (Tsuang et al., 2001). It is difficult to say, however, whether those influences were necessarily associated with schizophrenia-related pathologies.

Do these scores matter, however, when it comes to clinical significance? Although individuals in the SocAnh group had higher scores across all schizophrenia-spectrum indices, their overall mean dimensional scores were between one and two, whereas one must meet four (for schizoid and paranoid personality disorders) or five (for schizotypal personality disorder) criteria to cross the DSM-IV-TR diagnostic threshold for the disorders. Perhaps this can be accounted for by the fact that "pure" groups were used. An empirical strategy was used (i.e., including at-risk individuals with only one scale elevation) to rule out a potential confound, but this method may only serve to confirm that homogenous groups do not evidence greater pathology. That is (as similarly mentioned earlier with regard to the neurocognitive data), perhaps it is the combination of both positive and negative schizotypy characteristics that leads to outcomes falling within the diagnostic threshold range. Thus, the degree of schizotypy found in the at-risk participants in the current study may not accurately represent true population levels.

The functional impact of dimensional pathology, however, may be reflected in the negative association between GAF scores and schizophrenia-spectrum personality characteristics. However, diagnostic-threshold considerations aside, it should be noted that in the Kwapil (1998) sample, many individuals did not meet diagnostic criteria at baseline, and symptoms at baseline were predictive of later schizophrenia-spectrum diagnoses. Given the young age of this sample, only time will tell if they develop more schizophrenia-spectrum characteristics as they age.

Can the Schizotaxic Individual Be Identified by Neurocognitive Measures?

These results should not be interpreted to mean that neurocognitive indices cannot be useful in the identification of individuals at-risk for schizophrenia. Rather, they speak to the fallibility of the Chapman scales and/or the strategy of the current study in reliably identifying individuals with not only the behavioral signs of the phenotype, but the neurocognitive characteristics of the endophenotype. Presumably, both are useful in determining schizophrenia risk. Perhaps a different approach would be useful; neurocognitive indices should be used to identify a putative at-risk group, and then the Chapman scales can be used as an adjunct to enhance accurate prediction.

Long-term follow-up will be essential in determining the neurocognitive deficits of putative schizotypes. As mentioned in an earlier section, many of the individuals determined to be at-risk in this study may show a diminished neurocognitive and clinical profile several years from now. Taking into account the above-presented studies which demonstrated cognitive decline after the development of psychosis, this appears to be a genuine possibility. Thus, the Chapman scale method of identifying schizotypes should not be dismissed. The possibility remains that neurocognitive indices can reliably predict the development of schizophrenia and related disorders in Chapman-scale identified putatively at-risk individuals.

The examination of neurocognition has shown to be effective in separating non-Chapman scale identified putative schizotypes from control participants. Hawkins and colleagues (2004) used a non-Chapman approach in identifying schizotypes and examined their cognitive performance. Clinicians conferred prodromal status on the basis of attenuated positive-like characteristics, and putatively prodromal participants

performed significantly worse on measures of attention, working memory, processing speed, fine motor functioning, spatial performance, memory, and executive functioning than did controls. In addition, they performed better than did schizophrenia patients in many of these same domains, lending to the authors' conclusion that early identification efforts could halt the diminishing of cognitive abilities over time in individuals at risk for schizophrenia. This study identified at-risk individuals on the basis of positive characteristics rather than social anhedonia (a negative one), but it speaks to the utility of identifying individuals while they are expressing enough symptomatology to warrant clinical treatment.

## Future Directions

The findings in this study, which showed that groups putatively at-risk for the development of schizophrenia and related disorders do not differ on neurocognitive indices, suggest several interesting directions for future research.

First is a methodological consideration. Long-term follow-up seems essential for this type of study, as neurocognitive functioning may deteriorate after the expression of more frank psychotic symptomatology. The data from the current investigation were drawn from the first phase of a three-year longitudinal study that will employ some of the same neurocognitive measures. It will be interesting to see how the samples fare as they enter early adulthood.

As another methodological direction, the possibility of ensuring comparability across studies should be explored. That is, it is difficult to compare existing investigations because the neurocognitive batteries employed differ across studies. With a body of research that consistently uses the same measures, one can avoid equivocation

about conclusions on the basis of study comparability. Standardization has the potential to be a contentious issue, as there are benefits and drawbacks to any measure and each researcher may have particular favorites, but perhaps these issues could be overcome in the interest of illuminating who is most at risk for schizophrenia.

As an additional consideration, these results highlight the importance of using mixed-race samples in future studies to ensure the generalizability of results beyond Caucasians. The current data demonstrate that individuals from different racial groups may not be comparable to each other on neurocognitive measures. Demographically-adjusted norms may be helpful in illuminating true group differences irrespective of racial differences.

Besides methodological improvements, several interesting studies could follow from this one. For instance, this study did not take into consideration environmental factors that can impact neurocognitive functioning. Walker and Diforio (1997) posit that external stressors activate the hypothalamic-pituitary-adrenal (HPA) axis, which in turn augments dopamine synthesis and damages the hippocampus. Presumably, this could lead to neurocognitive dysfunction. External stressors in individuals at risk for schizophrenia may have the same impact on the brain as in individuals with expressed schizophrenia. In fact, it is possible that a high level of environmental stress and its resultant neurocognitive sequelae could potentiate psychosis due to a reduction in the ability to efficiently process and filter stimuli. Indeed, it has been proposed that deficits in neural synchrony result in both the positive and negative symptoms of schizophrenia (Andreasen, 1999), and this could be impacted by the influence of stress on the brain.

Thus, a measure of stressful events would be an important addition to a follow-up investigation.

Related to "true-to-life" experiences such as stress, another interesting direction would be to examine the relation of neurocognitive domains to functional capacity in real life. Individuals may perform differently on measures in the laboratory than they do on tasks requiring similar information processing requirements in daily life. This could be related to neurocognitive deficits, attenuated symptoms, or both. For instance, how does a social anhedonic individual perform on complex tasks in a workplace that requires social interactions, which that individual may find aversive? In this case, neurocognitive deficits, which may be mild or non-detectable on laboratory measures, may interact with symptom presentation such that there is a decrement in observable cognitive functioning. Existing research supports this conjecture; premorbid social adjustment in one study predicted symptom presentation, while academic performance was only predictive of later intelligence (Allen, Kelly, Miyatake, Gurklis, & VanKammen, 2001). Besides being related to clinical presentation, premorbid social adjustment is also related to later neurocognitive deficits (Silverstein, Maurdefteros, & Close, 2002), so a combined exploration of social and neurocognitive factors may prove to be especially fruitful.

Finally, other directions add more neural considerations into the mix. For instance, there have been no neuroimaging studies of putative schizotypes, but no neurocognitive differences among groups were found in the current study. Given that neurocognitive deficits have been linked to structural anomalies (Mozley, Gur, Gur, Mozley, & Alavi, 1996; Gur, Cowell, Turetsky, Gallacher, Cannon, et al., 1998), it might be prudent to first establish that neurocognitive dysfunction exists in putative schizotypes

before embarking on potentially expensive and time-consuming neuroimaging research. Neuroimaging studies of children at-risk for schizophrenia (as identified by positive family history of schizophrenia) have found that such children demonstrate anomalies such as neural asymmetries and reduction in amygdalar volume as compared to control children (Hendren, Hodde-Vargas, Yeo, & Vargas, 1995). It would follow that if individuals identified by the Chapman scales were at-risk for schizophrenia, they would also show some structural anomalies similar to individuals with schizophrenia. Extant research informs this hypothesis; Gur and colleagues (1998) found that volume reduction in the frontal and temporal lobes was predictive of fewer improvements in negative symptoms. The inclusion of neurocognitive measures may help to elucidate whether individuals with structural or metabolic anomalies will show cognitive difficulties before schizophrenia is expressed, and could help to determine if such measures are useful at the presumed premorbid stage.

## Conclusions

This study has shown that while individuals identified as socially anhedonic by the Chapman scales do show increased schizophrenia-spectrum characteristics as compared to individuals with perceptual aberrations/magical ideations and controls, they do not have a unique neurocognitive signature.

This information has important implications for future research with regard to the identification of individuals at-risk for schizophrenia, as well as implications for methods for studying those individuals. Although this study met with negative results with regard to neurocognitive characteristics of the presumably at-risk individuals, it is important that the group thought to be most likely to be at-risk (social anhedonic individuals)

demonstrated more schizophrenia-related clinical characteristics, which may aid in identifying at-risk individuals before schizophrenia is expressed. The prophylactic identification of individuals at-risk for schizophrenia remains a daunting, but important, cause.

TABLES

Table 1

*Demographic Characteristics Among Social Anhedonia (SocAnh), Perceptual Aberration/Magical Ideation (PerMag) and Control Groups*

| Characteristic | SocAnh (n = 33) | PerMag (n = 28) | Control (n = 89) | $\chi^2$ | p |
|---|---|---|---|---|---|
| Race | | | | 15.14 | .019 |
| Asian | 0 | 1 | 3 | | |
| Black | 19 (57.5%) | 8 (28.5%) | 36 (40%) | | |
| Caucasian | 12 | 13 | 43 | | |
| Other | 2 | 7 | 4 | | |
| Sex | | | | 0.95 | ns |
| Female | 20 (60%) | 14 (50%) | 47 (52.8%) | | |
| Male | 13 | 15 | 39 | | |

Table 2

*Neurocognitive Test Results Among Social Anhedonia (SocAnh), Perceptual Aberration/Magical Ideation (PerMag) and Control Groups*

| Domain | SocAnh (n = 33) M (SD) | PerMag (n = 28) M (SD) | Control (n = 89) M (SD) | F | p |
|---|---|---|---|---|---|
| General Intellect | | | | 2.08 | ns |
| Vocabulary [a,b] | 13.33 (3.56) | 14.11 (2.95) | 13.24 (3.17) | | |
| Block Design [a,b] | 9.85 (2.93) | 11.43 (2.75) | 11.22 (2.90) | | |
| General Memory | | | | 0.86 | ns |
| LogMem I [a] | 8.91 (2.85) | 9.03 (3.51) | 9.71 (3.43) | | |
| LogMem II [a] | 9.42 (3.26) | 10.29 (2.94) | 10.09 (3.23) | | |
| VisRep I [a,b] | 10.88 (2.94) | 12.25 (3.00) | 11.35 (2.93) | | |
| VisRep II [a,b] | 12.15 (3.08) | 12.82 (3.79) | 13.07 (3.22) | | |
| VisRep Recog [a] | 10.94 (2.40) | 11.18 (3.09) | 11.38 (2.38) | | |
| Working Memory | | | | 0.63 | ns |
| Digit Span | 10.67 (2.85) | 11.50 (2.70) | 10.78 (2.95) | | |
| Spatial Span [a,c] | 10.70 (2.49) | 11.50 (2.25) | 10.99 (2.66) | | |
| LNS [d,e] | 10.42 (2.78) | 10.71 (2.23) | 10.73 (2.67) | | |
| Sustained Attention | | | | 1.61 | ns |
| DSCPT d' [a] | 1.99 (1.04) | 2.02 (.90) | 2.29 (.91) | | |

*Note.* Superscripts indicate a significant difference at the $p < .05$ level by Tukey's LSD as follows: [a] Caucasian > African-American; [b] Caucasian > Another Race; [c] Asian >African-American; [d] Asian > Caucasian; [e] Asian > Another Race.

LogMem I = Logical Memory I; LogMem II = Logical Memory II; VisRep I = Visual Reproduction I; VisRep II = Visual Reproduction II; VisRep Recog = Visual Reproduction Recognition; DSCPT d' = Degraded Stimulus Continuous Performance Task d'.

Table 3

*Clinical Pathology (Axis I) Diagnostic Characteristics Among Social Anhedonia (SocAnh), Perceptual Aberration/Magical Ideation (PerMag) and Control Groups*

| Characteristic | SocAnh (n = 33) | PerMag (n = 28) | Control (n = 89) | $\chi^2$ | p |
|---|---|---|---|---|---|
| Individuals with Lifetime Major Depressive Disorder | 10 (30%) | 5 (18%) | 10 (11%) | 5.91 | ns |
| Individuals with Lifetime Substance Use Disorder | 2 (3%) | 8 (29%) | 16 (18.3%) | 5.39 | ns |
| Individuals with Any Axis I Diagnosis | 12 (36%) | 12 (43%) | 23 (26%) | 14.49 | ns |

*Note.* The number of individuals with Lifetime Major Depressive Disorder and Lifetime Substance Use Disorders may exceed the number of individuals with any Axis I diagnosis because some individuals had more than one diagnosis.

Table 4

*Schizophrenia-Spectrum Diagnoses Among Social Anhedonia (SocAnh), Perceptual Aberration/Magical Ideation (PerMag), and Control Groups*

| | Number of Diagnoses | | | | |
|---|---|---|---|---|---|
| Diagnosis | SocAnh (n = 33) | PerMag (n =28) | Control (n = 89) | $\chi^2$ | p |
| Schizoid | 6 (18%) | 0 (0%) | 2 (2.2%) | 13.64 | .001 |
| Schizotypal | 1 (3%) | 0 (0%) | 1 (1.1%) | 1.10 | ns |
| Paranoid | 6 (18%) | 0 (0%) | 3 (3.3%) | 11.21 | .007 |

*Note.* The SocAnh group had significantly more Schizoid and Paranoid personality disorder diagnoses than did the other two groups.

Table 5

*Schizophrenia-Spectrum IPDE Dimensional Scores Among Social Anhedonia (SocAnh), Perceptual Aberration/Magical Ideation (PerMag) and Control Groups*

| Characteristic | SocAnh (n = 33) M (SD) | PerMag (n =28) M (SD) | Control (n = 89) M (SD) | F | p |
|---|---|---|---|---|---|
| Schizoid | 1.93 (2.33)[a] | .38 (.73)[b] | .37 (.87) | 18.02 | .000 |
| Schizotypal | 1.40 (1.54) [a] | .76 (1.21)[b] | .50 (1.03) | 6.69 | .002 |
| Paranoid | 1.30 (2.04) [a] | .34 (.70)[b] | .60 (1.03) | 5.05 | .008 |
| IPDE Total Score | 4.64 (4.72) [a] | 1.48 (1.62)[b] | 1.48 (2.19) | 15.42 | .000 |

*Note.* Means with different superscripts are significantly different at p <.05.

Table 6

*Correlations Between Neurocognitive Variables and IPDE Dimensional Scores: Social Anhedonia Group (N = 33)*

| | Schizoid | Paranoid | Schizotypal | Total IPDE Score |
|---|---|---|---|---|
| Vocabulary | -.09 | .01 | .32 | -.10 |
| Block Design | .04 | -.10 | .11 | -.01 |
| LogMemI | .07 | .17 | -.15 | .06 |
| LogMemII | .02 | .23 | -.12 | .07 |
| VisRep I | .12 | .11 | .01 | .11 |
| VisRep II | -.16 | .03 | -.18 | -.12 |
| VisRep Recog | -.25 | .02 | -.42* | -.26 |
| Digit Span | .20 | .05 | .07 | .14 |
| Spatial Span | -.20 | -.21 | -.34 | -.30 |
| LNS | -.06 | .10 | -.21 | -.05 |
| DSCPT d’ | .27 | -.14 | .12 | .12 |

*Note.* Asterisks indicate a significant difference at the $p < .05$ level by the Pearson’s $r$ statistic.

LogMem I = Logical Memory I; LogMem II = Logical Memory II; VisRep I = Visual Reproduction I; VisRep II = Visual Reproduction II; VisRep Recog = Visual Reproduction Recognition; DSCPT d’ = Degraded Stimulus Continuous Performance Task d’.

Table 7

*Correlations Between Neurocognitive Variables and IPDE Dimensional Scores: Perceptual Aberration/Magical Ideation Group (N = 28)*

| | Schizoid | Paranoid | Schizotypal | Total IPDE Score |
|---|---|---|---|---|
| Vocabulary | -.07 | -.03 | -.11 | -.13 |
| Block Design | -.19 | -.08 | -.19 | -.26 |
| LogMemI | .27 | -.19 | .15 | .16 |
| LogMemII | .12 | -.27 | .01 | -.05 |
| VisRep I | .25 | -.47* | -.15 | -.20 |
| VisRep II | .21 | -.51* | .13 | -.02 |
| VisRep Recog | .13 | .07 | .15 | .20 |
| Digit Span | -.05 | -.08 | -.22 | -.22 |
| Spatial Span | .04 | -.15 | -.35 | -.31 |
| LNS | -.18 | -.15 | -.21 | -.31 |
| DSCPT d’ | -.04 | .06 | -.04 | -.02 |

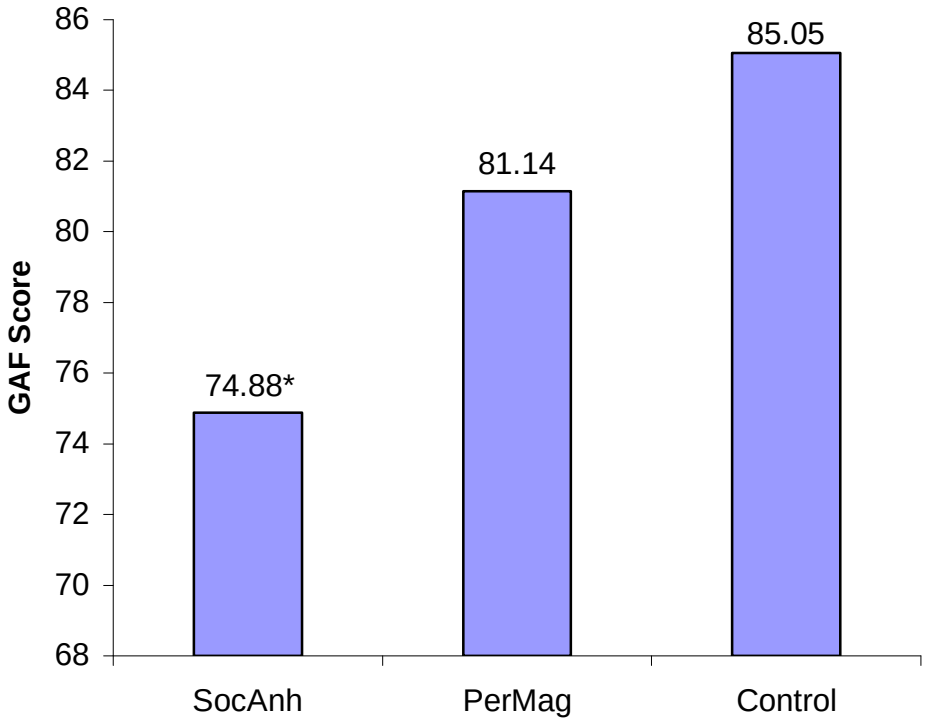
*Note.* Asterisks indicate a significant difference at the $p < .05$ level by the Pearson’s $r$ statistic.

LogMem I = Logical Memory I; LogMem II = Logical Memory II; VisRep I = Visual Reproduction I; VisRep II = Visual Reproduction II; VisRep Recog = Visual Reproduction Recognition; DSCPT d’ = Degraded Stimulus Continuous Performance Task d’.

FIGURE

Figure 1

*Global Assessment of Functioning (GAF) Scores Among Social Anhedonia (SocAnh; N =33), Perceptual Aberration/Magical Ideation (PerMag; N = 28), and Control (N = 89) Groups*




*Note.* Asterisk indicates significant difference at the $p < .05$ level.

References


Albus, M., Hubmann, W., Scherer, J., Dreikorn, B., Hecht, S., et al., (2002). A prospective 2-year follow-up study of neurocognitive functioning in patients with first-episode schizophrenia. *European Archives of Psychiatry and Clinical Neuroscience, 252* (6), 262-267.

Alemon, A., Hijman, R., deHaan, E., & Kahn, R. (1999). Memory impairment in schizophrenia: A meta-analysis. *American Journal of Psychiatry, 156,* 1358-1366.

Allen, D., Kelly, M., Miyatake, R., Gurklis, J., & van Kammen, D. (2001). Confirmation of a two-factor model of premorbid adjustment in males with schizophrenia. *Schizophrenia Bulletin, 27* (1), 39-46.

Allen, J., Chapman, L., & Chapman, J. (1987). Cognitive slippage and depression in Hypothetically psychosis-prone college students. *Journal of Nervous and Mental Disorders, 175* (6), 347-353.

Allen, J., Chapman, L., Chapman, J., Vuchetich, J., & Frost, L. (1987). Prediction of psychotic-like symptoms in hypothetically psychosis-prone college students. *Journal of Abnormal Psychology, 96* (2), 83-88.

Allen, J. & Schuldberg, D. (1989). Positive thought disorder in a hypothetically psychosis-prone population. *Journal of Abnormal Psychology, 98,* (4), 491-494.

American Psychiatric Association (1994). Diagnostic and Statistical Manual of Mental Disorders (4th ed.). Washington, D.C.: Author.

Amminger, G., Edwards, J., Brewer, W., Harrigan, S., & McGorry, P. (2002). Duration of untreated psychosis and cognitive deteriorating in first-episode schizophrenia. *Schizophrenia Research, 54* (3), 223-230.

Andreasen, N., Rezai, K., & Alliger, R. (1992). Hypofrontality in neuroleptic-naïve patients and in patients with chronic schizophrenia: Assessment with xenon 133 single-photon emission computed tomography and the Tower of London. *Archives of General Psychiatry, 49* (1), 943-958.

Ang, Y. & Tan, H. (2004). Academic deterioration prior to first episode schizophrenia in young Singaporean males. *Psychiatry Research, 121* (3), 303-307.

Asarnow, R., Neuchterlein, K., & Fogelson, D. (2001). Schizophrenia and schizophrenia-spectrum disorders in the first-degree relatives of children with schizophrenia: The UCLA family study. *Archives of General Psychiatry, 58* (6), 581-588.

Baddeley, A. (1986). *Working memory.* New York: Oxford University Press.

Bailey, B., West, K., Widiger, T., & Freiman, K. (1993). The convergent and discriminant validity of the Chapman Scales. *Journal of Personality Assessment, 61* (1), 121-135.

Balogh, D. & Merrit, R. (1985). Susceptibility to Type A backward pattern masking among hypothetically psychosis-prone college students. *Journal of Abnormal Psychology, 94*(3), 377-383.

Basso, M., Nasrallah, H., Olson, S., & Bornstein, R. (1998). Neuropsychological characteristics of negative, disorganized, and psychotic symptoms in schizophrenia. *Schizophrenia Research, 31,* 99-111.

Battaglia, M., Gasperini, M., Sciuto, S., Scherillo., P., Diaferia, A., & Belodi, M. (1991). Psychiatric disorders in family members of schizotypal subjects. *Schizophrenia Bulletin, 17*(4), 659-668.

Berenbaum, H. & McGrew, J. (1993). Familial resemblace of schizotypic traits. *Psychological Medicine, 23* (2), 327-333.

Blanchard, J. (1998). Hedonic capacity. In W.F. Flack and J.D. Laird (Eds.), *Emotions in Psychopathology: Theory and Research* (pp. 336-352). New York: Oxford University Press.

Blanchard, J. & Brown, S. (1999). Social anhedonia and other indicators of schizotypy: taxometric considerations and group comparisons. Paper presented at the annual meeting of the Society for Research in Psychopathology, Montreal, Canada.

Blanchard, J., Bellack, A., & Mueser, K. (1994). Affective and social-behavioral correlates of physical anhedonia and social anhedonia in schizophrenia. *Journal of Abnormal Psychology, 103*(4), 719-728.

Blanchard, J., Gangestad, S., Brown, S., & Horan, W. (2000). Hedonic capacity and schizotypy revisited: A taxometric analysis of social anhedonia. *Journal of Abnormal Psychology, 109*(1), 87-95.

Blanchard, J., Horan, W., & Brown, S. (2001). Diagnostic differences in social anhedonia: A longitudinal study of schizophrenia and major depressive disorder. *Journal of Abnormal Psychology, 110* (3), 1-9.

Blanchard J., Mueser, K., & Bellack, A. (1998). Anhedonia, positive and negative affect, and social functioning in schizophrenia. *Schizophrenia Bulletin, 24*(4), 413-424.

Blanchard, J. & Neale, J. (1994). The neuropsychological signature of schizophrenia: Generalized or differential deficit? *American Journal of Psychiatry, 151*(1), 40-48.

Braff, D. (1993). Information processing and attention dysfunctions in schizophrenia. *Schizophrenia Bulletin, 19* (2), 233-259.

Brekke, J., Raine, A., Ansel, M., Lencz, T., & Bird, L. (1997). Neuropsychological and psychophysiological correlates of psychosocial functioning in schizophrenia. *Schizophrenia Bulletin, 23*(1), 19-28.

Bryson, G., Whelahan, H., & Bell, M. (2001). Memory and executive function impairments in deficit syndrome schizophrenia. *Psychiatry Research, 102* (1), 29-37.

Buchanan, R., Kirkpatrick, B., & Heinrichs, D. (1990). Clinical correlates of the deficit syndrome of schizophrenia. *American Journal of Psychiatry, 147* (3), 290-294.

Buchanan, R. & Carpenter, W. (1994). Domains of psychopathology: An approach to the reduction of heterogeneity in schizophrenia. *Journal of Nervous and Mental Disease, 182* (4), 193-204.

Buchanan, R., Strauss, M., Breier, A, Kirkpatrick, B., and Carpenter, W. (1997). Attentional impairments in deficit and nondeficit forms of schizophrenia. *American Journal of Psychiatry, 154* (3), 363-370.

Buchsbaum, M., Neuchterlein, K., Haier, R., Wu, J., Sicotte, N., Hazlett, E. et al. (1990). Glucose metabolic rate in normals and schizophrenics during the continuous performance task assessed by positron emission tomography. *British Journal of Psychiatry, 156,* 216-227.

Carter, C., Robertson, L., Nordahl, T., Chaderjian, M., Kraft, L., & O'Shara-Celaya, L. (1996). Spatial working memory deficits and their relation to negative symptoms in unmedicated schizophrenia patients. *Biological Psychiatry, 40,* 930-932.

Cavanagh, J., VanBeck, M., & Blackwood, D. (2001). Case control study of neurocognitive functioning in patients with bipolar disorder: An association with mania. *British Journal of Psychiatry, 108,* 320-326.

Chapman, L. & Chapman, J. (1978). The measurement of differential deficit. *Journal of Psychiatric Research, 14* (1, Suppl 4), 303-311.

Chapman, L., Edell, W., & Chapman, J. (1980). Physical anhedonia, perceptual aberration, and psychosis proneness. *Schizophrenia Bulletin, 6* (4), 639-653.

Chapman, J. & Chapman, L. (1987). Handedness of hypothetically psychosis-prone subjects. *Journal of Abnormal Psychology, 96*(2), 89-93.

Chapman, L., Chapman, J., Kwapil, T., Eckblad, M., & Zinser, M. (1994). Putatively psychosis-prone subjects ten years later. *Journal of Abnormal Psychology, 103,* 171-183.

Chapman, L., Chapman, J. and Raulin, M. (1978). Body image aberration in schizophrenia. *Journal of Abnormal Psychology, 87,* 399-407.

Chen, W., Liu, S., Change, C., Lien, Y., Chang, Y., & Hwu, H. (1998). Sustained attention deficits and schizotypal personality features in nonpsychotic relatives of schizophrenia patients. *American Journal of Psychiatry, 155*(9), 1214-1220.

Clark, L., Iverson, S. & Goodwine, G. (2002). Sustained attention deficit in bipolar disorder. *British Journal of Psychiatry, 180,* 313-319.

Coleman, M., Levy, D., Lenzenweger, M., Holzman, P. (1996). Thought disorder, perceptual aberrations, and schizotypy. *Journal of Abnormal Psychology, 105*(3), 469-473.

Conklin, H., Curtis, C., Katsanis, J., & Iacono, W. (2000). Verbal working memory impairment in schizophrenics and their first-degree relatives: Evidence from the digit span task. *American Journal of Psychiatry, 157,* 275-277.

Cornblatt, B. & Erlenmeyer-Kimling, L. (1986). Sustained attention in children at risk for schizophrenia: Findings with two visual continuous performance tests in a new sample. *Journal of Abnormal Child Psychology, 14* (3), 365-385.

Cornblatt, B., Lenzenweger, M., & Erlenmeyer-Kimling, L. (1989). The Continuous Performance Test, Identical Pairs version: II. Contrasting attentional profiles in schizophrenic and depressed patients. *Psychiatry Research, 29* (1), 65-86.

Cornblatt, B. & Keilp, J. (1994). Impaired attention, genetics, and the pathophysiology of schizophrenia. *Schizophrenia Bulletin, 20* (1), 31-46.

Davidson, M., Reichenberg, A., Rabinowitz, J., Weiser, M., Kaplan, Z., & Mark, M. (1999). Behavioral and intellectual markers for schizophrenia in apparently healthy male adolescents. *American Journal of Psychiatry, 156,* 1328-1335.

Dickerson, L., Diaz, M., & Kwapil, T. (2002). Two-year follow-up assessment of social anhedonia and control participants. Poster presented at the annual meeting of the Society for Research in Psychopathology, Madison, Wisconsin

DiForio, D., Walker, E., & Kestler, L. (2000). Executive functioning in adolescents with schizotypal personality disorder. *Schizophrenia Research, 42,* 125-134.

Downhill, J., Buchsbaum, M., Wei, T., Spiegel-Cohen, J., Hazlett, E., Haznedar, M., Silverman, J., & Siever, C. (2000). Shape and size of the corpus callosum in schizophrenia and schizotypal personality disorder. *Schizophrenia Research, 42,* 193-208.

Eckblad, M. and Chapman, L. (1983). Magical ideation as an indicator of schizotypy. *Journal of Consulting and Clinical Psychology, 51,* 215-225.

Eckblad, M., Chapman, L., Chapman, J., and Mishlove, R. (1982). The revised social anhedonia scale. Unpublished test.

Erlenmeyer-Kimling, L. & Cornblatt, B. (1987). The New York High-Risk Project: A follow-up report. *Schizophrenia Bulletin, 13* (3), 451-461.

Erlenmeyer-Kimling, L., Rock, D., & Roberts, S. (2000). Attention, memory, and motor skills as childhood predictors of schizophrenia-related psychoses: The New York high-risk project. *American Journal of Psychiatry, 157* (9), 1416-1422.

Eysenck, H. J., & Eysenck, S. B. G. (1976). Psychoticism as a dimension of per sonality. London: Hodder.

Faraone, S., Green, A., Seidman, L., and Tsuang, M. (2001). "Schizotaxia": Clinical implications and new directions for research. *Schizophrenia Bulletin, 27*(1), 1-18.

Faraone, S., Kremen, W., Lyons, M., Pepple, J., Seidman, L., and Tsuang, M. (1995). Diagnostic accuracy and linkage analysis: how useful are schizophrenia spectrum phenotypes? *American Journal of Psychiatry, 152,*1286-1290.

Faraone, S., Seidman, L., Kremen, W., Toomey, R., Pepple, J., & Tsuang, M. (1999). Neuropsychological functioning among non-psychotic relatives of schizophrenia patients: A four year follow-up study. *Journal of Abnormal Psychology, 108*(1), 176-181.

Faraone, S., Seidman, L., Kremen, W., Toomey, R., Pepple, J., & Tsuang, M. (2000). Neuropsychological functioning among the non-psychotic relatives of schizophrenia patients: The effect of genetic loading. *Biological Psychiatry, 48,* 120-126.

Faraone, S., Green, A., & Seidman, L. (2001). "Schizotaxia": Clinical implications and new directions for research. *Schizophrenia Bulletin, 27* (1), 1-18.

Farmer, C. O'Donnell, B., Niznikiewicz, M., Voglmaier, M., McCarley, R., & Shenton, M. (2000). Visual perception and working memory in schizotypal personality disorder. *American Journal of Psychiatry, 157,* 781-786.

Ferrier, I. & Thompson, J. (2000). Cognitive impairment in bipolar affective disorder: Implications for the bipolar diathesis. *British Journal of Psychiatry, 180,* 293-295.

Franke, P. Maier, W., Hah, C., & Klingler T. (1992). The Wisconsin Card Sorting Test: An indicator of vulnerability to schizophrenia? *Schizophrenia Research, 6,* 243-249.

Fujioka, T. & Chapman, L. (1984). Comparison of the 2-7-8 MMPI profile and the Perceptual Aberration-Magical Ideation Scales in identifying hypothetically psychosis-prone college students. *Journal of Consulting and Clinical Psychology, 52*(3), 458-467.

Fuller, R., Nopolous, P., Arndt, S. (2002). Longitudinal assessment of premorbid cognitive functioning in patients with schizophrenia through examination of standardized scholastic test performance. *American Journal of Psychiatry, 159* (7), 1183-1189.

Gilvarry, C., Russell, A., Hemsley, D., & Murray, R. (2001). Neuropsychological performance and spectrum personality traits in the relatives of patients with schizophrenia and affective psychosis. *Psychiatry Research, 101,* 89-100.

Gold, J. (1998). Schizophrenia and intellectual decline. *American Journal of Psychiatry, 155* (11), 1633-1634.

Gold, J., Randolph, C., Carpenter, C., Goldberg, T., & Weinberger, D. (1992). The performance of patients with schizophrenia on the WMS-R. *The Clinical Neuropsychologist, 6* (4), 367-373.

Goldberg, T., Saint-Cyr, J., & Weinberger, D. (1990). Assessment of procedural learning and problem solving in schizophrenic patients by Tower of Hanoi type tasks. *Journal of Neuropsychiatry and Clinical Neurosciences, 2* (2), 165-173.

Golden, R. & Meehl, P. (1979). Schizoidia scale. Unpublished self-report measure.

Goldman-Rakic, P. (1991). Prefrontal cortical dysfunction in schizophrenia: The relevance of working memory. In B. Carroll (Ed.), *Psychopathology and the Brain*. New York: Raven Press.

Goldman-Rackic, P. (1996). The functional parcellation of the dorsolateral prefrontal cortex and the heterogeneous facets of schizophrenia. In S. Matthysse, D. Levy, J. Kagan, & F. Benes (Eds.), *Psychopathology: The Evolving Science of Mental Disorders*(pp. 7 -33). New York: Cambridge University Press.

Goldman-Rakic, P. and Selemon, L (1997). Functional and anatomical aspects of prefrontal pathology in schizophrenia. *Schizophrenia Bulletin, 23*(3), 437-458.

Gooding, D. (1999). Antisaccade task performance in questionnaire identified schizotypes. *Schizophrenia Research, 35,* 157-166.

Gooding, D. & Tallent, K. (2001). The association between antisaccade task and working memory task performance in schizophrenia and bipolar disorder. *Journal of Nervous and Mental Disease, 189,* 8-16.

Gooding, D. & Tallent, K. (2002). Letter to the editor. *Psychiatric Medicine, 32,* 181.

Gooding, D., Davidson, R., Putnam, K., & Tallent, K. (2002). Normative emotion-modulated startle response in individuals at risk for schizophrenia-spectrum disorders. *Schizophrenia Research, 57*(1), 109-120.

Gooding, D., Kwapil T., & Tallent, K. (1999). Wisconsin Card Sorting Test deficits in schizotypic individuals. *Schizophrenia Research, 40,* 210-209.

Gooding, D., Grabowski, J., & Hendershot, C. (2000). Fixation stability in Schizophrenia, bipolar disorder, and control subjects. *Psychiatry Research, 97,*119-128.

Gooding, D., Miller, M., & Kwapil, T. (2000). Smooth pursuit eye tracking and visual fixation in psychosis prone individuals. *Psychiatry Research, 93,* 41-54.

Gooding, D., Tallent, K., & Hiegyi, J. (2001). Cognitive slippage in schizotypic individuals. *Journal of Nervous and Mental Disease, 189,*750-756.

Gottesman, I. (1991). *Schizophrenia genesis: The origins of madness.* San Francisco: Freeman.

Gottesman, I. & Shields, J. (1972). Genetics and schizophrenia: A twin study vantage point. Oxford, England: Academic Press.

Gottesman, I. & Erlenmeyer-Kimling, L. (2001). Family and twin strategies as a head start in defining prodromes and endophenotypes for hypothetical early interventions in schizophrenia. *Schizophrenia Research, 51,* 93-102.

Grove, W. (1982). Psychometric detection of schizotypy. *Psychological Bulletin, 92* (1), 27-38.

Gruzelier, J., Seymour, K., Wilson, L., Jolley, A., et al. (1988). Impairments on neuropsychologic tests of temporohippocampal and frontohippocampal functions and word fluency in remitting schizophrenia and affective disorders. *Archives of General Psychiatry, 45* (7), 623-629.

Gualtieri, C. (1995). The contribution of frontal lobes to a theory of psychopathology. In J. Ratey (Ed.), *Neuropsychiatry of Personality Disorders,* (pp. 149-171). Cambridge, MA: Blackwell Science.

Gur, R.E., Cowell, P., Turetsky, B., Gallacher, F., Cannon, T., et al. (1998). A follow-up magnetic resonance imaging study of schizophrenia: Relationship of neuroanatomical changes to clinical and neurobehavioral measures. *Archives of General Psychiatry, 55* (2), 145-152.

Haberman, M., Chapman, L, Numbers, J., & McFall, R. (1979). Relation of social Competence to scores on two scales of psychosis proneness. *Journal of Abnormal Psychology, 88*(6), 675-677.

Hazlett, E., Dawson, M., Filion, D., Schell, A., & Neuchterlein, K. (1997). Autonomic orienting and the allocation of processing resources in schizophrenia patients and putatively at-risk individuals. *Journal of Abnormal Psychology, 106*(2), 171-181.

Hendren, R., Hodde-Vargas, J., Yeo, R., Vargas, L., et al. (1995). Neuropsychophysiological study of children at risk for schizophrenia: A preliminary report. *Journal of the American Academy of Child & Adolescent Psychiatry, 34* (1), 1284-1291.

Hoff, A., Riordan, H, O'Donnell, D., Stritzke, P., et al. (1992). Anomalous lateral sulcus asymmetry and cognitive function in first-episode schizophrenia. *Schizophrenia Bulletin, 18* (2), 257-272.

Horan, W., Blanchard, J., Gangestad, S., & Kwapil, T. The psychometric detection of schizotypy: Do putative schizotypy indicators identify the same latent class? Unpublished manuscript.

Jeyakumar, S., Warriner, E., Raval, V. & Ahmad, R. (2004). Balancing the need for reliability and time efficiency: Short forms fo the Wechsler Adult Intelligence Scale-III. *Educational and Psychological Measurement, 64* (1), 71-87.

Jones, L., Cardno, A., Sanders, R., Owen, M., & Williams, J. (2001). Sustained and selective attention as measures of genetic liability to schizophrenia. *Schizophrenia Research, 48,* 263-272.

Katsanis, J., Iacono, W., & Beiser, M. (1990). Anhedonia and perceptual aberrations in first episode psychotic patients and their relatives. *Journal of Abnormal Psychology, 99* (2), 202-206.

Katsanis, J., Iacono, W., & Beiser, M. (1992). Clinical correlates of anhedonia and perceptual aberration in first-episode patients with schizophrenia and affective disorder. *Journal of Abnormal Psychology, 101* (1), 184-191.

Keefe, R., Silverman, J., Mohs, R., Siever, L., Harvey, P., Friedman, L., et al. (1997). Eye tracking, attention, and schizotypal symptoms in nonpsychotic relatives of patients with schizophrenia. *Archives of General Psychiatry, 56,* 169-176.

Kendler, K. (1988). Familial aggregation of schizophrenia and schizophrenia spectrum disorders: Evaluation of conflicting results. *Archives of General Psychiatry, 45* (4), 377-383.

Kendler, K. & Gardner, C. (1997). The risk for psychiatric disorders in relatives of schizophrenia and control probands: A comparison of three independent studies. *Psychiatric Medicine, 27,* 411-419.

Keri, S., Kelemen, O., Benedek, G., & Ianka, Z. (2001). Different trait markers for schizophrenia and bipolar disorder: A neurocognitive approach. *Psychiatric Medicine, 31,* 915-922.

Kerns, J. & Berenbaum, H. (2000). Aberrant semantic and affective processing in people at risk for psychosis. *Journal of Abnormal Psychology, 4,* 728-732.

Kinney, D., Levy, D., Yurgelun-Todd, D., Tramer, S., Holzman, P. (1998). Inverse relationship of perinatal complications and eye tracking dysfunction in relatives of patients with schizophrenia: Evidence for a two-factor model. *American Journal of Psychiatry, 155* (7), 976-978.

Kurtz, M., Raglund, J., Bilker, W., Gur, R., & Gur, R. (2001). Comparison of the continuous performance task with and without working memory demands in healthy controls and patients with schizophrenia. *Schizophrenia Research, 48,* 307-316.

Kwapil, T. (1998). Social anhedonia as a predictor of the development of schizophrenia-spectrum disorders. *Journal of Abnormal Psychology, 107* (4), 558-565.

Kwapil, T., Chapman, L., & Chapman, J. (1999). Validity and usefulness of the Wisconsin Manual for assessing psychotic-like experiences. *Schizophrenia Bulletin, 25* (2), 363-375.

Kwapil, T., Crump, R., & Pickup, D. (2002). Assessment of psychosis proneness in African-American college students. *Journal of Clinical Psychology, 58* (12), 601-S1614.

Kwapil, T., Miller, M., Zinser, M., Chapman, J., & Chapman, L. (1997). Magical ideation and social anhedonia as predictors of psychosis proneness: A partial replication. *Journal of Abnormal Psychology, 106*(3), 491-495.

Laurent, A., Biloa-Tang, M., Bougerol, T., Duly, D., Anchisi, A., Bosson, J., et al. (2000). Executive/attentional performance and measures of schizotypy in patients with schizophrenia and in their nonpsychotic first degree relatives. *Schizophrenia Research, 46,* 269-283.

Laurent, A., Duly, D., Murry, P., Foussard, N., Boccara, S., Mingat, F., et al. (2001). Wisconsin Card Sorting Test performance and schizotypal features in first degree relatives of patients with schizophrenia. *Psychiatry Research, 104,* 133-144.

Leak, G. (1991). An examination of the construct validity of the social anhedonia scale. *Journal of Personality Assessment, 56*(1), 84-95.

Lenzenweger, M. (1991). Confirming schizotypic personality configurations in hypothetically psychosis-prone college students. *Psychiatry Research, 37,* 81-96.

Lenzenweger, M. (1998). Schizotypy and schizotypic psychopathology: Mapping an alternative expression of schizophrenia liability. In M. Lenzenweger & M. Dworkin (Eds.), *Origins and development of schizophrenia* (pp. 93-121). Washington, D.C.: American Psychological Association.

Lenzenweger, M. (2001). Reaction time slowing during high-load, sustained attention task performance in relation to psychometrically-identified schizotypy. *Journal of Abnormal Psychology, 110* (2), 290-296.

Lenzenweger, M., Cornblatt, B., & Putnick, M. (1991). Schizotypy and sustained attention. *Journal of Abnormal Psychology, 100*(1), 84-89.

Lenzenweger, M. & Gold, J. (2000). Auditory working memory and verbal recall memory in schizotypy. *Schizophrenia Research, 42* (2), 101-110.

Lenzenweger, M. & Korfine, L. (1994). Perceptual aberrations, schizotypy, and the Wisconsin Card Sorting Test. *Schizophrenia Bulletin, 20*(2), 345-357.

Lenzenweger, M. & Loranger, A. (1989a). Detection of familial schizotypy using a psychometric measure of schizotypy. *Archives of General Psychiatry, 46,* 902-907.

Lenzenweger, M. & Loranger, A. (1989b). Psychosis-proneness and clinical psychopathology: Examination of the correlates of schizotypy. *Journal of Abnormal Psychology, 98*(1), 3-8.

Lubdo, R., Kaplan, O., & DeLaCasa, G. (2001). Performance on the visual search analog of latent inhibition is modulated by an interaction between schizotypy and gender. *Schizophrenia Research, 52*, 275-287.

Luh, K. & Gooding, D. (1999). Perceptual biases in psychosis-prone individuals. *Journal of Abnormal Psychology, 108,* 283-289.

Lewine, R., Haden, C., Caudle, J., & Shurett, R. (1997). Sex-onset effects of neuropsychological function in schizophrenia. *Schizophrenia Bulletin, 23* (1), 51-61.

Loranger, M., et al. (1995). International Personality Disorders Evaluation.

Lyons, M., Toomey, R., Faraone, S., Kremen, W., Yeung, A., & Tsuang, M. (1995). Correlates of psychosis-proneness in relatives of schizophrenia patients. *Journal of Abnormal Psychology, 104*(2), 390-394.

Maier, W., Franke, P., Haine, C., Kopp, B., & Rist, F. (1992). Neuropsychological Indicators of the vulnerability to schizophrenia. *Progress in Neuropsychopharmacology and Biological Psychiatry, 16,* 703-715.

Maier, W., Lichterman, D., Minges, J., Heun, R. (1994). Personality disorders among relatives of schizophrenia patients. *Schizophrenia Bulletin, 20*(3), 481-493.

McGorry, P., Yung, A., Phillips, L. (2001). Ethics and early intervention in psychosis: Keeping up the pace and staying in step. *Schizophrenia Research, 51* (1), 17-29.

Meehl, P. (1962). Schizotaxia, schizotypy, schizophrenia. *American Psychologist, 17* (12), 827-838.

Meehl, P. (1990). Toward an integrated theory of schizotaxia, schizotypy, and schizophrenia. *Journal of Personality Disorders, 4* (1), 1-99.

Meehl, P. (2001). Primary and secondary hypohedonia. *Journal of Abnormal Psychology, 110* (1), 188-193.

Meltzer, H. (1999). Outcome in schizophrenia: Beyond symptom reduction. *Journal of Clinical Psychiatry, 60* (Suppl 3), 3-8.

Merritt, R., Balogh, D., & DeVinney, S. (1993). Use of the MMPI to assess the construct validity of the revised Social Anhedonia Scale as an index of schizotypy. *Journal of Personality Assessment, 60* (2), 227-238.

Michie, P., Kent, A., Stienstra, R., Castine, Z., Johnston, J., Dedman, K., et al. (2000). Phenotypic markers as risk factors in schizophrenia: Neurocognitive functions *Australia and New Zealand Journal of Psychology, 34*(suppl.), 574-585.

Miller, G. (1986). Information processing deficits in anhedonia and perceptual aberrations: A psychophysiological analysis. *Biological Psychiatry, 21*, 100-115.

Miller, E. & Chapman, J. (1983). Continued word association in hypothetically psychosis-prone college students. *Journal of Abnormal Psychology, 92*(4), 468-478.

Mishlove, M. & Chapman, L. (1985). Social anhedonia in the prediction of psychosis-proneness. *Journal of Abnormal Psychology, 94*(3), 384-396.

Mozley, L., Gur, R.C., Gur, R.E., Mozley, P.D., & Alavi, A. (1996). Relationships between verbal memory performance and the cerebral distribution of fluorodeoxyglucose in patients with schizophrenia. *Biological Psychiatry, 40* (6), 443-451.

Moritz, S., Andresen, B., Naber, D., Krausz, M., & Probstehein, E. (1999). Neuropsychological correlates of schizotypal disorganization. *Cognitive Neuropsychiatry, 4*(4), 343-349.

Newman, D., Moffitt, T., Caspi, A., & Silva, P. (1998). Comorbid mental disorders: Implications for treatment and sample selection. *Journal of Abnormal Psychology, 107* (2), 305-311.

Nieuwenstein, M., Aleman, A., deHaan, E. (2001). Relationship between symptom dimensions and neurocognitive functioning in schizophrenia: A meta-analysis of Wisconsin Card Sorting Test and continuous performance task studies. *Journal of Psychiatric Research, 35,* 119-125.

Nuchpongsai, P., Arakaki, H., Langman, P., & Ogura, C. (1999). N2 and P3b components of the event-related potential in students at risk for psychosis. *Psychiatric Research, 88,* 131-141.

Nuechterlein, K., Asarnow, R., Subotnik, K., Fogelson, D., Ventura, J., et al., (1998). Neurocognitive vulnerability factors for schizophrenia: Convergence across genetic risk studies and longitudinal trait-state studies. In M. Lenzenweger and R. Dworkin (Eds.). *Origins and development of schizophrenia* (pp. 299-327). Washington, D.C.: American Psychological Association.

Numbers, J. & Chapman, L. (1982). Social deficits in hypothetically psychosis-prone college women. *Journal of Abnormal Psychology, 91*(4), 255-260.

Obiols, J., Serrano, F., Caparros, B., Subira, S., & Barrantes, N. (1999). Neurological soft signs in adolescents with poor performance on the continuous performance task: Markers of liability for schizophrenia spectrum disorders? *Psychiatry Research, 86,* 217-228.

Orzack, M. & Kornetsky, C. (1971). Environmental and familial predictors of attention behavior in chronic schizophrenics. *Journal of Psychiatric Research, 9* (1), 21-29.

Overby, L. (1992). Perceptual asymmetry in psychosis-prone college students: Evidence for left-hemisphere overactivation. *Journal of Abnormal Psychology, 101*(1), 96-103.

Park, S. & Holzman, P. (1992). Schizophrenics show spatial working memory deficits. *Archives of General Psychiatry, 49* (12), 975-982.

Park, S., Holzman, P., & Goldman-Rakic, P. (1995). Spatial working memory deficits in the relatives of schizophrenia patients. *Archives of General Psychiatry, 52*, 821-828.

Park, S., Holzman, P. & Lenzenweger, M. (1995). Individual differences in spatial working memory in relation to schizotypy. *Journal of Abnormal Psychology, 104*(2), 355-363.

Park, S. & McTigue, K. (1997). Working memory and the syndromes of schizotypy. *Schizophrenia Research, 26*, 213-220.

Parnas, J., Cannon, T., & Jacobsen, B. (1993). Lifetime DSM-III-R diagnostic outcomes in the offspring of schizophrenic mothers: Results form the Copenhagen high-risk study. *Archives of General Psychiatry, 50* (9), 707-714.Perlstein, W.,

Carter, C., Noll, D., & Cohen, J. (2001). Relation of prefrontal cortex dysfunction to working memory and symptoms in schizophrenia. *American Journal of Psychiatry, 158*, 1105-1113.

Perry, W., Heaton, R., Potterat, E., Roebuck, T., Minassian, A., & Bratt, D. (2001). Working memory in schizophrenia: Transient online storage vs. executive functioning. *Schizophrenia Bulletin, 27*(1), 157-176.

Pizzagalli, D., Lehman, D., Gianotti, L., Koenig, T., Tanaka, H., Wackerman, J. et al. (2000). Brain electric correlates of strong belief in paranormal phenomena: intracerebral EEG source and regional Omega complexity analyses. *Psychiatry Research: Neuroimaging Section, 100*,139-154.

Raine, A. (1991). The SPQ: A scale for the assessment of schizotypal personality based on DSM-III-R criteria. *Schizophrenia Bulletin, 17* (4), 555-564.

Raine, A., Benishay, D., Lencz, T., & Scarpa, A. (1997). Abnormal orienting in Schizotypal personality disorder. *Schizophrenia Bulletin, 23*(1), 75-82.

Raine, A., Reynolds, C., Lencz, T., Scerbo, A., Triphon, N., & Kim, D. (1994). Cognitive-perceptual, interpersonal, and disorganized features of schizotypal personality. *Schizophrenia Bulletin, 20*, 191-201.

Randolph, C., Goldbert, T., & Weinberger, D. (1993). The neuropsychology of schizophrenia. In K. Heilman & Valenstein, E. (Eds.), *Clinical neuropsychology*. London: Oxford University Press.

Ratey, J. (1995). *Neuropsychiatry of Personality Disorders.* Cambridge, MA: Blackwell Science.

Roitman, S., Cornblatt, B., Bergman, A., Obuchowski, M., Mitropolan, V., Keefe, R., et al. (1997). Attentional functioning in schizotypal personality disorder. *American Journal of Psychiatry, 154*(5), 655-660.

Rosenfarb, I., Neuchterlein, K., Goldstein, M., & Subotnik, K. (2000). Neurocognitive vulnerability, interpersonal criticism, and the emergence of unusual thinking by schizophrenia patients during family transactions. *Archives of General Psychiatry, 57*, 1174-1179.

Ross, D., Thaker, G., Buchanan, R., Kirkpatrick, B., Lahti, A., et al. (1997). Eye tracking disorder in schizophrenia is characterized by specific ocular motor deficits and is associated with the deficit syndrome. *Biological Psychiatry, 42* (9), 781-796.

Russell, A., Munro, J., Jones, P., & Hemsley, D. (1997). Schizophrenia and the myth of intellectual decline. *American Journal of Psychiatry, 154* (5), 635-639.

Silverstein, M., Mavrolefteros, G., Close, D. (2002). Premorbid adjustment and neuropsychological performance in schizophrenia. *Schizophrenia Bulletin, 28* (1), 157-166.

Sponheim, S., Iacono, W., & Thuras, P. (2001). Using biological indices to classify schizophrenia and other psychotic patients. *Schizophrenia Research, 50* (3), 139-150.

Stirling, J., White, C., Lewis, S., Hopkinds, R., Tantam, D., et al. (2003). Neurocognitive function and outcome in first-episode schizophrenia: A 10-year follow-up of an epidemiological cohort. *Schizophrenia Research, 65* (2-3), 75-86.

Strandburg, D., Marsh, J., Brown, W., Asarnow, R., & Guthrie, D. (1994). Information processing deficits across childhood and adult-onset schizophrenia. *Schizophrenia Bulletin, 20*(3), 685-695.

Suhr, J. & Spitznagel, J. (2001). Factor vs. cluster models of schizotypal traits II: Relation to neuropsychological impairment. *Schizophrenia Research, 52,* 241-250.

Swanson, C., Gur, R., Bilker, W., Petty, R., & Gur, R. (1998). Premorbid educational attainment in schizophrenia: Association with symptoms, functioning, and neurobehavioral measures. *Biological Psychiatry, 44* (8), 739-747.

Tallent, K. & Gooding, D. (1999). Working memory and WCST performance in schizotypic individuals: A replication and extension. *Psychiatry Research, 89,* 161-170.

Tamminga, C., Thaker, G., & Buchanan, R. (1992). Limbic system abnormalities identified in schizophrenia using positron emission tomography with fluorodeoxyglucose and neocortical alterations with deficit syndrome. *Archives of General Psychiatry, 49* (7), 522-530.

Thaker, G., Moran, M., Adami, H. & Cassidy, S. (1993). Psychosis proneness scales in schizophrenia spectrum disorders: Familial vs. non-familial samples. *Psychiatry Research, 46,* 47-57.

Toomey, R., Faraone, S., Seidman, L., Kremen, W., Pepple, J., & Tsuang, M. (1998). Association of neuropsychological vulnerability markers in relatives of schizophrenia patients. *Schizophrenia Research, 31,* 89-98.

Torgersen, S. (1985). Relationship of schizotypal personality disorder to schizophrenia: Genetics. *Schizophrenia Bulletin, 4,* 554-563.

Trestman, R., Keefe, R., Mitroplou, V., Harvey, P., deVegvar, M., et al., (1995). Cognitive function and biological correlates of cognitive performance in schizotypal personality disorder. *Psychiatry Research, 59* (1-2), 127-136.

Tsuang, M., Stone, W., Faraone, S. (2000). Towards the prevention of schizophrenia. *Biological Psychiatry, 48* (5), 349-356.

U.S. Census Bureau (2002). 2000 U.S. Census. Washington, D.C.: United States Government Printing Office.

Walker, E., DiForio, D., & Baum, K. (1999). Developmental neuropathology and the precursors of schizophrenia. *Acta Psychiatrica Scandinavica* (Suppl. 395), 12-19.

Wechsler, D. (1997). Wechsler Memory Scales (3rd Ed.). Technical Manual. New York: Psychological Corporation.

Weickert, T., Goldberg, T., Gold, J., Bigelow, L., Egan, M., Weinberger, D. (2000). Cognitive impairments in patients with schizophrenia displaying preserved and compromised intellect. *Archives of General Psychiatry, 57,* 907-913.

Williams, J., Gibbon, M., & First, M. (1992). The Structured Clinical Interview for DSM-III-R (SCID): II. Multisite test re-test reliability. *Archives of General Psychiatry, 49* (8), 630-636.

Wyatt, R. (1995). Early intervention for schizophrenia: Can the course of the illness be alterered? *Biological Psychiatry, 38* (1), 1-3.

Yehuda, R., Edell, W., Meyer, J. (1986). Platelet MAO activity and psychosis proneness in college students. *Psychiatry Research, 20,* 129-142.

Yeo, R., Gangestad, S., & Thoma, R. (1997). Developmental instability and cerebral lateralization. *Neuropsychology, 11* (4), 552-561.

Yung, A., Phillips, L., McGorry, P. (1998). Prediction of psychosis: A step towards indicated prevention of schizophrenia. *British Journal of Psychiatry,172*(Suppl 33), 14-20.

Zaluska, M. (1998). Social functioning of schizophrenic patients 15 years from the first hospitalization. Part I: Estimation of social functioning. *Psychiatrica Polska, 32* (Suppl), 25-36.