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EMOTION RECOGNITION DEFICITS AS AN ENDOPHENOTYPE FOR SCHIZOPHRENIA SPECTRUM DISORDERS

A Dissertation

Presented to

The College of Graduate and Professional Studies

Department of Psychology

Indiana State University

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In Partial Fulfillment

of the Requirements for the Degree

Doctor of Psychology

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by

Alison V. James

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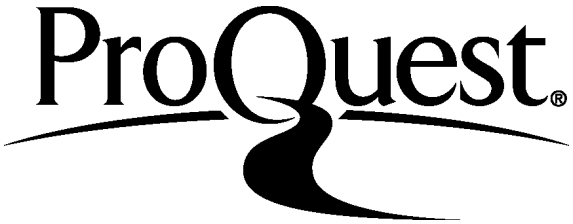

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ABSTRACT

Given the genetic complexity of schizophrenia, recent research has focused on the identification of endophenotypes as an effective method to increase our understanding of schizophrenia's genetic components and potential causes. Candidate endophenotypes for schizophrenia have been suggested to included personality disturbance, and deficits in both neurocognition and social cognition. With regard to the latter, evidence indicates that impairments in emotion recognition (ER) exist among individuals with schizophrenia, their unaffected relatives, and most recently, within a psychometrically psychosis prone population. However, no studies have examined whether these impairments exist as premorbid indicators of schizophrenia spectrum illnesses while concurrently also examining the relationship between genetic risk and ER abilities.

The current study is a cross-sectional component of a larger longitudinal study, and investigates whether individuals at risk (i.e., psychometric or genetic) for schizophrenia spectrum disorders possess ER deficits compared to matched controls (MC) and individuals without a family history of schizophrenia, respectively. ER performance was measured using the Penn Emotion Recognition Test-40 (ER40). Performance on the ER40 was also examined for positive versus negative schizotypes within the psychometric schizotype (PS) group. It was hypothesized that PS participants would exhibit greater ER deficits than MCs and that these deficits would be most pronounced for negative emotions and neutral expressions, and among negative schizotypes. It was also hypothesized that individuals with a family history of schizophrenia

would demonstrate greater ER deficits than those without a family history. Finally, it was hypothesized that the ER deficits observed would persist after statistically accounting for neurocognitive variance as measured by the Wechsler Adult Intelligence Scale–IV (WAIS-IV) and Wechsler Memory Scale-IV (WMS-IV).

Results revealed a more complex relationship between ER, neurocognition, and psychometric schizotypy than previously demonstrated. PS participants were significantly less accurate in their identification of overall emotion and negative emotion only after accounting for the variance associated with performance on the Working Memory Index (WMI) and Arithmetic subtest. The hypothesis that positive schizotypes would outperform negative schizotypes in their ability to accurately identify emotional expressions was not supported. Conversely, negative schizotypes were more accurate in their appraisal of emotional stimuli. The hypothesis that individuals with a family history of schizophrenia would perform worse than those without a similar family history on an ER task was supported.

Results are discussed within the broader category of candidate endophenotypes for schizophrenia. Future directions for research include a more detailed exploration of the relationship between working memory, psychometric schizotypy, and ER; incorporation of a more heterogeneous sample; and replication with other measures of schizotypy.

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CHAPTER 1

INTRODUCTION

Schizophrenia is a debilitating mental illness, affecting approximately 1% of the general population (Minzenberg, Yoon, & Carter, 2011), with lifetime prevalence rate reports ranging from 0.3% to 0.7% (American Psychiatric Association [APA], 2013). Despite a seemingly low prevalence rate, schizophrenia is listed as one of the top ten medical causes of disability throughout the world (World Health Organization, 2001). This is a direct reflection of the pervasive nature of the symptoms of schizophrenia. The illness is characterized by both positive and negative symptoms. Positive symptoms include hallucinations and delusions, whereas negative symptoms include deficits in one's social and emotional functioning, as well as poverty in the thought process (Minzenberg et al., 2011).

Research has also revealed that the risk of developing schizophrenia increases drastically in the presence of a family history of schizophrenia. For example, a biological first-degree relative of an individual with schizophrenia is approximately 10 times more likely to develop the disorder than an individual in the general population. In addition to having increased liability for schizophrenia, biological first-degree relatives have also been found to be at greater risk for a group of mental illnesses known as the schizophrenia spectrum, which includes schizoaffective disorder and schizotypal personality disorder (APA, 2013). Further, the degree of relative risk for

schizophrenia is related, although not linearly, to the degree of genetic relationship to the affected individual (c.f., Gottesman, 1991).

Despite this documentation of the hereditability of schizophrenia, much remains unknown about the influence of specific genes on the development of the disorder. Research by McGue and Gottesman (1989) revealed that genetic transmission of schizophrenia does not abide by simple Mendelian principles. Rather, it is the interaction between multiple genes and the environment that predisposes an individual to the development of schizophrenia (Braff, Schork, & Gottesman, 2007). Given the genetic complexity of psychiatric disorders such as schizophrenia, recent research has utilized endophenotypes as a way to gather information about the genetic components of schizophrenia. Endophenotypes are intermediate markers that are not only associated with an increased risk of the development of a disease or disorder, but also lie along the causal pathway between the disorder and its genotype (Lenzenweger, 2013). Although endophenotypes were originally applied to diseases, they have been gaining popularity in their use to assess the etiology of schizophrenia and other mental disorders. Gottesman and Gould (2003) stated that endophenotypes can be utilized as a simpler clue to the genetic factors associated with a given illness, and may be neuropsychological, cognitive, neuroanatomical, biochemical, endocrinological or neurophysiological. They also asserted that to be an effective marker, an endophenotype should be heritable, state independent, demonstrate cosegregation with the disorder, and must occur in non-affected relatives.

In addition to those endophenotypes that can be clearly recognized as being either biological or genetic, research has demonstrated that specific personality traits may also serve as predisposing factors for subsequent development of schizophrenia. Specifically, personality characteristics from the “Cluster A” personality disorders (Paranoid, Schizoid, and Schizotypal

Personality Disorders) have been identified as endophenotypes of schizophrenia (Braff, Freedman, Schork, & Gottesman, 2007). As such, previous research by Bolinskey and Gottesman (2010) has utilized the Chapman Psychosis Proneness Scale (CPPS) to detect premorbid personality dysfunction within a psychometric schizotype sample.

Disruptions in affective experiences and expression are also widely recognized as a hallmark symptom of schizophrenia. Recent research has expanded on these affective deficits to include deficits not only in the expression of emotion but also deficits in the perception of the emotions of others (Edwards, Jackson, & Pattison, 2002). Results have consistently demonstrated social cognition deficits among individuals diagnosed with schizophrenia, with recent findings suggesting that social cognition is a distinct, yet related, construct to neurocognitive deficits and negative symptoms (Sergi et al., 2007). Some of the specific social cognitive deficits that have been documented include deficits in emotion perception, perspective taking, theory of mind, interpersonal attribution, and social cue recognition (Eack et al., 2010). Findings have suggested that these deficits are not only present in patients diagnosed with schizophrenia, but can also be found to a lesser degree in their unaffected relatives (Snitz, MacDonald, & Carter, 2006). More recent research has begun to explore social cognitive deficits among individuals psychometrically identified to be at high risk for psychosis, and has found decreased overall accuracy in emotion recognition as well as decreased accuracy in the identification of negative emotions when compared to normal controls (Abbott & Green, 2013; Williams, Henry, & Green, 2007). Given these findings, researchers have posited that social cognition deficits should be included as an endophenotype of schizophrenia (Snitz et al., 2006).

Research on emotion recognition has spanned the last several decades and its assessment has included a variety of different stimuli, including photographs, drawings, and cartoons

(Edwards et al., 2002). The most commonly utilized emotion recognition measure was developed by Ekman and Friesen (1975), and consists of black and white photographs displaying the universal emotions of sadness, fear, happiness, anger, surprise, and disgust. More recent research by Kohler and colleagues (2003) has sought to improve upon the measure created by Ekman and Friesen (1975) by substituting color photographs that include both genders as well as Caucasians and non-Caucasians (i.e., African American, Asian, and Hispanic). Using the Penn Emotion Recognition Test (Kohler et al., 2003), Kohler and colleagues (2003) have demonstrated emotion recognition deficits in schizophrenia patients when compared to normal controls. These deficits were most pronounced with fear, disgust and neutral expressions.

The current study sought to examine emotion recognition deficits within a psychometric schizotype (PS) population, thus expanding on the previous research done by Williams and colleagues (2007) and Abbott and Green (2013). Unlike the previous studies, the current study sought to demonstrate similar deficits in emotion recognition within a group that is psychometrically identified as psychosis prone using the Chapman Psychosis Proneness Scales (CPPS). It was predicted that individuals in the PS group would demonstrate statistically significant deficits in emotion recognition when compared to a matched control (MC) sample. Specifically, it was predicted that PS participants would be less accurate when asked to identify the emotion of facial stimuli when compared to matched controls. Consistent with the research by Williams and colleagues (2007) and Kohler and colleagues (2003), it was further predicted that PS participants would be less accurate when asked to identify negative emotions (e.g., sad, anger, fear) and neutral expressions when compared to MC participants.

The current study also sought to replicate Eack and colleagues' (2010) findings that unaffected relatives of individuals with schizophrenia also demonstrate emotion recognition

deficits. It was, therefore, additionally predicted that individuals possessing a family history of schizophrenia would demonstrate statistically significant deficits in their ability to correctly identify the emotions of facial stimuli when compared to individuals without a family history of schizophrenia.

Lastly, given the findings from Sergi and colleagues (2007) that suggest that social cognition deficits are a distinct, yet related factor to neurocognitive deficits and negative symptoms of schizophrenia, it was predicted that emotion recognition deficits would remain after statistically accounting for the variance associated with neurocognitive deficits. Since the previous research also suggests that the relationship between negative symptoms and social cognition should be further explored, it was also predicted that emotion recognition deficits would be stronger among negative schizotypes (e.g., those scoring greater than 1.96 standard deviations above the mean on the *SocAnh* Chapman scale).

CHAPTER 2

REVIEW OF RELATED LITERATURE

Schizophrenia

The obvious symptoms of schizophrenia typically emerge in late adolescence or early adulthood, with men often experiencing onset at a younger age than women (Minzenberg et al, 2011). The average age of onset for men ranges from the early to mid-20s, whereas women tend to first develop obvious symptoms in their late 20s. These gender differences in age of onset also relate to prognostic differences for the disorder, as an earlier age of onset is associated with poorer premorbid adjustment, structural brain abnormalities, negative symptoms, cognitive impairment, and a poorer prognosis overall. Prior to the onset of the disorder, many individuals may gradually display prodromal symptoms such as social withdrawal, unusual behaviors, loss of interest in school or work, anger outbursts, or deficits in hygiene (APA, 2013).

The history of our current conceptualization of schizophrenia can be traced back several hundreds of years, with the first descriptions of the disorder credited to John Haslam and Philippe Pinel in the early nineteenth century. It was not until later that century that the term *dementia praecox* was coined by Bénédict Augustin Morel to describe the disorder now referred to as schizophrenia. Morel conceptualized the disorder as a premature dementia, highlighting its early onset and degenerative course. Although Morel is credited with coining the term dementia

praecox, Emil Kraepelin is often described as exerting the greatest impact on the disorder, as he was the first to describe and define it (Mizenberg et al., 2011).

Historical Conceptualizations of Schizophrenia

Kraepelin defined dementia praecox as possessing two major characteristics. The first characteristic of the disorder is the onset typically begins early in an individual's life (hence, "praecox"). The second characteristic of the disorder was that it resulted in pervasive impairment to a variety of cognitive and behavioral functions (hence, "dementia"). Although Kraepelin acknowledged the heterogeneity of symptom presentation in dementia praecox, he highlighted the disorder's poor outcome and chronic course as its most salient features that could be used to discern dementia praecox from manic-depressive disorders. In addition to Kraepelin's identification of course and outcome as key features of the disorder, he also emphasized negative symptoms and cognitive disturbances as fundamental for determining an individual's prognosis, response to treatment, and the level of impairment (Kraepelin, 1909/1971).

Eugen Bleuler was the first to introduce the term *schizophrenia* because he believed that the name *dementia praecox* was misleading (Keller, Fischer, & Carpenter, 2011). Bleuler criticized the concept of dementia praecox, pointing out that not all patients presented with an early onset or stable course of the disorder (Mizenberg et al., 2011). Unlike Kraepelin, Bleuler asserted that schizophrenia actually consisted of a group of disorders with a similar presentation (Keller et al., 2011). As such, Bleuler focused his efforts on describing the symptoms of schizophrenia as opposed to its course or outcome (Andreasen & Carpenter, 1993).

Bleuler emphasized cognition in his conceptualization of schizophrenia, asserting the "splitting of associations" to be the most fundamental symptom of the disorder. Other features he believed to be central to the presentation of schizophrenia included affective flattening,

ambivalence, and autism. He described these four symptom clusters as *fundamental*, because they were unique to the presentation of schizophrenia. Other symptoms of the disorder such as delusions, hallucinations, and catatonia were categorized as *accessory* symptoms, because their presence was possible in other disorders as well (Bleuler 1911/1950). Bleuler's reformulation of schizophrenia ultimately superseded Kraepelin's to become the prominent conceptualization of the disorder for much of the 20th century (Andreasen, 1997).

Although neither Kraepelin nor Bleuler directly addressed the demarcation between *positive* and *negative* symptoms of schizophrenia, it is apparent that Bleuler's conceptualization of fundamental and accessory symptoms closely parallels this modern dichotomy of schizophrenia symptoms. John Hughlings Jackson is often credited as one of the first to utilize the terminology of *positive* and *negative* symptoms. Jackson asserted that positive symptoms could be defined as an excess of a normal function, whereas negative symptoms represented a deficit or loss of function. As such, Kraepelin, Bleuler, and Jackson are often identified as fundamental influences in the conceptualization of schizophrenia (Andreasen, 1997).

Schneider's first-rank symptoms

Kurt Schneider was another influential figure in the conceptualization of schizophrenia and one who believed the disorder to be an organic illness (Fox, 1978). Much like Bleuler, Schneider sought to identify fundamental symptoms that were indicative of schizophrenia, thus focusing his efforts on the symptoms of the disorder and not its course. Schneider's primary intent in identifying these fundamental symptoms was to increase the reliability of a diagnosis in schizophrenia.

Schneider offered a conceptualization of schizophrenia that grouped psychological symptoms into either abnormalities of expression (e.g., negative symptoms) or abnormalities of

experience (e.g., positive symptoms). Unlike his predecessors Kraepelin and Bleuler, Schneider believed that positive symptoms of schizophrenia were most useful for the differential diagnosis of schizophrenia from other mental illnesses (Andreasen & Carpenter, 1993). As such, Schneider identified 11 "first rank" abnormalities of experience (see Appendix A) that he believed allowed one to more easily distinguish schizophrenia. Of the 11 first rank symptoms, three pertained to auditory hallucinations, one to somatic hallucinations, six to delusions of external control, and one to delusions of reference. Schneider referred to abnormalities of expression as "second rank" symptoms. Although Schneider noted that first rank symptoms were most useful for differential diagnosis of schizophrenia, he also cautioned that these symptoms were not required for diagnosis. According to Schneider, schizophrenia may be diagnosed in the absence of first rank symptoms if the second rank symptoms such as flat affect and loose associations experienced are pervasive (Fox, 1978). Given the reliability of a diagnosis of schizophrenia using Schneider's first rank symptoms, his diagnostic criteria were ultimately incorporated into structured diagnostic interviews and the Diagnostic and Statistical Manual- III (DSM-III; APA, 1980; Andreasen & Carpenter, 1993). However, it should be noted that an overreliance on Schneider's first rank symptoms for the diagnosis of schizophrenia might result in over diagnosis of schizophrenia at the expense of under-diagnosis of bipolar disorder (Gottesman, 1991).

Despite Schneider's efforts to increase diagnostic accuracy and reliability with the delineation of his first and second rank symptoms, recent research has criticized the sensitivity and specificity of these symptoms when diagnosing schizophrenia. For example, numerous studies have documented the variable prevalence of first rank symptoms in schizophrenia, ranging from 25.4% to 88%. In addition to the variable presence of first rank symptoms in schizophrenia, other studies have demonstrated the prevalence of first rank symptoms in

disorders other than schizophrenia, with rates ranging from 1 to 32%. Although the rates of first rank symptoms are less prevalent for other disorders when compared to schizophrenia, it is clear that the diagnostic utility of first rank symptoms is not as fundamental as once thought. However, it should be noted that the variable prevalence rates of first rank symptoms is likely due in part to the use of differing definitions of the symptoms by the researchers, which could be reflective of errors in translation from Schneider's original texts written in German (Saddichha, Kumar, Sur & Sinha, 2010).

Recent Conceptualizations of Schizophrenia

Dopamine hypothesis

The dopamine hypothesis was catalyzed by the French surgeon Henri Laborit's discovery that the drug chlorpromazine, which was originally created to be a pre-anesthetic agent, elicited a calming effect when administered to patients. As a result, Laborit recommended the use of chlorpromazine to calm agitated patients. From there, the drug was also quickly applied with positive results to the treatment of other psychiatric illnesses such as schizophrenia. Given the drug's antidopaminergic and anticholinergic properties, it was deduced that chlorpromazine's efficacy in treating the symptoms of schizophrenia was due to the fact that those with the disorder possessed increased levels of dopamine. These findings proved to be crucial for the development of the dopamine hypothesis, which theorizes that the symptoms of schizophrenia result from a dysregulation of the neurotransmitter dopamine (Mizenberg et al., 2011).

Subsequent research during the 1960s and 1970s sought to confirm the role of dopamine dysfunction in schizophrenia. For example, Carlsson and Lindqvist's (1963) use of animal models concluded that the efficacy of neuroleptic drugs such as chlorpromazine and haloperidol in treating psychotic symptoms was due to their ability to block dopamine receptors in the brain.

It was likewise observed that the administration of substances such as amphetamines, which increase the action of dopamine in the brain, catalyzed the manifestation of psychotic symptoms. As such, it was proposed that schizophrenia symptoms were the result of overstimulation at dopamine receptor sites (Mizenberg et al., 2011).

It was not until the mid-1970s that the dopamine hypothesis became accepted and widely regarded as an etiological factor in the development of schizophrenia (Moncrieff, 2009). Further research in the 1970s indicated that binding to the D_2 receptor site, specifically, was responsible for the clinically efficacious effects of neuroleptic medications (Creese, Burt, & Snyder, 1976). However, the popularity of the dopamine hypothesis began to decline during the 1980s and 1990s, largely due to the discovery that neuroleptic drugs such as chlorpromazine had little effect in decreasing the presence of the negative symptoms of schizophrenia (Moncrieff, 2009). In an effort to account for this lack of therapeutic effect on negative symptoms, alternative hypotheses were subsequently offered. One such theory proposed by Davis, Kahn, Ko, and Davidson(1991) suggested that the symptomology of schizophrenia is due to the presence of both increased dopamine activity as well as a decrease in activity in the frontal cortex. Davis et al. (1991) indicated that negative symptoms are primarily the result of this cortical underactivity in the frontal cortex.

More recent research on the dopamine hypothesis has benefited from the incorporation of a variety of neuroimaging tests, including positron emission tomography (PET). Research utilizing PET scans has revealed that dopaminergic action in individuals with schizophrenia is more multifaceted than previously hypothesized. Abi-Dargham and colleagues (2000) suggested that increased activity of D_2 receptors might be the cause of positive symptoms, whereas underactivity in the D_1 receptors may cause the cognitive deficits associated with schizophrenia.

Given the shortcomings of the dopamine hypothesis, Howes and Kapur (2009) proposed that dopamine dysregulation is likely the result of the combined effects of both genetic and environmental factors. Specifically, research has suggested that factors such as childhood stress may potentiate striatal dopamine release. This has been confirmed by studies examining the relationship between low maternal care during childhood and subsequent dopaminergic function. Other environmental factors such as obstetric complication may also modulate other neurotransmitter systems such as gamma-aminobutyric acid (GABA), which has been demonstrated to be involved in subcortical dopaminergic dysfunction (Howes & Kapur, 2009). Likewise, research has also suggested that deficits of the frontal and temporal lobes may predispose individuals to the effects of obstetric complications.

Brain structural differences

Studies of the brain structure abnormalities commonly found in individuals with schizophrenia have been ubiquitous since the latter half of the 20th century. Fortunately, technological advances in recent decades have increased the accuracy of these studies through the utilization of new neuroimaging techniques such as magnetic resonance imaging (MRI) and computerized tomography (CT) scans. Subsequent studies have documented several anatomical brain abnormalities that are often found in schizophrenia patients, including enlarged ventricles, whole brain and temporal lobe volume deficits, decreased brain region connectivity, and a reduction of gray matter (GM) (Mizenberg et al., 2011). Although GM deficits have been the focus of previous volumetric studies, recent research has suggested that abnormalities in white matter (WM) are also present in schizophrenia (Colibazzi et al., 2013).

Johnstone, Crow, Frith, Husband, and Kreel (1976) report is lauded as the pioneering work for the study of brain structural deficits in schizophrenia. In their report, they described an

enlargement of the lateral cerebral ventricles and a concomitant reduction in global brain volume in chronic schizophrenia patients when compared to normal controls, thus confirming prior assertions by Kraeplin that schizophrenia is an organic and brain-based disease (Harrison, 1999). Subsequent research has replicated these findings, with MRI review studies documenting a median 40% increase in lateral and third ventricle size among schizophrenia patients and a subsequent decrease of approximately 3% in brain tissue (Lawrie & Abukmeil, 1998). Further analyses by Lawrie and Abukmeil (1998) revealed that the greatest brain tissue reduction occurred in the temporal lobe and medial temporal structures (e.g., amygdala, hippocampus, parahippocampal gyrus) with 8% and 4-12% reductions, respectively.

Previous MRI studies of schizophrenia have demonstrated the presence of temporo-limbic abnormalities, including findings of reduced volume in the temporal lobe, hippocampus, amygdala, superior temporal gyrus (STG), and parahippocampal gyrus. These structures of the temporal lobe and its circuitry are largely responsible for the modulation of emotion and cognition. Furthermore, the temporal lobe and its subregions have also been connected to symptoms of schizophrenia, including auditory hallucinations and thought disorder.

Gur and colleagues (2000) compared 100 patients with schizophrenia and 100 healthy control participants using neurocognitive testing, clinical assessments, and MRI studies. The patients with schizophrenia were screened so as not to have any history of any other disorder that might influence their brain functioning. The results of the study were consistent with previous research that found reductions in GM, global brain volume, and temporal subregion volume among schizophrenia patients when compared to normal controls. Specifically, volumetric reductions were found across sexes in the hippocampus (7% for men, 8.5% for women), STG (11.5% for men, 4% for women), and temporal pole (10% for men, 8.5% for women).

Interestingly, sex differences were reported for amygdala volume, with men demonstrating a 7% reduction whereas women showed a 10.5% increase in volume. Given these findings on the amygdala, Gur and colleagues (2000) suggested that the volumetric reduction found in previous studies might be due to the presence of a greater proportion of male participants.

These ventricle and GM abnormalities have not only been documented in chronic schizophrenia populations, but have also been found in patients experiencing their first episode of schizophrenia. In a meta-analysis by Vita, De Peri, Silenzi, and Dieci (2006), similar brain abnormalities were found among first episode participants, including reductions in total brain volume and hippocampus size, along with increased ventricle size. Unlike their chronic counterparts, those individuals experiencing their first episode did not possess significant abnormalities in temporal lobe or limbic structure volume. It was therefore proposed that limbic changes might play a greater role in chronic patients than those suffering from their first episode.

Prior research has also demonstrated changes in brain structure of schizophrenia patients over time. For example Woods, Yurgelun-Tood, Benens, Frankerburg, Pope, and McSparren (1990) assessed nine schizophrenia patients and nine bipolar patients using CT scans over intervals of one to four years, and found that in addition to an increased ventricle-brain ratio for schizophrenia patients (25% versus 11% for bipolar participants) at baseline, schizophrenia participants also showed a significant progression in their ventricle size from the first to final scan.

Although much of the previous literature on brain volumetric abnormalities of schizophrenia patients has focused on deficits in GM, recent research by Colibazzi et al. (2013) posits that deficits in white matter (WM) can also be observed in schizophrenia patients. They attribute this difference in findings of WM volume in schizophrenia to previous research designs

that focused on large cortical regions, or failed to examine GM and WM concurrently. Colibazzi et al. assessed 76 outpatients with schizophrenia and 57 normal control participants using neurological and clinical measures, as well as MRIs. They found abnormalities in surface brain tissue volume to be related to underlying deficits in WM volume, as opposed to a reduction of the cortical GM thickness. Furthermore, they also found that WM abnormalities were correlated with measures of working memory. Thus, they proposed that WM abnormalities might be the fundamental anatomical deficit and may contribute to the observation of reductions in GM within schizophrenia patients.

Crow's type I and type II schizophrenia

Crow's characterization of schizophrenia as two dimensional was largely precipitated by two challenges to the dopamine hypothesis of schizophrenia. As previously noted, the popularity of the dopamine hypothesis plummeted during the 1980s and 1990s after it was discovered that neuroleptic drugs such as chlorpromazine failed to exert an effect on the presence of negative symptoms despite their efficacy on the treatment of positive symptoms. Further, some patients simply did not show clinical response to neuroleptic medications. Along with these observations, Crow (1980) also noted that the presence of cognitive impairments among some patients was incongruent with the notion that the disorder was simply attributable to dopaminergic dysregulation.

As such, Crow (1980) posited that the symptoms of schizophrenia were reflective of two syndromes with differing etiologies. According to Crow, those with type I syndrome possess a better prognosis, and are characterized by positive symptoms such as hallucinations, delusions and thought disorder. The type I syndrome is also reflective of increased D_2 dopamine receptors in the brain, and therefore can be successfully treated with dopamine antagonist medications.

Conversely, Crow argued that the type II syndrome includes negative symptoms such as flat affect and poverty of speech, and is generally associated with a poorer prognosis. These symptoms do not respond well to neuroleptic treatment, and, as a result, are thought to be irreversible. The type II syndrome is also often accompanied by intellectual impairment and abnormal involuntary movements. Crow suggested that underlying cause of these symptoms might be due to cell loss (e.g., peptide-containing interneurons) within subcortical temporal lobe structures such as the hippocampus, parahippocampal gyrus, and amygdala (Crow, 1985). Crow's conceptualization was the first to address the variable course of the disorder, suggesting that chronicity depends on the specific symptoms the individual suffers from.

Although Crow suggested that type I and type II syndromes represent two separate etiologies of pathology, he emphasized that they still represent components of the same disease. Crow's assertion was supported by the findings from Owens and Johnstone (1980), who assessed 500 schizophrenia patients' cognitive and neurological status as well as their behavioral impairments, and found significant correlations between negative symptoms, poor behavior, intellectual impairment, and neurological symptoms, and were found to be independent of positive symptoms.

Later work by Meltzer (1985) challenged some of Crow's previous assertions regarding the type II syndrome. For example, Meltzer stated that the type II syndrome might be the result of deficits in dopaminergic activity. Also contrary to Crow's theory, Meltzer demonstrated the efficacy of neuroleptics in the treatment of negative symptoms. This finding established support for the role of dopamine in the etiology of these symptoms, and provided evidence that negative symptoms can be effectively treated. Despite these contradictory findings by Meltzer, the

presence of two symptom syndromes has been well established within the study of schizophrenia.

DSM-5 changes

The diagnostic criteria for schizophrenia underwent several major changes in the most recent publication of the DSM, the DSM-5 (APA, 2013; see Appendix B). These changes include two alterations to the DSM-IV's Criterion A (see Appendix C) and the elimination of the schizophrenia subtypes (APA, 2000). In the DSM-IV, the presence of one symptom (as opposed to two of the remaining symptoms) could satisfy the requirement for Criterion A if Schneiderian first-rank auditory hallucinations or bizarre delusions were present. This caveat was removed in the DSM-5 due to concerns about the poor diagnostic reliability in discerning bizarre delusions from non-bizarre delusions and the lack of specificity of first rank symptoms. As such, Criterion A in DSM-5 requires that two of its symptoms are present. The second change to Criterion A is the requirement of at least one core positive symptom (e.g., delusions, hallucinations, or disorganized speech) due to concerns about the diagnostic reliability of the determination of negative symptoms. As previously mentioned, the final change to the DSM-5 with regard to schizophrenia is the removal of its subtypes (e.g., catatonic, disorganized, paranoid, residual, undifferentiated). The subtypes were removed due to poor diagnostic reliability and validity among clinicians, and their inadequate diagnostic stability. Furthermore, the subtypes of schizophrenia as diagnosed by clinicians were not found to be related to the course of the disorder or response to treatment. A dimensional rating scale is provided in section III of the DSM-5, which can be used to rate the severity of symptoms, although it is optional (APA, 2013).

Negative symptoms

As previously mentioned, negative symptoms of schizophrenia are defined as an absence of, or deficit in, normal mental functions and are found in social, affective, and cognitive realms of functioning (Minzenberg et al., 2011). Negative symptoms may be divided into primary and secondary symptoms. Primary negative symptoms are regarded as being a part of schizophrenia, whereas secondary symptoms may conversely have other causes (e.g., they may be the result of a preoccupation with hallucinations or delusions, or may be a side effect of medication). Approximately 50-90% of schizophrenia patients possess negative symptoms during the onset of the disorder, whereas roughly 20-40% of patients experience these symptoms pervasively after their first episode. Previous research has indicated that individuals suffering from negative symptoms at onset have a poorer prognosis when compared to those without negative symptoms. The presence of negative symptoms has also been correlated with social, professional, economic, and functional disabilities, thus affecting the quality of life of those suffering from negative symptoms (Mäkinen, Miettunen, Isohanni, & Koponen, 2008).

Negative symptoms include blunted affect, alogia, anhedonia, avolition, and asociality. The disruptions of affective processes that occur with blunted affect are perhaps one of the most readily identified symptoms of schizophrenia. Those with blunted affect possess decreases in their display and range of emotion (Minzenberg et al., 2011). They may also demonstrate poor eye contact, use few communicative gestures, use few spontaneous movements, and lack modulation in their voice. Alogia refers to a poverty of speech, where individuals talk infrequently and use few words while speaking. Anhedonia is a diminished capacity to experience pleasure, and may take the form of reduced interest in sexual activities or a decreased frequency of recreational or leisure activities. Avolition is reflective of a reduction in motivation

and may manifest as poor hygiene. Lastly, asociality refers to a decreased interest in social interactions, which may present as a lack of friendships or the presence of poor relationships with others (Mäkinen et al., 2008).

Schizotypy

Kraepelin (1909/1971) and Bleuler (1911/1950) were the first to articulate the relationship between schizophrenia and the presence of attenuated symptoms of the disorder among family members, describing these individuals' subtle symptomology as *latent schizophrenia*. Bleuler expanded upon this phenomenon, suggesting that the presence of these latent schizophrenia symptoms may represent a midpoint between the disorder itself and a "normal" personality. Bleuler's conceptualization of latent schizophrenia is essentially regarded as one of the first references to a continuum-based view of mental illness symptomology.

Several decades later, Rado (1953) continued with the development of this concept by coining the term *schizotype*, which he defined as meaning a schizophrenic phenotype. Similar to his predecessors, Rado asserted that schizotypal symptoms present on a continuum, and can manifest in a number of outcomes from compensated schizotypy to a diagnosis of schizophrenia. He proposed that the term schizotype was reflective of a cluster of impairments that co-occurred in both latent traits and symptoms of schizophrenia. Of these impairments, he identified a compromised ability to experience pleasure and an aberrant awareness of one's body as key traits. This pleasure deficit could include a lack of interest in vocation, interpersonal relationships, or pleasurable activities, whereas aberrant awareness of the body is often characterized as body-image distortions.

Meehl's model of schizotaxia, schizotypy, and schizophrenia

Paul Meehl's (1962) model of schizotaxia, schizotypy, and schizophrenia arose during a time where the prevailing *zeitgeist* attributed the etiology of schizophrenia to injurious childrearing practices (e.g., the "schizophrenogenic mother"). Meehl argued that the concept of the schizophrenogenic mother did not provide an adequate etiology, because it did not explain why a schizophrenia patient is a *patient*, as opposed to being merely an individual with a bad mother. Conversely to then-current models of schizophrenia development, he emphasized the role of genetic factors in the etiology of the disorder and how genetics relate to clinical symptomology and social-learning influences (Lenzenweger, 2006).

Meehl's model asserted that during development, a single *schizogene* interacts with the development of the central nervous system (CNS), resulting in a CNS aberration, which he called *hypokrisia*. The neuronal aberration inherent in hypokrisia results in a disruption in CNS neural transmission, and behaviorally manifests as cognitive slippage. Meehl believed that the effects of hypokrisia were evident throughout the brain, and thus reflected a global disruption in CNS functioning and genetic predisposition to the development of schizophrenia that he termed *schizotaxia*.

It should be noted that although schizotaxia is related to a genetic predisposition for schizophrenia, it is the intersection of this predisposition, social learning, and other *polygenetic potentiators* such as personality characteristics that together catalyze the development of schizophrenia. These polygenetic potentiators occur independently of schizotaxia, and include personality dimensions such as a proneness to anxiety, introversion, diminished pleasure capacity, and aggressivity (Lenzenweger, 2006). Meehl asserted that the vast majority of

schizotaxic individuals go on to develop schizotypy, which he referred to as the interaction between social learning influences and schizotaxia.

Assessing schizotypy

Given the relationship between schizotypy and liability to schizophrenia, several researchers have utilized factor analyses to determine the degree to which signs and symptoms of schizotypy resemble the structure of symptoms seen with schizophrenia. These studies have largely yielded three factor models as the best reflection of the symptoms of schizotypy, and include cognitive/perceptual, disorganization, and interpersonal components (Raine, Reynolds, Lencz, & Scerbo, 1994). Conversely, the three factors that have been demonstrated as specific to schizophrenia symptoms include reality distortion (e.g., delusions or hallucinations), disorganization (e.g., thought disorder), and negative symptoms (e.g., avolition or flat affect). Likewise, Lenzenweger and Dworkin (1996) have additionally proposed that a fourth factor associated with premorbid social functioning also exists.

These cognitive/perceptual, disorganization, and interpersonal components of schizotypy have been utilized in psychodiagnostic assessments for the detection of schizotypy traits. These assessments are largely divided into two categories, each of which possesses strengths and weaknesses. The first of these methods is the diagnostic interview. Although diagnostic interviews allow for increased contact with the interviewee, they can be subject to poor inter-rater reliability. The second method for assessing schizotypy is self-report inventories. These inventories allow for increased data collection, as they are both time and cost effective. Also, unlike the use of diagnostic interviews, the use of self-report inventories leaves little room for subjective interpretation among raters, and as such allows for the precise measurement of schizotypy symptoms (Lenzenweger, 2006, 2010).

One self-report inventory that has been found to effectively assess for schizotypy is the Chapman Psychosis Proneness Scales (CPPS). The CPPS were based in part on Meehl's (1962) work, and as such, the CPPS includes separate scales to assess the specific constructs of schizotypy. These scales include the Perceptual Aberration Scale (*PerAb*; Chapman, Chapman, & Raulin, 1978), the Magical Ideation Scale (*MagId*: Eckblad & Chapman, 1983), the Revised Physical Anhedonia Scale (*PhyAnh*; Chapman, Chapman, & Raulin, 1976), and the Revised Social Anhedonia Scale (*SocAnh*; Eckblad, Chapman, Chapman, & Mishlove, 1982). Of these scales, the first two are considered to assess the positive symptoms of schizotypy, whereas the latter two scales assess the negative symptoms of schizotypy.

Previous research has demonstrated the psychometric reliability in assessing schizotypy with the CPPS. For example, Chapman, Chapman, Kwapil, Eckblad, and Zinser (1994) demonstrated that not all individuals identified as schizotypes on the basis of their CPPS scores decompensate into diagnosable schizophrenia. This finding is consistent with Meehl's assertion that although schizotaxia reflects a genetic predisposition for schizophrenia, other factors (e.g., one's environment) interact with this genetic predisposition to produce the disorder. Likewise, Chapman and colleagues (1994) also demonstrated that the personality characteristics identified using the CPPS were stable. Results of their 10-year longitudinal study revealed that nearly 30% of individuals who scored in the deviant range on the *PerAb* scale met criteria for schizophrenia at ten-year follow up. They additionally found that a greater number of psychotic experiences were reported among participants with deviant scores on the *PerAb* and *MagId* scales.

A subsequent longitudinal study by Kwapil (1998) demonstrated similar predictive validity utilizing the *SocAnh* scale, with 24% of participants who scored in the deviant range on the scale having been diagnosed with a schizophrenia spectrum disorder at 10-year follow up.

Additionally, participants with deviant *SocAnh* scores were also more likely to report psychotic symptoms of greater severity, and possessed poorer adjustment levels at follow up when compared to the control group. The results of these studies therefore illustrate the CPPS' ability to identify individuals who are at an increased risk for developing schizophrenia spectrum disorders. As such, research by Bolinskey and Gottesman (2010) has utilized the Chapman Psychosis Proneness Scale (CPPS) to detect premorbid personality dysfunction within a psychometric schizotype sample.

Endophenotypes

Endophenotypes are intermediate factors that are associated with an increased risk of the development of a disease or disorder. Although the term endophenotype is often used interchangeably with other terms such as intermediate phenotype and biomarker in the literature on psychopathology, these terms in fact represent discrete constructs pertaining to different domains. Lenzenweger (2013) suggested that the term intermediate phenotype be reserved for its traditional use in Mendelian genetics as a descriptor of the genetic phenomenon of partial dominance. Similarly, he suggested that the term biomarker may be best utilized when discussing the biological impact that exogenous factors (e.g., one's environment) may have on psychopathology. Although endophenotypes were originally applied to diseases, they have been gaining popularity in their use to assess the etiology of schizophrenia and other mental disorders. Gottesman and Gould (2003) stated that endophenotypes can be utilized as a simpler clue to the genetic factors associated with a given illness, and may be either neuropsychological, cognitive, neuroanatomical, biochemical, endocrinological, or neurophysiological. Gottesman and Gould also asserted that to be an effective marker, an endophenotype should be: associated with the

illness, heritable, state independent (e.g., is present in the absence of active symptoms), demonstrate cosegregation with the disorder, and must occur in non-affected relatives at a greater rate than the general population.

In addition to biological and genetic endophenotypes, research has also demonstrated that specific personality traits may also serve as predisposing factors for subsequent development of schizophrenia. Specifically, personality characteristics from the Cluster A (Schizotypal, Schizoid, Paranoid) personality disorders have been identified as endophenotypes of schizophrenia (Braff, Freedman, Schork, & Gottesman, 2007).

Social Cognition and Schizotypy

Social cognition is an expansive construct that encompasses several abilities related to the way individuals process and think about themselves and others within a social context. Penn and colleagues (2008) identified four major themes in the research literature on social cognition. The first of these themes is mentalism, or an emphasis on mental representations such as schemas. These mental representations can assist individuals in navigating their social world by providing them with an organizational framework with which to organize social cues, ideas, or attitudes. Second, social cognition research is process orientated in that it focuses on gathering information to understand the processes or mechanisms that occur between social stimuli and product behaviors. Third, social cognition research is dependent on a cross-fertilization of ideas from multiple psychology disciplines, including cognitive, social, clinical, and developmental psychology, and neuroscience. For example, neuroscience research has identified specific areas of the brain that are activated during certain social processes. Fourth, the research literature has emphasized real-world applications for social cognition findings. Results have been applied in a

number of settings, including court cases looking to assess factors such as prejudice and discrimination (Penn, Sanna, & Roberts, 2008).

Research on social cognition in schizophrenia has increased dramatically in recent years as a defining feature of the disorder, with individuals with schizophrenia demonstrating deficits in a number of social cognitive domains. Abnormalities in these areas of social functioning have been demonstrated as stable features of schizophrenia, and have been documented during the prodromal phase, at diagnosis, and also throughout the course of the disorder. Likewise, these deficits have also been found premorbidly and among first-degree relatives of those with schizophrenia. Some of the social cognitive abilities that have been most frequently examined in the research literature include social perception, theory of mind, attributional style, and emotion recognition (Couture, Penn, & Roberts, 2006; Mizenberg et al., 2011).

Social perception is defined as the ability for one to determine social cues based on another individual's behavior during a social context. It is often closely linked to social knowledge, which is a person's understanding of social conventions or rules that are often stored in social schemas, and as such they are often discussed concurrently. Likewise, theory of mind is also comprised of two parts: the capacity to recognize that other individuals possess mental states that may differ from one's own, and the ability to correctly infer the content of other's mental states such as their beliefs or intentions. The capacity for theory of mind is often described as one's ability to comprehend things such as false beliefs, verbal hints, deception, irony, and metaphor.

Attributional style refers to the explanations individuals make about the reason for certain events in their life. Typically, individuals will take responsibility for the positive events in their life, while attributing negative events to others. Attribution style deficits in schizophrenia are

most clearly demonstrated among those suffering from paranoia or persecutory delusions, as these patients tend to blame negative outcomes on individuals rather than situations. This attributional tendency is known as the "personalizing bias." Finally, emotion recognition is defined as one's ability to make inferences about another's emotional state or feelings from their vocal inflections, facial expressions, or both (Couture et al., 2006; Penn et al., 2008).

Social cognition, neurocognitive deficits, and negative symptoms

One area of recent study in social cognition is its relationship with other symptoms of schizophrenia such as neurocognitive deficits and negative symptoms. Although the examination of social cognition has become an increasing popular area of study in schizophrenia, little is known about the nature of these deficits. Previous research has proposed that social cognition is a construct independent of both neurocognition and negative symptoms (Sergi et al., 2007).

Sergi and colleagues (2007) utilized correlational and structural equation modeling to assess whether social cognition's relationships with neurocognition and negative symptoms are better explained utilizing one- or two-factor models. They analyzed the three constructs concurrently to assess which factor social cognition was more closely related to. Their findings suggest that, although related to neurocognition, social cognition operates as a mediator between neurocognition and functional outcomes in schizophrenia. Their analyses also showed that two-factor models of social cognition and neurocognition, and social cognition and negative symptoms, described the data more accurately than a one-factor model. Finally, when analyzed concurrently, they found the relationship between social cognition and negative symptoms to be weaker than the association between social cognition and neurocognition.

The results of these findings by Sergi and colleagues (2007), therefore, offer preliminary support for the conceptualization of social cognition as a separate, albeit related, construct to

neurocognition and provide provisional information regarding social cognition's relationship with negative symptoms. As such, the researchers suggested that the relationship between social cognition and negative symptoms should be addressed in future research, with attention being focused on the relationship between specific negative symptoms (e.g., anhedonia and affective flattening) and social cognition (Sergi, Rassovsky, Nuechterlein, & Green, 2006; Sergi et al., 2007).

Emotion recognition

Research on emotion recognition has spanned the last several decades and its assessment has included a variety of different stimuli, including photographs, drawings, and cartoons (Edwards et al., 2002). As previously noted, the relationship between emotion recognition and schizophrenia has been widely researched in recent years. A literature review by Couture and colleagues (2006) revealed several trends in the literature pertaining to emotion recognition deficits in schizophrenia, including moderate to large effect sizes for the relationship between emotion recognition and social behavior, as well as consistent findings for relationships with emotion recognition and social skill, and emotion recognition and community functioning.

The most commonly utilized emotion recognition measure was developed by Ekman and Friesen (1975), and consists of black and white photographs displaying the universal emotions of sadness, fear, happiness, anger, surprise, and disgust. More recent research by Kohler and colleagues (2003) has sought to improve upon the measure created by Ekman and Friesen by substituting color photographs that include both genders as well as Caucasians and non-Caucasians (i.e., African American, Asian, and Hispanic). Referred to as the Penn Emotion Recognition Test (Kohler et al., 2003), Kohler and colleagues have demonstrated emotion

recognition deficits in schizophrenia patients when compared to normal controls. These deficits were most pronounced with fear, disgust and neutral expressions.

Our understanding of the relationship between schizophrenia and emotion recognition has been expanded through more specific studies of the relationship between emotion recognition and prodrome and first episode patients, unaffected first-degree relatives, and those at clinical high risk for schizotypy. For example, Addington, Penn, Woods, Addington, and Perkins (2008) compared individuals determined to be at clinical high risk for the disorder, who were psychometrically identified as meeting criteria for the prodrome state, to individuals experiencing their first episode of psychosis and normal controls. They found that those individuals at clinical high risk performed much more poorly on emotion recognition tasks when compared to normal controls, and just as poorly on these tasks when compared to individuals in the first episode group. With regard to research on emotion recognition deficits among first degree relatives of patients with schizophrenia, both Kee, Horan, Mintz, and Green (2004) and Leppänen, Niehaus, Koen, Du Toit, Schoeman, and Emsley (2008) found siblings of schizophrenia patients to perform considerably worse on emotion recognition tasks when compared to healthy control groups. In addition to performing worse than healthy controls, Kee and colleagues (2004) found that the siblings performed slightly better on emotion recognition tasks than their affected siblings.

In addition to these findings on emotion recognition deficits among first-degree relatives, more recent research has sought to examine the relationship between emotion recognition and psychometric schizotype (PS) groups given the paucity of emotion recognition research with this population. One such study by Williams and colleagues (2007) identified PS individuals on the basis of their scores on the Schizotypal Personality Questionnaire (SPQ; Raine, 1991). Results

from the study demonstrated decreased emotion recognition accuracy of negative emotions. However, these results were specific to the negative aspects of schizotypy (e.g., this was not found in positive or disorganized aspects). Subsequent research by Abbott and Green (2013) also sought to examine emotion recognition deficits in PS participants utilizing the same measure of schizotypy, and found that PS participants were less accurate in identifying facial affect when compared to normal controls. These results were found to be specific to the interpersonal features of schizotypy (e.g., social anxiety, constricted affect, lack of friends), and were not found within the cognitive-perceptual aspects of schizotypy.

The Penn Emotion Recognition Test. Prior research by Kohler and colleagues (2003) compared schizophrenia patients and healthy controls on their 96-item Penn Emotion Recognition Test. Results from their study indicate that schizophrenia patients perform worse across facial emotional stimuli when compared to healthy controls (63.6% correct vs. 71% correct; odds ratio = 0.71, $p < .001$), with schizophrenia patients being 0.71 times as likely to correctly identify the affective expression when compared to the healthy control participants. Additionally, schizophrenia patients were also found to be less accurate in emotion recognition when compared to healthy controls on both high intensity (67.6% vs. 74.9%) and low intensity (57.1% vs. 61.1%) emotional stimuli. With regard to the recognition of specific emotions, no differences were found between control participants and schizophrenia patients on happy, sad, or angry expressions. However, schizophrenia patients performed worse on the identification of disgusted (40.9% vs. 49.1%), fearful (60.0% vs. 74.4%) and neutral (69.6% vs. 85.9%) affective displays.

The 96-item Penn Emotion Recognition Test has also been utilized to assess emotion recognition capacity among healthy participants (Kohler, Turner, Gur, & Gur, 2004). Among

healthy participants, happy emotional stimuli are the most accurately recognized, followed by neutral, fear, sadness, anger, and disgust facial emotional stimuli, respectively. Additionally, aside from disgust, higher intensity emotional stimuli were recognized more accurately than lower intensity stimuli.

Present Study

The goal of the current study was to examine the presence of emotion recognition deficits among individuals psychometrically identified as being at-risk for developing schizophrenia. The present study sought to expand upon previous research on endophenotypes of schizophrenia, and specifically examined emotion recognition performance as a potential endophenotype for individuals identified to be psychometrically psychosis prone.

Given that endophenotypes are intermediate factors associated with an increased risk for the development of a disease, they must be observable prior to the onset of signs and symptoms of schizophrenia. As such, participants for the study were selected from a college population ranging in age from 18 to 25, as this age range represents the period of highest risk for the development of schizophrenia. Participants for the current study were identified as psychometric schizotype (schizotypes) on the basis of their CPPS scores, and these schizotypes were matched to control participants who do not possess deviant scores on the CPPS. These control participants were matched on the basis of age, sex, and ethnicity to control for the presence of confounding demographic variables. Emotion recognition was measured by performances on the Penn ER – 40 (Kohler et al., 2003).

Hypotheses

Based on previous research, the following hypotheses were offered:

1. Schizotypes would demonstrate greater deficits in emotion recognition compared to their matched controls.
2. Schizotypes would demonstrate greater deficits in successfully identifying negative emotions and neutral expressions when compared to their matched controls.
3. Individuals possessing a family history of schizophrenia would demonstrate greater deficits in emotion recognition when compared to those without a family history.
4. Emotion recognition deficits would persist after statistically accounting for the variance associated with neurocognitive working memory deficits.
5. Emotion recognition deficits would be strongest for those with greater social anhedonia (i.e., negative schizotypes) scores.

CHAPTER 3

METHODOLOGY

Overview and Design

The current study is a cross-sectional piece of a larger, 10-year longitudinal study assessing psychosis proneness in a college sample. The focus of the current study was to examine deficits in emotion recognition as a potential endophenotype in psychometric schizotypes. Group membership was determined using the Chapman Psychosis Proneness Scales (CPPS). Participants with scores greater than 1.96 standard deviations above the mean on a minimum of one CPPS scale were categorized as a psychometric schizotype (PS) participant. PS participants were compared to an equal number of matched control (MC) participants on emotion recognition, which assessed using the Penn Emotion Recognition Test – 40 (ER40; Kohler et al., 2003). Neurocognitive abilities were assessed using the working memory index (WMI) from the Wechsler Adult Intelligence Scale –IV (WAIS-IV) and the visual working memory index (VWMI) from the Wechsler Memory Scale –IV (WMS-IV).

Power Analysis

An *a priori* power analysis was conducted based on a desired power of .80 and an alpha level of .05. To detect a small effect size, for a global MANOVA with five response variables, 58 participants were needed, with 29 in each group. To detect a medium effect size for between-group comparisons of individual variables, 128 participants were needed, or 64 in each group.

Participants

Participant Screening Process

The initial participant pool consisted of 785 undergraduate students from Indiana State University (ISU) who were recruited from Introductory Psychology courses as part of the larger 10-year longitudinal study. Participants were limited to individuals 18 to 25 years of age, in order for the larger study to follow the participants during the period of greatest risk for development of psychosis.

The following procedures were used for determination of inclusion in the larger, longitudinal study. Participants in the initial pool completed three of the Chapman Psychosis Proneness Scales (*PerAb;* Chapman et al., 1978; *MagId;* Eckblad & Chapman, 1983; *SocAnh;* Eckblad et al., 1982), the Personality Diagnostic Questionnaire –IV (PDQ-4; Hyler, 1994), the Minnesota Multiphasic Personality Inventory -2 (MMPI-2; Butcher, Dahlstrom, Graham, Tellegan, & Kaemmer, 1989). In addition to these measures used for inclusion in the study, they also completed a personal and family history demographic questionnaire, as well as the Penn Emotion Recognition Test -40 (ER40; Kohler et al., 2003), the Edinburg Handedness Inventory (Oldfield, 1971), and the Temporal Experience of Pleasure Scale (TEPS; Gard, Gard, Kring, & John, 2006).

Participant data from the PDQ-IV and CPPS were first assessed for validity using each test's respective manuals. The data on these assessments as well as the MMPI-2 was examined for validity to determine whether they should be included in the final sample, and participants with invalid data on any of these measures were removed from further analyses. For the CPPS, data with three or more *Infrequency* scale item endorsements were excluded from future analyses. Endorsement of more than two items on the *Too Good* scale from the PDQ-IV, and

scores greater than zero on the *Suspect Questionnaire* scale from the PDQ-IV also resulted in exclusions from subsequent analyses. With regard to the MMPI-2, moderate validity criteria were implemented to prevent the exclusion of potentially valid profiles that may be indicative of more severe pathology, as opposed to invalid profiles. Exclusion criteria used for the MMPI-2 were as follows: *VRIN* ≥ 13, *TRIN* < 5 or > 13, *F*≥ 30, *Fb* > 20, *Fp* T score > 120, *Cannot Say* ≥ 20, and *L* T score ≥ 83.

After the exclusion of invalid profiles, the remaining participant data were assessed for deviancy on the CPPS. Those with scores greater than 1.96 standard deviations above the mean on any CPPS scale were assigned to the PS group. MC participants were subsequently chosen for each PS participant utilizing the method of Bolinskey and Gottesman (2010) and Hunter et al. (2014) for matching participants on gender, ethnicity, age, and their college major (respectively) from the remainder of participants with valid profiles. In the event that it was not possible to match the control participant based on all of the four criteria, MCs with the closest match (e.g., match on as many demographics as possible) to the PS participant were chosen. PS participants and their MC were subsequently invited to participate in the second phase of the study where they completed the WAIS-IV, WMS-IV, and other measures not utilized for the current study. Those who participated in the second phase of the study received $20 for their participation.

Final sample

The final sample consisted of 568 college students from a rural, Midwestern university enrolled in introduction to psychology. Participants' ages in the final sample ranged from 18 to 24 years (M = 19.11; SD = 1.05). Female participants (N = 409) comprised 72% of the sample, and male participants comprising the remaining 28% (N = 159). With regard to ethnicity, 78.9% (N = 448) of participants identified as Caucasian, 14.1% (N = 80) as African American, 2.6% (N

= 15) as other, 2.5% (*N* = 14) as Hispanic, 1.6% (*N* = 9) as Asian, and 0.4% (*N* = 2) as Native American.

Of the 568 participants included in the final sample, 86 individuals were identified as PS based on their scores on the CPPS, and 86 additional participants were selected to be in the MC group. All of these participants were used in the present study Of the 86 participants identified as PS, 8 possessed deviant scores on the *MagId* scale, 5 were deviant on the *PerAb* scale, and 78 participants possessed deviant scores on the *SocAnh* scale. Among PS participants, the mean score on *SocAnh* for those deviant on the scale was 20.14 (*SD* = 5.14). The mean score of PS participants who scored in the deviant range on the *PerAb* scale was 7.13 (*SD* = 5.80), and the mean score of those deviant on the *MagId* scale was 10.60 (*SD* = 6.29). The age of PS participants ranged from 18 to 22 years (*M* = 19.03, *SD* = 0.98); 87.2% (*N* = 75) of PS participants were female, whereas 12.8% (*N* = 11) were male. With regard to ethnic group membership of the PS sample, 77.9% (*N* = 67) participants identified as Caucasian, 17.4% (*N* = 15) as African American, 2.3% (*N* = 2) as Hispanic, 1.2% (*N* = 1) as Asian, and 1.2% (*N* = 1) as other.

For the MC group, mean scores on the Chapman scales were 7.10 (*SD* = 4.70) on the *MagId* scale, 3.66 (*SD* = 3.16) on the *PerAb* scale, and 8.74 (*SD* = 4.70) on the *SocAnh* scale. The age of MC participants ranged from 18 to 21 years (*M* = 18.85; *SD* = 0.77); 84.9% (*N* = 73) of MC participants were female, and the remaining 15.1% (*N* = 13) were male. With regard to ethnicity, 75.6% (*N* = 65) of MC participants identified as Caucasian, 22.1% (*N* = 19) as African American, 1.2% (*N* = 1) as Hispanic, and 1.2% (*N* = 1) as other.

Measures

Demographic Questionnaire

All participants completed a 60-item demographic questionnaire that included items about the participants' age, gender, ethnicity and college major, as well as questions that asked about the participant's developmental history, relationship history, alcohol and drug use, prior treatment of mental illness, family history of mental illness (e.g., schizophrenia), and hand dominance.

Chapman Psychosis Proneness Scales (CPPS)

The CPPS include three subscales that are utilized to assess for both positive and negative symptoms of schizotypy. These subscales include the Revised Social Anhedonia Scale (*SocAnh*; Eckblad et al., 1982), the Perceptual Aberration Scale (*PerAb;* Chapman et al., 1978), and the Magical Ideation Scale (*MagId*; Eckblad & Chapman, 1983). As noted above, these scales were used to determine group membership in the parent study. See appendix D for gender-based descriptive statistics and cutoff scores for each scale.

Test-retest reliability coefficients by Chapman and colleagues were reported to be between .75 and .85 for these scales. Additionally, these three subscales were constructed to maximize internal consistency and construct validity by ensuring that only items with low correlations with social desirability and acquiescence were included in final versions of the scales (Chapman et al., 1994). Descriptions of each scale follow.

The Social Anhedonia Scale (*SocAnh*). The *SocAnh* scale consists of 40 true-false items that assess for social disinterest associated with schizotypy. The scale was later revised to limit items that assessed social anxiety (Chapman & Chapman, 1985). Scores on *SocAnh* range from 0 to 40 with deviance cutoff scores differing across genders (women ≥ 16, men ≥ 20). Cronbach's

alpha for both genders was .79 (Kwapil, Barrantes-Vidal, & Silvia, 2008). Women with a score of 16 or greater, and men with a score of 20 or greater were identified as *SocAnh* schizotypes.

The Perceptual Aberration Scale (*PerAb*). The *PerAb* scale consists of 35 true-false items that assess for unusual perceptions of one's body related to schizophrenia-spectrum pathology, including odd interpretations of external stimuli and physical perceptions (Chapman & Chapman, 1985). Scores on *PerAb* range from 0 to 35 with deviant scores of ≥ 19 for both genders (Kwapil et al., 2008). The alpha coefficient for the *PerAb* scale was found to be 0.89, and the scale has also been demonstrated to have high test-retest reliability (r = 0.75; Lenzenweger, 2010).

The Magical Ideation Scale (*MagId*). The *MagId* scale consists of 30 true-false items that measure sub-clinical delusional beliefs that are considered to be magical and culturally invalid (Chapman & Chapman, 1985). Scores on *MagId* range from 0 to 30, with scores ≥ 21 and ≥ 22 being classified as deviant for women and men, respectively. Cronbach's alpha for the scale differs across genders, with 0.83 for women and 0.85 for men (Kwapil et al., 2008).

The Penn Emotion Recognition Test-40 (ER40)

The Penn Emotion Recognition Test-40 (ER40; Kohler et al., 2003) is an abbreviated version of the 96-item Penn Emotion Recognition Test. Both computer-based tests are widely used in schizophrenia research to assess emotion recognition abilities. The 96-item test consists of 96 color photographs of faces displaying happy, sad, angry, fearful, disgusted, and neutral expressions. The test includes eight high and low intensity expressions for each emotion and 16 neutral expressions. The Penn Emotion Recognition Test-40 includes 40 color photographs of faces demonstrating a variety of emotional (happy, sad, angry, or fearful) and nonemotional (neutral) displays. Participants are asked to choose the emotional label that best matches the

displayed face. The test utilizes a forced-chose format where participants must label each face presented from the options “happy,” “sad,” “angry,” “fearful,” or “neutral.” As with the 96-item test, the faces displayed include both low intensity and high intensity emotional displays from men and woman, as well as Caucasians and non-Caucasians (African American, Asian, and Hispanic). Performance on the ER40 for the current study is based on the participant’s accuracy in identifying emotional and neutral facial stimuli. Previous research has additionally assessed performance on the task based on the response speed of the participants (Eack et al., 2010).

Wechsler Adult Intelligence Scale – IV (WAIS-IV)

The WAIS-IV (Wechsler, 2008a, 2008b) is a commonly used intelligence test used for individuals aged 16-90. The WAIS is reviewed and re-standardized at regular intervals in order to insure the validity of the measure, and is currently in its fourth edition (Wechsler, 2008a). The WAIS-IV consists of four index scores, which represent the four major components of the construct of intelligence (i.e., Verbal Comprehension Index, Perceptual Reasoning Index, Working Memory Index, and Processing Speed Index). Although the entire test was administered as part of the larger longitudinal study, only the Working Memory Index was utilized for the present study. Cronbach’s alpha for the WMI subtests fall between .88 and .93, and the overall stability coefficient is .87.

The WMI is comprised of two subtests: Digit Span (DS) and Arithmetic (AR). For DS, the examinee is required to repeat a series of numbers back to the examiner, which increase in length after each trial. During DS Forward (DSF), the examinee is required to repeat the numbers back to the examiner in the same order they were given. Conversely, for DS Backward (DSB), the examinee is required to repeat the numbers in reverse order. Lastly, DS Sequencing (DSS) requires the examinee to repeat the numbers in numerical order, starting with the lowest number.

Completion of the DS subtest requires attention, concentration, auditory verbal processing, visuospatial imaging, and mental control and manipulation. Reported test-retest reliability for the DS subtest is .93 (Wechsler, 2008b).

The AR subtest requires the examinee to solve mathematical problems that have been presented to them orally. Responses are time limited, and examinees are not permitted to use written calculations to assist them in solving the problems. Like DS, the AR subtest requires concentration and attention, and additionally requires mental manipulation, numerical reasoning, and short and long-term memory. Reported test-retest reliability for the AR subtest is .88 (Wechsler, 2008b).

Wechsler Memory Scale –IV (WMS-IV)

The WMS-IV (Wechsler, 2009a) is a commonly used neuropsychological measure of memory functioning in individuals aged 16 to 90 years. Currently, the WMS is in its fourth edition, and the measure was designed and co-normed for use with the WAIS-IV. The WMS-IV possesses moderate to high internal consistency, with a stability coefficient of .72 (Wechsler, 2009b). The WMS-IV contains five indices: Visual Working Memory (VWMI), Visual Memory (VMI), Auditory Memory (AMI), Immediate Memory (IMI), and Delayed Memory (DMI). The current study utilized the VWMI, which consists of two subtests: spatial addition (SA) and symbol span (SS). SA requires that examinees recall a design of colored dots in a variety of spatial organizations after briefly being presented with the design. The examinee must selectively attend to relevant stimuli, while ignoring competing stimuli. The SA subtest was designed to measure spatial working memory and the ability to retain and manipulate information. Test-retest reliability for SA is .91 (Wechsler, 2009b).

The SS subtest is designed to be a visual nonverbal analog to the DS subtest in the WAIS-IV. During SS, the examinee is required to remember and reproduce a series of symbols in the correct order. With each subsequent trial, the complexity and number of symbols in the target sequence increases. The SS subtest assesses an individual's ability to retain mental images and to determine an object's relative position. Test-retest reliability for SS is .85, and Cronbach's alpha is reported at .88 (Wechsler, 2009b).

Procedures

As noted above, participants for the current study were recruited as a part of a larger 10-year longitudinal study assessing for a variety of deficits in a psychometric schizotype (PS) sample. Participants were informed that the purpose of the study is to examine "Mental Health Development in Early Adulthood," in order to prevent any undue stress or stigma that might result from the use of the terminology "psychosis" or "schizotype."

Informed consent was obtained prior to data collection. Following the informed consent, participants were instructed to complete contact forms so that they may be contacted in the future for the longitudinal aspect of the study. All personal contact information is stored in a password-protected database on a computer stored in a locked room. All participants were assigned unique personal identification numbers that were used for all subsequent data collection. These personal identification numbers and collected data are stored separately from participant personal contact information and stored in a locked file cabinet in a locked room.

Data collection for the larger longitudinal study occurred at two time points. During the first time point, participants completed the contact form, MMPI-2, and PDQ-IV. At this time, participants were also asked to complete additional assessments online within 48 hours of the initial appointment. The online assessments included the CPPS, a family history demographic

questionnaire, the ER40, the Edinburg Handedness Inventory, and the TEPS. At the initial appointment, participants were also informed that approximately 20% of participants will be invited to participate in the second phase of the study, and that these participants will receive $20 for their participation. As previously stated, participants with valid data with scores greater than 1.96 standard deviations on the CPPS were identified as PS. PS participants were then matched to a participant whose scores fall within the normal range on the CPPS. The principle investigator of the longitudinal study, who was not involved in data collection, assigned group membership. All graduate research assistants involved in data collection were blind to participant group membership (PS or MC).

A graduate research assistant contacted participants chosen to continue with the second phase of the study via phone or email to schedule the second data collection time point. Participants who elected to continue with the second phase of the study completed the WAIS-IV, WMS-IV, and additional assessments not used for the present study.

Statistical Analyses

All statistical analyses were performed utilizing the IBM SPSS Statistics 20.0 package (SPSS, 2011). In order to describe the sample in terms of age, gender, and ethnicity, descriptive statistics were calculated. Since participants were matched on the basis of these demographic variables, between-group analyses of these demographic variables were not conducted. Pearson correlations were calculated in order to determine the relationship between demographic variables and performance on the ER40 and CPPS.

To assess for muticollinearity, Pearson analyses were calculated to examine the relationship between the dependent variables happy, sad, angry, fearful, and neutral. This analysis was performed in order to ensure that each variable makes a unique contribution to

further analyses and is not redundant. Given that the correlations between dependent variables were not excessive, MANOVA was utilized to test the first three hypotheses and hypothesis five. General linear models (GLMs) incorporating contrast codes for the presence and type of schizotypy and scores on neurocognitive measures were used to test the fourth hypothesis that statistically significant emotion recognition deficits would persist after accounting for the variance associated with neurocognitive performance.

CHAPTER 4

RESULTS

Eighty-six participants were identified for membership in the PS group. Of these 86 participants, 78 had scores within the deviant range on *SocAnh*, 8 were deviant on *MagId*, and 5 were deviant on *PerAb*.

Pearson correlation analyses revealed low to modest correlations among the primary dependent variables of the abilities to recognize happiness, sadness, anger, fear, and neutral emotional displays (see Table 1). The lack of high (i.e., above .70) correlations among the variables indicates a lack of multicollinearity. Thus, it was permissible to proceed with multivariate analyses including each of the variables.

Comparisons of PS and MC participants

To assess the hypothesis that schizotypes will demonstrate greater overall deficits in emotion recognition when compared to their matched controls, MANOVA was conducted. The results of this analysis indicated no significant differences between schizotypes and their matched controls in their abilities to correctly identify emotions. Table 2 displays the mean scores by group with associated results and effect sizes.

This MANOVA also assessed the second hypothesis that schizotypes will demonstrate greater deficits in identifying negative (e.g., anger, fear, sadness) and neutral expressions when compared to their matched controls. Although the results of this analysis were not significant, a

Table 1:

Pearson correlations between primary dependent variables.

	Happiness	*Sadness*	*Anger*	*Fear*	*Neutral*
Happiness	-				
Sadness	.141	-			
Anger	.161	.078	-		
Fear	.245	.133	.056	-	
Neutral	.039	.061	-.015	.167	-

Table 2:

Emotion recognition by total score, valence, and type by group.

Emotion	*Group*	*Mean*	*SD*	*F*	*d*
Total	Normal	34.70	3.44	0.26	0.08
	Schizotype	34.45	2.75		
Negative	Normal	19.53	2.62	0.04	-0.03
	Schizotype	19.60	2.15		
Anger	Normal	5.88	1.29	1.82	0.21
	Schizotype	5.60	1.42		
Fear	Normal	7.12	1.34	3.28	-0.28
	Schizotype	7.42	0.77		
Sadness	Normal	6.53	1.05	0.08	-0.04
	Schizotype	6.58	1.11		
Happiness	Normal	7.85	0.79	0.49	0.11
	Schizotype	7.78	0.47		
Neutral	Normal	7.31	1.15	1.66	0.20
	Schizotype	7.07	1.33		
Female	Normal	17.78	2.11	0.29	0.08
	Schizotype	17.63	1.52		
Male	Normal	16.92	1.85	0.11	0.05
	Schizotype	16.83	1.76		

Note: $N = 86$ for both Normal and Schizotype groups. $^{*} = p < .05$ $^{**} = p < .01$

small effect size (d = 0.20) was detected, indicating less accuracy within the schizotype group (M = 7.07, SD = 1.33) for correctly identifying neutral expressions when compared to the matched controls (M = 7.31, SD = 1.15). Similarly, a small effect size (d = 0.21) was also detected in the hypothesized direction for angry expressions, with schizotypes demonstrating less accuracy (M = 5.60, SD = 1.42) in their ability to correctly identify these expressions when compared to the matched control group (M = 5.88, SD = 1.29). Lastly, a small effect size (d = -0.28) opposite the hypothesized direction was observed for fearful expressions, with schizotypes (M = 7.42, SD = 0.77) outperforming their matched control counterparts (M = 7.12, SD = 1.34) in their ability to correctly discern and identify fearful expressions.

Emotional display intensity

A subsequent MANOVA was conducted to determine whether schizotypes and their matched controls significantly differed in their ability to identify emotional displays of different intensities (i.e., moderate or extreme). Table 3 displays the mean scores by group with associated results and effect sizes for the moderate and extreme emotional displays. Results of this analysis indicated that the schizotypes (M = 3.86, SD = 0.38) significantly outperformed matched controls (M = 3.64, SD = 0.72) in their ability to correctly identify extreme displays of fear, F (1, 170) = 6.36, p = .013, d = -0.38. No other significant group differences were detected for either extreme or moderate displays. However, a small effect size (d = 0.21) was detected for moderate anger displays, indicating that schizotypes identified these emotional displays less accurately (M = 2.19, SD = 1.00) than the matched control group (M = 2.40, SD = 0.97).

Comparisons of family history and no family history participants

To test the third hypothesis that individuals with a family history of schizophrenia will demonstrate greater emotion recognition deficits when compared to their matched controls,

Table 3:

Emotion recognition by total score, valence, and type, by presence or absence of schizotypy.

Emotion	Group	*M*	*SD*	*F*	*d*
Moderate	Normal	12.59	1.98	0.43	0.10
	Schizotype	12.41	1.72		
Negative	Normal	9.50	1.75	0.01	0.01
	Schizotype	9.48	1.62		
Anger	Normal	2.40	0.97	1.93	0.21
	Schizotype	2.19	1.00		
Fear	Normal	3.48	0.82	0.55	-0.11
	Schizotype	3.56	0.61		
Sadness	Normal	3.63	0.84	0.52	-0.11
	Schizotype	3.73	1.05		
Happiness	Normal	3.90	0.49	0.73	0.13
	Schizotype	3.84	0.40		
Extreme	Normal	14.79	1.58	0.77	-0.13
	Schizotype	14.98	1.18		
Negative	Normal	10.03	1.25	0.28	-0.08
	Schizotype	10.13	1.06		
Anger	Normal	3.49	0.63	0.35	0.09
	Schizotype	3.42	0.89		
Fear	Normal	3.64	0.72	6.36	-0.38
	Schizotype	3.86	0.38		
Sadness	Normal	2.91	0.39	0.94	0.15
	Schizotype	2.85	0.39		
Happiness	Normal	3.95	0.34	0.07	0.04
	Schizotype	3.94	0.24		

Note: N = 86 for both Normal and Schizotype groups. * = $p < .05$ ** = $p < .01$

a MANOVA was also conducted. Table 4 displays the mean scores by group with associated results and effect sizes. Results of this analysis indicated that those with a family history of schizophrenia (M = 7.65, SD = 0.81) performed worse than those without a family history of schizophrenia (M = 7.88, SD = 0.39) in their ability to correctly identify expressions of happiness, F (1, 566) = 6.76, p = .010, d = 0.48. Those with a family history of schizophrenia (M = 18.84, SD = 3.90) also performed worse than those without a family history (M = 19.71, SD = 2.05) in their ability to correctly identify negative emotions, F (1, 566) = 4.67, p = .031, d = 0.40. Although the analysis did not yield any other significant differences between the schizotype and matched control group, several small effect sizes were detected, suggesting less accurate emotional display identification by the schizotypes. Notably, these small effect sizes included an overall (d = 0.29) deficit in emotion recognition accuracy for those with a family history of schizophrenia (M = 33.97, SD = 4.84) when compared to those without a family history (M = 34.76, SD = 2.60). Small effects were also noted for the identification of fear (d = 0.23), with less accurate identification by those with a family history (M = 7.06, SD = 1.53) when compared to those without a family history (M = 7.29, SD = 0.96), and sadness (d = 0.34), where individuals with a family history also performed worse (M = 6.26, SD = 1.63) than those without a family history (M = 6.65, SD = 1.14). Additionally, a small effect size (d = 0.31) was also noted for female participants with a family history of schizophrenia (M = 17.32, SD = 2.76) proving to be less accurate in their identification of emotion than female participants without a family history (M = 17.83, SD = 1.54). Finally, there was one small effect size (d = -0.26) observed counter to the hypothesized prediction. Those with a family history of schizophrenia (M = 7.48, SD = 0.81) proved to be more accurate in their identification of neutral expressions than participants without a family history of the disorder (M = 7.17, SD = 1.22).

Table 4:

Emotion recognition by total score, valence, and type by presence or absence of family history of schizophrenia.

Emotion	*Group*	*Mean*	*SD*	*F*	*d*
Total	No Family History	34.76	2.60	2.41	0.29
	Family History	33.97	4.84		
Negative	No Family History	19.71	2.05	4.67*	0.40
	Family History	18.84	3.90		
Anger	No Family History	5.77	1.30	1.09	0.19
	Family History	5.52	1.75		
Fear	No Family History	7.29	0.96	1.50	0.23
	Family History	7.06	1.53		
Sadness	No Family History	6.65	1.14	3.31	0.34
	Family History	6.26	1.63		
Happiness	No Family History	7.88	0.39	6.76**	0.48
	Family History	7.65	1.28		
Neutral	No Family History	7.17	1.22	1.96	-0.26
	Family History	7.48	0.81		
Female	No Family History	17.83	1.54	2.84	0.31
	Family History	17.32	2.76		
Male	No Family History	16.93	1.67	0.82	0.17
	Family History	16.65	2.33		

Note: N = 537 for No Family History of schizophrenia group. N = 31 for Family History of schizophrenia group. * = $p < .05$ ** = $p < .01$

Emotional display intensity

An additional MANOVA was conducted to assess whether participants with a family history of schizophrenia significantly differed in their ability to identify facial expressions depending on the intensity (i.e., moderate or extreme) of the emotional display. The results of this analysis are displayed in Table 5. This analysis yielded a number of significant findings, all of which were consistent with the hypothesized direction. Individuals with a family history of schizophrenia (M = 3.74, SD = 0.77) were significantly less accurate in their identification of moderate displays of happiness than those lacking a family history (M = 3.91, SD = 0.29) of schizophrenia, F (1, 566) = 7.70, p = .006, d = 0.51.

Significant overall effects and a moderate effect size were also found for extreme emotional displays, with those possessing a family history (M = 14.23, SD = 2.43) proving to be less accurate in identifying extreme emotional displays than those without (M = 14.93, SD = 1.12) a family history, F (1, 566) = 9.73, p = .002, d = 0.58. In addition, the results also yielded a significant effect for extreme displays of negative emotions (i.e., anger, fear, sadness). A moderate effect size (d = 0.49) was noted for these extreme displays of negative emotions, F (1, 566) = 7.07, p = .008, again with participants with a family history (M = 9.61, SD = 1.78) less accurately identifying these emotions than participants without a family history (M = 10.13, SD = 0.99). A significant effect was also detected for extreme displays of a specific emotion. Participants with a family history (M = 2.68, SD = 0.70) were less accurate than participants without a family history (M = 2.86, SD = 0.41) in their ability to correctly identify extreme displays of sadness, F (1, 566) = 5.27, p = .022, d = 0.42.

In addition to these significant effects reported, the analysis also yielded a number of small effect sizes in the hypothesized direction for both moderate and extreme emotional

Table 5:

Emotion recognition by total score, valence, and type, split by intensity of emotional display and by presence or absence of family history of schizophrenia.

Emotion	Group	*M*	*SD*	*F*	*d*
Moderate	No Family History	12.66	1.59	1.62	0.24
	Family History	12.26	2.92		
Negative	No Family History	9.58	1.60	1.38	0.22
	Family History	9.23	2.39		
Anger	No Family History	2.28	0.98	0.01	0.02
	Family History	2.26	1.29		
Fear	No Family History	3.52	0.70	0.96	0.18
	Family History	3.39	0.92		
Sadness	No Family History	3.79	1.01	1.26	0.21
	Family History	3.58	1.12		
Happiness	No Family History	3.91	0.29	7.70**	0.51
	Family History	3.74	0.77		
Extreme	No Family History	14.93	1.12	9.73**	0.58
	Family History	14.23	2.43		
Negative	No Family History	10.13	0.99	7.07**	0.49
	Family History	9.61	1.78		
Anger	No Family History	3.49	0.72	3.12	0.33
	Family History	3.26	0.77		
Fear	No Family History	3.77	0.50	1.04	0.19
	Family History	3.68	0.79		
Sadness	No Family History	2.86	0.41	5.27*	0.42
	Family History	2.68	0.70		
Happiness	No Family History	3.96	0.22	1.58	0.23
	Family History	3.90	0.54		

Note: N = 537 for No Family History of schizophrenia group. N = 31 for Family History of schizophrenia group. * = $p < .05$ ** = $p < .01$

displays. With regard to moderate emotional displays, an overall small effect size (d = 0.24) was observed for these expressions, with individuals with a family history (M = 12.26, SD = 2.92) identifying these moderate displays less accurately than those without a family history (M = 12.66, SD = 1.59). A small effect (d = 0.22) was also noted for moderate expressions of negative emotions (i.e., anger, fear, and sadness), again with participants without a family history (M = 9.58, SD = 1.60) outperforming those with a family history (M = 9.23, SD = 2.39) of schizophrenia. Similarly, a small effect size (d = 0.21) was detected for the identification of moderate displays of sadness with those with a family history (M = 3.58, SD = 1.12) demonstrating less accuracy in the identification of these expressions than those without a family history (M = 3.79, SD = 1.01).

Small effect sizes were also observed for extreme emotional displays as well. These effect sizes were noted for the extreme expression of both anger (d = 0.33) and happiness (d = 0.23). Participants with a family history performed worse (M = 3.26, SD = 0.77) on the identification of extreme anger displays when compared to those without a family history (M = 3.49, SD = 0.72). Similarly, the participants with a family history (M = 3.90, SD = 0.54) were also less accurate in their identification of happiness than those without family histories (M = 3.96, SD = 0.22).

Comparisons of positive and negative schizotypes

To test the fifth hypothesis, that emotion recognition deficits will be strongest for negative schizotypes (i.e., those scoring in the deviant range on *SocAnh*) when compared to positive schizotypes (i.e., those scoring in the deviant range on *PerAb* or *MagId*), a MANOVA was conducted. Table 6 displays the mean scores by group with associated results and effect sizes. The results of this analysis yielded several significant results, albeit counter to the

hypothesized direction. Overall, positive schizotypes (M = 32.82, SD = 3.09) were significantly less accurate than negative schizotypes (M = 34.69, SD = 2.63) in their identification of emotions, F (1, 84) = 4.66, p = .034, d = -0.70. In addition to this overall deficit in their ability to correctly identify emotions, positive schizotypes (M = 18.27, SD = 2.05) were also significantly less accurate than negative schizotypes (M = 19.80, SD = 2.11) in their identification of negative emotions, F (1, 84) = 5.05, p = .027, d = -0.73. A significant effect was also found specific to the identification of displays of sadness, again with positive schizotypes (M = 5.64, SD = 1.29) performing worse than negative schizotypes (M = 7.45, SD = 0.78) in their ability to correctly identify these displays, F (1, 84) = 10.10, p = .002, d = -1.03. In addition to deficits in the identification of negative emotions such as sadness, positive schizotypes (M = 16.45, SD = 1.29) were significantly less accurate than negative schizotypes (M = 17.80, SD = 1.48) in their ability to correctly identify emotional expressions presented using female faces, F (1, 84) = 8.16, p= .005, d = -0.92. It is also of importance to highlight that not only were statistically significant differences detected between positive and negative schizotypes for the overall identification of emotions, negative emotions, sadness, and female displays, but large effect sizes for these relationships were also detected.

In addition to the significant differences uncovered between positive and negative schizotypes, a number of small effect sizes were detected among several of the remaining variables that failed to reach statistical significance. The first of these small effect sizes (d = -0.35) was for fearful emotional expressions. Positive schizotypes (M = 7.18, SD = 0.75) were less accurate in identifying these expressions than negative schizotypes (M = 7.45, SD = 0.78). A second small effect (d = -0.30) was also observed for neutral expressions, again with the positive schizotypes (M = 6.73, SD = 1.62) less accurately identifying these expressions when

Table 6:

Emotion recognition by total score, valence, type, and sex of individual demonstrating the emotion by positive and negative schizotypy.

Emotion	*Group*	*Mean*	*SD*	*F*	*d*
Total	Positive Schizotype	32.82	3.09	4.66*	-0.70
	Negative Schizotype	34.69	2.63		
Negative	Positive Schizotype	18.27	2.05	5.05*	-0.73
	Negative Schizotype	19.80	2.11		
Anger	Positive Schizotype	5.45	1.29	0.14	-0.12
	Negative Schizotype	5.63	1.45		
Fear	Positive Schizotype	7.18	0.75	1.18	-0.35
	Negative Schizotype	7.45	0.78		
Sadness	Positive Schizotype	5.64	1.29	10.10**	-1.03
	Negative Schizotype	6.72	1.02		
Happiness	Positive Schizotype	7.82	0.40	0.09	0.09
	Negative Schizotype	7.77	0.48		
Neutral	Positive Schizotype	6.73	1.62	0.84	-0.30
	Negative Schizotype	7.12	1.28		
Female	Positive Schizotype	16.45	1.29	8.16**	-0.92
	Negative Schizotype	17.80	1.48		
Male	Positive Schizotype	16.36	2.34	0.86	-0.30
	Negative Schizotype	16.89	1.67		

Note: $N = 11$ for Positive Schizotype group. $N = 75$ for Negative Schizotype group. * = $p < .05$ ** = $p < .01$

compared to the negative schizotypes (M = 7.12, SD = 1.28). Finally, a small effect (d = -0.30) size was also detected for the identification of emotions displayed using male faces. Positive schizotypes (M = 16.36, SD = 2.34) were outperformed by negative schizotypes (M = 16.89, SD = 1.67), who more accurately identified the emotional expressions displayed using male faces.

Emotional display intensity

An additional MANOVA was performed to detect whether significant differences in emotion recognition existed between positive and negative schizotypes as a function of emotional intensity. These results can be viewed in Table 7. The results of this analysis yielded several significant effects as well as small effect sizes among a number of variables that failed to reach statistical significance. Consistent with the previous analysis, each of the significant results detected were counter to the hypothesized direction. Significant differences were found for the overall identification of moderate displays of emotion, with positive schizotypes (M = 11.36, SD = 1.75) less accurate than negative schizotypes (M = 12.56, SD = 1.67) in their identification of emotional displays of moderate intensity, F (1, 84) = 4.87, p = .03, d = -0.70. Similarly, a significant difference was also found for the identification of moderate displays of negative emotions. Again, positive schizotypes (M = 8.36, SD = 1.63) correctly identified these expressions less frequently than their negative schizotype (M = 9.64, SD = 1.57) peers, F (1, 84) = 6.32, p = .014, d = -0.81. Positive schizotypes (M = 2.91, SD = 1.38) were also significantly less accurate in the identification of moderate displays of sadness, F (1, 84) = 8.52, p = .005, d = -0. 94, when compared to negative schizotypes (M = 3.85, SD = 0.94).

Although the analysis yielded no further significant differences between the positive and negative schizotypes in emotion recognition, a number of small effect sizes were observed among some of the other dependent variables. For moderate displays of emotion, each of the

Table 7:

Emotion recognition by total score, valence, type, and sex of individual demonstrating the emotion by group, split by intensity of emotional display, by positive and negative schizotypy.

Variable	*Group*	*M*	*SD*	*F*	*d*
Moderate	Positive Schizotype	11.36	1.75	4.87*	-0.71
	Negative Schizotype	12.56	1.67		
Negative	Positive Schizotype	8.36	1.63	6.32*	-0.81
	Negative Schizotype	9.64	1.57		
Anger	Positive Schizotype	2.00	0.89	0.43	-0.21
	Negative Schizotype	2.21	1.02		
Fear	Positive Schizotype	3.45	0.52	0.37	-0.20
	Negative Schizotype	3.57	0.62		
Sadness	Positive Schizotype	2.91	1.38	8.52**	-0.94
	Negative Schizotype	3.85	0.94		
Happiness	Positive Schizotype	3.91	0.30	0.40	0.20
	Negative Schizotype	3.83	0.42		
Extreme	Positive Schizotype	14.73	1.10	0.56	-0.24
	Negative Schizotype	15.01	1.19		
Negative	Positive Schizotype	9.91	0.83	0.53	-0.24
	Negative Schizotype	10.16	1.09		
Anger	Positive Schizotype	3.45	0.69	0.02	0.05
	Negative Schizotype	3.41	0.92		
Fear	Positive Schizotype	3.73	0.47	1.55	-0.40
	Negative Schizotype	3.88	0.37		
Sadness	Positive Schizotype	2.73	0.47	1.22	-0.36
	Negative Schizotype	2.87	0.38		
Happiness	Positive Schizotype	3.91	0.30	0.24	-0.16
	Negative Schizotype	3.95	0.23		

Note: $N = 11$ for Positive Schizotype group. $N = 75$ for Negative Schizotype group. $^{*} = p < .05$ $^{**} = p < .01$

remaining emotions (i.e., anger, fear, happiness) yielded small effect sizes. Positive schizotypes were found to be less accurate (M = 2.0, SD = 0.89) than negative schizotypes (M = 2.21, SD = 1.02) when identifying anger displays of moderate intensity (d = -0.21). Similarly, positive schizotypes (M = 3.45, SD = 0.52) were also less accurate than negative schizotypes (M = 3.57, SD = 0.62) in the identification of moderate displays of fear (d = -0.20). As previously noted, the finding that negative schizotypes outperformed the positive schizotypes in the correct identification of these emotions was counter to the hypothesized direction. However, a small effect (d = 0.20) in the hypothesized direction was observed for the identification of moderate displays of happiness, with negative schizotypes (M = 3.83, SD = 0.42) less accurately identifying these expressions when compared to the positive schizotypes (M = 3.91, SD = 0.30).

The remainder of the small effects observed for extreme emotional displays were consistent with the previously reported results, counter to the hypothesized direction. A small effect (d = -0.24) was noted for the identification of extreme emotional expressions overall, with positive schizotypes (M = 14.73, SD = 1.10) demonstrating worse performances than negative schizotypes (M = 15.01, SD = 1.19). Extreme displays of negative emotions (d = -0.24) were also less frequently identified as correct by the positive schizotypes (M = 9.91, SD = 0.83) than the negative schizotypes (M = 10.16, SD = 1.09). Finally, small effect sizes were also noted for the identification of extreme displays of fear and sadness. Positive schizotype participants were again less accurate (M = 3.73, SD = 0.47) than negative schizotype participants (M = 3.88, SD = 0.37) when identifying extreme displays of fear (d = -0.40). Likewise, positive schizotypes (M = 2.73, SD = 0.47) were also less correct than negative schizotypes (M = 2.87, SD = 0.38) in labeling extreme displays of sadness (d = -0.36). Significant differences or effect sizes were not observed for extreme displays of anger or happiness.

To further examine the relationship between CPPS scores and emotion recognition scores, bivariate correlations were calculated for each type of emotion recognition and each CPPS scale score for the entire sample (see Table 8). No significant correlations were found for any type of emotion recognition and *SocAnh* scores. Higher scores on *PerAb* were associated with poorer performance for total emotion recognition, sadness, neutral expressions, emotion recognition in female target stimuli, and recognition of moderate sadness. Higher scores on *MagId* were associated with poorer performance for recognition of sadness and moderate sadness.

Neurocognition and emotion recognition

Correlations and a GLM analysis were utilized to test the hypothesis that the significant emotion recognition deficits from the prior analyses would persist after statistically accounting for the variance associated with neurocognitive deficits. Results of the correlation analysis can be seen in Table 9.

Briefly, the WMI and the AR subscale of the WMI from the WAIS-IV, showed significant correlations with measures of emotion recognition. Both the WMI and AR subscale demonstrated modest relationships with total emotion recognition, negative emotion recognition, emotion recognition in males, and recognition of moderate-level emotions. Additionally, AR but not the WMI also possessed a modest relationship with the recognition of moderate anger. It is noted that the relationships were nominally, but consistently stronger, with the AR subscale than the full WMI. As none of the primary predictors had demonstrated a significant relationship with the recognition of emotion in male target stimuli, or with recognition of moderate anger, no further analyses were performed with that target variable. The remaining relationships between WMI and the AR subscale with our predictor variables were further examined using a GLM.

Table 8:

Correlations between emotion recognition scores and CPPS scales.

	CPPS Scale		
Emotion	*SocAnh*	*PerAb*	*MagId*
Total	-.01	-.11**	-.08
Negative	.01	-.07	-.06
Anger	.00	-.01	.04
Fear	.03	-.02	-.06
Sadness	-.01	-.10*	-.10*
Happiness	-.07	-.05	-.04
Neutral	-.01	-.11*	-.06
Female	-.01	-.13**	-.06
Male	.00	-.06	-.07
Moderate	-.03	-.08	-.08
Moderate Negative	.00	-.07	-.08
Moderate Anger	.00	-.02	.01
Moderate Fear	-.01	.00	-.06
Moderate Sadness	.00	-.09*	-.09*
Moderate Happiness	-.06	-.02	-.04
Extreme	.03	-.04	-.01
Extreme Negative	.02	-.03	-.01
Extreme Anger	.01	.01	.06
Extreme Fear	.06	-.03	-.04
Extreme Sadness	-.03	-.06	-.07
Extreme Happiness	-.05	-.07	-.03

Note: $N = 568$. * = $p < .05$ ** = $p < .01$

Table 9:

Correlations between emotion recognition scores and neurocognitive measure scores.

Emotion	*WMI*	*DS*	*AR*	*VWMI*	*SA*	*SS*	*PE*	*CC*	*Trials*
Total	.21*	.09	.26**	.10	.05	.11	.16	.11	-.03
Negative	.20*	.12	.22*	.11	.07	.11	.12	.07	-.05
Anger	.12	.00	.19*	.18	.06	.23*	.03	.03	-.04
Fear	.13	.15	.08	-.01	.02	-.03	.08	.195	-.19*
Sadness	.14	.12	.12	-.02	.03	-.07	.16	-.04	.11
Happiness	.11	.04	.14	-.15	-.11	-.14	.10	.08	-.03
Neutral	.09	-.01	.15	.08	.03	.10	.11	.10	.03
Female	.11	.03	.14	.12	.11	.09	.13	.06	-.03
Male	.24*	.12	.28**	.05	-.01	.10	.14	.12	-.03
Moderate	.19*	.10	.22*	.07	.03	.09	.15	.09	-.15
Moderate Negative	.20*	.12	.21*	.10	.07	.09	.16	.07	-.12
Moderate Anger	.124	-.01	.20*	.11	.01	.18	.07	.036	-.10
Moderate Fear	.06	.12	-.02	.02	.04	-.016	.08	.26**	-.29**
Moderate Sadness	.15	.12	.14	.02	.07	-.05	.15	-.09	.10
Moderate Happiness	.05	.00	.08	-.11	-.11	-.06	.20	.04	-.04
Extreme	.14	.10	.14	.05	.05	.04	.04	.02	.11
Extreme Negative	.11	.07	.12	.09	.04	.10	.01	.03	.09
Extreme Anger	.05	.02	.08	.18	.10	.19*	-.04	.01	.06
Extreme Fear	.17	.10	.18	-.04	-.02	-.05	.04	-.03	.06
Extreme Sadness	-.01	.03	-.04	-.10	-.08	-.09	.06	.10	.04
Extreme Happiness	.12	.07	.13	-.12	-.04	-.17	-.07	.08	.00

Note: WMI = Working Memory Index score from WAIS-IV. Digit Span = Digit Span subtest scale score from WAIS-IV. AR = Arithmetic subtest scale score from WAIS-IV. VWMI = Visual Working Memory Index score from WMS-IV. SA = Spatial Addition subtest scale score from WMS-IV. SS = Symbol Span subtest scale score from WMS-IV. PE = Perseverative Errors standard score from WCST. CC = Number of Categories Completed standard score from WCST. Trials = number of trials needed to complete first category on WCST. * = $p < .05$ ** = $p < .01$

Given that several of the outcome variables are subsets of other outcome variables, analyses were performed at the univariate level, rather than at the multivariate level. The initial model for each analysis consisted of a single group (positive vs. negative schizotypy) contrast variable and the neurocognitive measure (either WMI or AR) as a covariate whose effects would be accounted for. The second model for each analysis also included a second group (PS vs. MC) contrast score, although the results of the MANOVA assessing for differences between PS and MC participants did not yield significant results in the hypothesized direction.

Since the number of participants with a family history of schizophrenia who completed the neurocognitive measures (N = 12) was not sufficient for subsequent analyses, no follow-up analyses were performed with this group.

WMI and emotion recognition

Results of the correlation analyses between emotion recognition and neurocognitive variables were noted above. Separate analyses were performed for total emotion recognition, negative emotion recognition, recognition of moderate-level emotions, and recognition of moderate negative emotions.

WMI and total emotion recognition score. The initial model examined whether the relationship between schizotypy type (positive or negative) would remain consistent after including WMI scores as a predictor. Results of the GLM analysis revealed that schizotypy type (F (1,55) = 2.99, p = .090, η^2 = .05) no longer remained a significant predictor of total emotion recognition, although WMI score (F (1,55) = 5.94, p = .018, η^2 = .09) continued to serve as a significant predictor.

The second model included a second group contrast code for the presence or absence of schizotypy (PS vs. MC), in general. When this variable was included, both the *presence* (F

(1,109) = 6.85, p = .010, η^2 = .06), and the *type* (F (1,109) = 4.12, p = .045, η^2 = .03) of schizotypy made significant contributions to the prediction of emotion recognition, as did WMI score (F (1,109) = 5.03, p = .027, η^2 = .04).

WMI and recognition of negative emotion. The first model tested whether the relationship between schizotypy type and recognition of negative emotion would remain significant after including WMI scores as a predictor. Results of the GLM analysis revealed that schizotypy type (F (1,55) = 4.526, p = .038, η^2 = .07) did remain a significant predictor of negative emotion recognition, although WMI score (F (1,55) = 3.25, p = .077, η^2 = .05) no longer was a significant predictor.

The group contrast code for the presence or absence of schizotypy was included in the second model. For this model, both the presence (F (1,109) = 5.73, p = .018, η^2 = .05), and type (F (1,109) = 5.908, p = .017, η^2 = .05) of schizotypy made significant contributions to the prediction of emotion recognition. Unlike the first model, WMI score also made a significant contribution to the prediction of negative emotion recognition (F (1,109) = 5.03, p = .027, η^2 = .04).

WMI and recognition of moderate levels of emotion. The initial model examined whether the relationship between schizotypy type (positive or negative) and recognition of moderate levels of emotions would remain consistent after including WMI scores as a predictor. Results of the GLM analysis revealed that schizotypy type (F (1,55) = 8.58, p = .005, η^2 = .12) remained a significant predictor of total emotion recognition, as did WMI score (F (1,55) = 4.65, p = .035, η^2 = .07).

The second model included a second group contrast code for the presence or absence of schizotypy (PS vs. MC), in general. When this variable was included, both presence (F (1,109) =

10.79, $p = .001$, $\eta^2 = .09$) and type (F (1,109) = 9.86, $p = .002$, $\eta^2 = .08$) of schizotypy made significant contributions to the prediction of emotion recognition; however, WMI score (F (1,109) = 3.78, $p = .054$, $\eta^2 = .03$) fell just short of remaining significant.

WMI and recognition of moderate levels of negative emotion. The first model tested whether the relationship between schizotypy type and recognition of moderate levels of negative emotion would remain significant after including WMI scores as a predictor. Results of this analysis revealed that both schizotypy type (F (1,55) = 8.02, $p = .006$, $\eta^2 = .11$) and WMI score (F (1,55) = 5.20, $p = .007$, $\eta^2 = .07$) were significant predictors of total emotion recognition.

The group contrast code for the presence or absence of schizotypy was included in the second model. For this model, both the presence (F (1,109) = 7.02, $p = .009$, $\eta^2 = .06$), and type (F (1,109) = 8.90, $p = .004$, $\eta^2 = .07$) of schizotypy made significant contributions to the prediction of emotion recognition, as did WMI score (F (1,109) = 4.11, $p = .045$, $\eta^2 = .03$).

Arithmetic (AR) and emotion recognition

Results of the correlation analyses between emotion recognition and AR scores were noted above. Separate analyses were again performed for total emotion recognition, negative emotion recognition, recognition of moderate-level emotions, and recognition of moderate-level negative emotions.

AR and total emotion recognition score. The initial model examined whether the relationship between schizotypy type (positive or negative) would remain consistent after including AR scores as a predictor. Results of the GLM analysis revealed that neither schizotypy type (F (1,55) = 3.02, $p = .088$, $\eta^2 = .05$) nor AR score (F (1,55) = 3.61, $p = .063$, $\eta^2 = .06$) remained a significant predictor of total emotion recognition.

The second model included a second group contrast code for the presence or absence of schizotypy (PS vs. MC), in general. The inclusion of this variable led to significant results, as both the *presence* (F (1,109) = 6.03, p = .016, η^2 = .05), and the *type* (F (1,109) = 3.96, p = .049, η^2 = .03) of schizotypy made significant contributions to the prediction of emotion recognition, as did AR score (F (1,109) = 6.92, p = .010, η^2 = .06).

AR and recognition of negative emotion. The first model tested whether the relationship between schizotypy type and recognition of negative emotion would remain significant after including AR scores as a predictor. Results of the GLM analysis revealed that schizotypy type (F (1,55) = 4.67, p = .035, η^2 = .08) did remain a significant predictor of negative emotion recognition, although AR score (F (1,55) = 1.12, p = .295, η^2 = .02) no longer was a significant predictor.

The group contrast code for the presence or absence of schizotypy was included in the second model. For this model, both the presence (F (1,109) = 5.04, p = .027, η^2 = .04), and type (F (1,109) = 5.044, p = .027, η^2 = .04) of schizotypy made significant contributions to the prediction of emotion recognition. Unlike the previous model, AR score also made a significant contribution to the prediction of negative emotion recognition (F (1,109) = 4.76, p = .031, η^2 = .04).

AR and recognition of moderate levels of emotion. The initial model examined whether the relationship between schizotypy type (positive or negative) and recognition of moderate levels of emotions would remain consistent after including AR scores as a predictor. Results of the GLM analysis revealed that schizotypy type (F (1,55) = 8.64, p = .005, η^2 = .13) remained a significant predictor of moderate emotion recognition, but AR score (F (1,55) = 1.44, p = .236, η^2 = .02) did not.

The second model included a second group contrast code for the presence or absence of schizotypy (PS vs. MC), in general. When this variable was included, both presence (F (1,109) = 9.89, p = 0.002, η^2 = .08) and type (F (1,109) = 9.70, p = .002, η^2 = .08) of schizotypy made significant contributions to the prediction of moderate emotion recognition, as did AR score (F (1,109) = 4.61, p = .034, η^2 = .04).

AR and recognition of moderate levels of negative emotion. The first model tested whether the relationship between schizotypy type and recognition of moderate levels of negative emotion would remain significant after including AR scores as a predictor. Results of this analysis revealed that schizotypy type (F (1,55) = 8.09, p = .006, η^2 = .12) remained significant predictors of moderate levels of negative emotion recognition. However, AR score (F (1,55) = 1.40, p = .242, η^2 = .02) was not a significant predictor in this model.

The group contrast code for the presence or absence of schizotypy was included in the second model. For this model, both the presence (F (1,109) = 6.28, p = .014, η^2 = .05), and type (F (1,109) = 8.75, p = .004, η^2 = .07) of schizotypy made significant contributions to the prediction of moderate negative emotion recognition, as did AR score (F (1,109) = 4.41, p = .038, η^2 = .04).

CHAPTER 5

DISCUSSION

Schizotypes and Emotion Recognition

Results of the present study provided support for the hypotheses that PS participants would demonstrate significantly greater deficits in overall emotion recognition and negative emotion recognition when compared to their MCs. Although these significant emotion recognition differences were not detected during the initial MANOVA analyses, significant between group differences were discovered after accounting for the variance associated with neurocognitive performance on the WMI and AR.

Specifically, results of the GLM analyses revealed that both the presence and type (positive or negative) of schizotypy made significant contributions to the prediction of emotion recognition for the overall identification of emotions, the identification of negative emotions (anger, fear, sadness), the identification of moderate emotional displays, and the identification of moderate displays of negative emotion. Additionally, WMI and AR also made significant contributions to the prediction of overall emotion recognition, the recognition of negative emotions, and the recognition of moderate displays of negative emotions. AR, but not WMI, significantly contributed to the prediction of the identification of moderate emotional displays.

In addition to the analyses assessing group differences in emotion recognition between PS and MC participants, separate analyses were also conducted to assess the fifth hypothesis that

sub-group differences would exist between negative (i.e., deviant scores on *SocAnh*) and positive (i.e., deviant scores on *PerAb* or *MagId*) schizotypes. Although results of the current study failed to support the hypothesis that negative schizotypes would be less accurate in their identification of emotions, the analyses yielded several significant findings in the opposite direction, with negative schizotypes outperforming the positive schizotypes.

The negative schizotypes (i.e., *SocAnh* schizotypes) proved to be significantly more accurate in identification of emotional stimuli overall, negative emotions, and sad expressions. Negative schizotypes were also found to be significantly more accurate than positive schizotypes when identifying emotional displays in females. Additional significant effects were also detected for the overall identification of moderate emotional displays, as well as moderate displays of negative emotions, and moderate displays of sadness. Notably, moderate to large effect sizes were also observed for these significant findings, suggesting robust differences between positive and negative schizotypes in emotion recognition accuracy. In addition to these findings, small effects were observed for the majority of other emotional stimuli, including small effects in the detection of fear (moderate and extreme displays), neutral expressions, moderate displays of anger and happiness, extreme emotional displays, extreme negative emotion displays, and extreme displays of sadness. Conversely, those with higher scores on *PerAb* performed significantly worse in their overall ability to identify emotions as well as their ability to identify expressions of sadness, moderate sadness, neutral, and the emotions of female target stimuli. Lastly, higher scores on *MagId* were related to poorer recognition of sadness and moderate sadness expressions.

As with the first two hypotheses, subsequent GLM analyses were conducted to assess the hypothesis that significant emotion recognition deficits would persist after statistically

accounting for the variance associated with neurocognitive performance on WMI and AR. Results of these analyses revealed that relationships between schizotypy type (positive or negative) remained a significant predictor for the identification of negative emotions, moderate emotional displays, and moderate negative emotional displays after accounting for the variance associated with WMI performance. The models revealed WMI, but not schizotypy type, as a significant predictor for total emotion recognition, as well as emotion recognition for moderate displays and moderate displays of negative emotions. Conversely, models that accounted for the variance associated with AR scores demonstrated that schizotypy type, but not AR performance, was a significant predictor for the overall recognition of emotions, recognition of negative emotions, moderate emotions, and moderate displays of negative emotions.

When taken together, the results of these analyses are consistent with previous research that has demonstrated emotion recognition deficits among individuals identified to be at high psychometric risk for schizophrenia (Abbott & Green, 2013; Williams et al., 2007). However, unlike prior research, the present study appears to be the first to also account for the variance associated with neurocognitive performance. Although initial analyses did not yield significant differences between PS and MC participants, differential emotion recognition performances were revealed after accounting for the variance associated with WMI and AR performance. Similar results were achieved in the models for schizotypy type, where schizotypy type remained a significant predictor of emotion recognition for all but one model (total emotion recognition). It appears that neurocognitive functioning obscures the differences between the PS and MC groups. As a result, the current study provides evidence for the presence of a more complex relationship between neurocognition (specifically, WMI and AR performance) and emotion recognition ability than previously observed.

Given that working memory deficits have been widely replicated within schizophrenia patients (e.g., Forbes, Carrick, McIntosh, & Lawrie, 2009), first-degree relatives (e.g., Conklin, Curtis, Katsanis, & Iacono, 2000), and more recently, schizotypy (Hunter, 2014; Matheson & Langdon, 2008; Tallent & Gooding, 1999), these unique findings may be attributable to the presence of neurocognitive deficits in both PS and MC groups. It is conjectured that the MC participants with working memory deficits might in fact represent a separate subset of schizotypes that lack personality liability and instead possess cognitive slippage. Given that the CPPS (i.e., personality liability) were utilized to determine group membership, these "cognitive" schizotypes were instead assigned to the MC group due to their failure to meet the personality liability threshold. Emotion recognition performance among these MC participants may have therefore become compromised as a result of neurocognitive, but not social cognitive deficits. Thus, once the variance associated with WMI and AR performance removed, only the emotion recognition deficits attributable to personality liability (i.e., unique to PS participants) remained. Notably, this potential explanation is consistent with Meehl's (1962) assertion that schizotypy is a product of neurological dysfunctions, which may behaviorally manifest as cognitive slippage, and polygenetic potentiators such as specific personality characteristics.

As previously mentioned, in addition to this novel finding on the relationship between neurocognition and emotion recognition, results of the present study also provided differential support for the specific schizotypy symptoms associated with greater emotion recognition deficits. Emotion recognition deficits were found to be strongest among positive schizotypes (i.e., those who scored in the deviant range on *PerAb* or *MagId*), whereas previous research has demonstrated decreased emotion recognition accuracy to be specific to negative and interpersonal aspects of schizotypy as opposed to positive, disorganized, or cognitive-perceptual

aspects of schizotypy (Abbott & Green, 2013; Williams et al., 2007). The results of the current study are even more striking when the large effect sizes and group sizes (positive schizotype *N* = 11; negative schizotype *N* = 75) are taken into consideration.

There are a number of possible explanations for these unexpected findings. One possibility for the disparity in these results is the use of different measures of schizotypy. The current study utilizes the CPPS, whereas previous studies by Abbott and Green (2013), and Williams and colleagues (2007) both utilized the SPQ (Raine, 1991). Although both measure three roughly analogous domains of schizotypy (i.e., social anhedonia, perceptual aberration, and magical ideation in the former versus interpersonal, cognitive-perceptual, and disorganized in the latter) it is possible that the differences in emotion recognition among specific symptom domains may be attributable to the use of different measures.

Another potential explanation for this disparity may be due to the design of the current study, which combined the perceptual aberration and magical ideation domains of the CPPS for analyses comparing positive and negative schizotypes. This methodology departs from the previous research, which has utilized three schizotypy domains for analyses. Although the prior research examining emotion recognition within a psychometric schizotypy has identified these deficits to be most profound among interpersonal and negative symptom domains, recent research utilizing patient populations has demonstrated significant relationships between disorganized symptoms and emotion recognition (Fett & Maat, 2011), and disorganized symptoms and social cognition (Minor & Lysaker, 2014). As such, it is recommended that future research examine the relationship between emotion recognition ability and each of the three schizotypy domains in the CPPS individually.

Relatedly, a final explanation for these unexpected results may be attributable to the factor structure of the *SocAnh* scale, which was utilized to test the fifth hypothesis that negative schizotypes would demonstrate poorer performances than positive schizotypes on the ER-40. Prior factor analyses by Kwapil and colleagues (2008) have revealed that counter to the conceptual standpoint in which social anhedonia is viewed as a facet of negative schizotypy, the *SocAnh* scale does not load exclusively on the negative schizotypy factor. The authors suggest that this finding may be reflective of a measurement issue with the scale that may be ameliorated with a purer measure of the social anhedonia construct. As such, future replications that include other measures of schizotypy are needed before definitive conclusions can be drawn about the relationship between emotion recognition and specific symptom domains.

Family History and Emotion Recognition

Results of the present study supported the hypothesis that participants with a family history of schizophrenia would demonstrate greater deficits in emotion recognition when compared to participants without a family history of the disorder. Specifically, participants with a family history demonstrated significant deficits in their overall ability to identify negative emotions and expressions of happiness. Additional small effect sizes were also detected for the overall identification of all emotions, as well as fearful, sad, and neutral expressions. A small effect size was also found for the identification of emotions displayed on female faces.

Significant results were also found in a separate analysis examining the identification of emotions of both moderate and extreme intensities. Results of this analysis indicate that participants with a family history of schizophrenia are significantly less accurate when identifying moderate displays of happiness, overall extreme emotional displays, extreme negative emotion displays, and extreme displays of sadness. Small effect sizes were detected for

the majority of the remaining non-significant emotional displays, including overall moderate displays of emotion, moderate displays of negative emotion, moderate displays of sadness, and extreme displays of anger and happiness.

Although the sample size was not sufficient to conduct additional analyses to further examine the relationship between emotion recognition and degree of relation, the results of the present study are consistent with prior research conducted that has demonstrated emotion recognition deficits among unaffected first-degree relatives of individuals with schizophrenia (Kee et al., 2004; Leppänen et al., 2008). As such, future research may seek to examine these factors within a larger sample, while also verifying family mental health status through the use of diagnostic interviews with those family members.

Strengths and implications of the current study

As previously mentioned, little remains known about the genetic transmission of schizophrenia, as the disorder does not appear to abide by simple Mendelian principles. As a result, research over the past 30 years has focused on the identification of endophenotypes as a way to gather more information about the genetic components of schizophrenia (McGue & Gottesman, 1989). As such, one of the strengths of the current study is its contribution to the growing research on social cognitive and endophenotypes of schizophrenia. Similarly, the current study also contributes to the more recent examination of emotion recognition capacities among those psychometrically identified as psychosis prone, and adds to the growing body of evidence for emotion recognition deficits within this population. Results of the present study also offer preliminary support for viewing emotion recognition deficits as an endophenotype of schizophrenia, as participants identified as both psychometrically and genetically prone to the disorder demonstrated statistically significantly deficits in their ability to correctly identify a

number of emotional expressions. An additional strength of the current study it its utilization of participants at psychometric and genetic risk for schizophrenia spectrum disorders. It does not appear than any study to date has examined these risk categories concurrently.

Finally, perhaps the greatest implication for the current study is for the treatment of schizophrenia. Research has pointed to the fact that the earlier the interventions for schizophrenia result in better prognoses and functional outcome. The current findings provide some provisional evidence for an emotion recognition endophenotype of schizophrenia, and with replication, these findings may assist with the diagnostic process and point to the use of specific treatment interventions aimed at increasing social cognition in individuals in the early stages of their psychotic illness.

Limitations of the current study

There are a number of limitations to the current study that should be mentioned. These limitations are primarily associated with the participant sample. Participants for the study were sampled from a rural Midwestern university. Women participants were overrepresented in the study's sample. The low number of male participants is problematic given that the fact that schizophrenia tends to be more prevalent in men compared to women, and generally is a more severe variant of schizophrenia. The age range (18 to 25 years) utilized for study is also another limitation. Although the age requirement was selected because it allows for assessment during the period of greatest risk for the onset of schizophrenia throughout the course of the 10-year study, it is also a limitation because psychosis is more likely to develop in men during the timeframe of the current study, but the onset is later for women, and the majority of the sample is women (APA, 2013). The high number of negative schizotypes (N = 78) relative to the low number of positive schizotypes (N = 13) is also a limitation of the present study. This unbalance

is especially problematic given the presence of hypotheses specific to schizotypy domains. In addition to the inadequate sample of male participants, the rural Midwestern setting for the current study may also present as another limitation, as the results of the study may not be generalizable to other geographic populations. Finally, one last limitation of the current study is its reliance on the self-report of a family history of schizophrenia. It is possible that these reports may be inaccurate or that family history of schizophrenia is under-reported given the stigma against mental illness.

Future directions

Although the current study offers preliminary evidence for an emotion recognition endophenotype of schizophrenia, future replications of the present methods are still needed before a definitive conclusion can be reached. Specifically, future research may seek to improve upon some of the weaknesses from the current study's sample, in which Caucasian, women, and negative schizotype participants were overrepresented. Similarly, it would also be beneficial to replicate the present methods in a larger sample, as the smaller sample size from the present study may not have been sufficient. For example, the low sample size of participants possessing a family history of schizophrenia in the current study precluded subsequent statistical analyses. Expanding upon the sample size, and number of male, minority, and positive schizotype participants in future studies would assist in adding to the generalizability of the research findings on psychometric schizotypes and emotion recognition. For example, it is possible that more equitable numbers of positive and negative psychometric schizotypes may result in different findings than the present study, both when comparing schizotypes to matched controls, as well as analyses comparing within group differences among psychometric schizotypes.

Future replications would also enable the establishment of more consistent trends for emotion recognition in a psychometric schizotype population. For example, the present study found positive schizotypes to perform worse on the emotion recognition task, whereas other research has demonstrated these deficits as being more profound among negative and interpersonal symptom domains (Abbott & Green, 2013; Williams et al., 2007).

As previously noted, the current study is subsumed under a larger ten-year longitudinal study examining several markers of schizophrenia spectrum disorders. The collection of follow-up data at 2, 5, 7, and 10 years after the present study affords the unique opportunity to further clarify the relationship between psychometric schizotypy, emotion recognition, and future onset of psychotic illness. Subsequent longitudinal analyses will also allow for the examination of the relationship between emotion recognition and the other proposed endophenotype markers from the parent study.

Future research may also specifically seek to further examine the unique relationship observed between neurocognitive tests of working memory (i.e., Arithmetic and Working Memory scores) and emotion recognition in the current study. As previously noted, initial analyses did not reveal significant emotion recognition deficits for psychometric schizotypes, but these differences became significant during subsequent GLM analyses that included either the WMI or AR along with schizotypy group membership (PS or MC). These results therefore demonstrate the presence of a more complex relationship between neurocognition, schizotypy, and emotion recognition, where neurocognitive performance and schizotypy group membership together both significantly contribute to the prediction of emotion recognition. Given these findings, it is suggested that future research also focus on further parsing out the relationship between these factors both statistically and theoretically. For example, it is possible that

cognitive flexibility, the ability to plan, and self-monitoring abilities necessary for working memory processes are significant driving forces in the recognition of the emotions of others.

Summary

Previous research findings have offered evidence for emotion recognition deficits within schizophrenia patients, first-degree relatives, and psychometrically identified schizotypes (Abbott & Green, 2013; Kee et al., 2004; Kohler et al., 2003; Leppänen et al., 2008; Williams et al., 2007). The present study appears to be one of the first to concurrently examine emotion recognition among individuals at psychometric and genetic risk for the disorder. The results of this study offer confirmatory evidence for the presence of emotion recognition deficits among psychometric schizotype participants as well as participants with a family history of schizophrenia. Additionally, the current study also revealed a more complex relationship between neurocognitive performance and emotion recognition abilities, a relationship that had been previously unexamined in prior research utilizing a psychometric schizotype population. Although preliminary analyses yielded no significant between group differences among PS and MC participants, subsequent analyses produced significant results after accounting for the variance associated with WMI and AR performance. These findings highlight the importance of future replication to further disentangle the relationship between neurocognitive performance, emotion recognition, and schizotypy both statistically and theoretically.

One of the most robust results from the present study demonstrated within group differences in the accuracy of emotion recognition among psychometric schizotypes, with positive schizotypes performing significantly worse than their negative schizotype counterparts. These results ran counter to not only the hypothesized direction, but also the previous findings by Abbott and Green (2013) and Williams and colleagues (2007), who found decreased emotion

recognition accuracy to be specific to the negative and interpersonal aspects of schizotypy, respectively. However, these findings do appear to be consistent with recent research by Fett and Maat (2011) and Minor and Lysaker (2014), which has demonstrated significant relationships between disorganized symptoms and emotion recognition, and disorganized symptoms and social cognition, respectively. This unanticipated finding underscores the importance of future replication to ascertain more a more definitive understanding of the relationship between emotion recognition deficits and specific symptom domains. As such, it is recommended that future examine the relationship between each of the three schizotypy domains to further clarify this relationship.

Results of the present study contribute to the growing body of research literature on emotion recognition abilities of psychometric schizotypes and individuals with a family history of schizophrenia. Although the study offers some preliminary evidence of an emotion recognition endophenotype of schizophrenia spectrum disorders, more research is needed before a definitive conclusion can be drawn. Given the genetic complexity of schizophrenia, the identification of endophenotypes of the disorder may offer simpler clues for the detection of symptoms and contribute to an overall greater understanding of schizophrenia.

REFERENCES

Abbott, G. R., & Green, M. J. (2013). Facial affect recognition and schizotypal personality characteristics. *Early Intervention in Psychiatry, 7,* 58-63.

Abi-Dargham, A., Martinez, D., Mawlawi, O., Simpson, N., Hwang, D. R., Slifstein, M.,…Laruelle, M. (2000). Measurement of striatal and extrastriatal dopamine D1 receptor binding potential with [11C]NNC 112 in humans: Validation and reproducibility. *Journal of Cerebral Blood Flow & Metabolism,* 20, 225-243.

Addington, J., Penn, D., Woods, S. W., Addington, D., Perkins, D.O. (2008). Facial affect recognition in individuals at clinical high risk for psychosis. *British Journal of Psychiatry, 192,* 67-68.

American Psychiatric Association (APA). *Diagnostic and Statistical Manual of Mental Disorders, 3rd Edition (DSM-III)*. Washington, D.C.: APA, 1980.

American Psychiatric Association (APA). *Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text-Revision (DSM-IV-TR)*. Washington, D.C.: APA, 2000.

American Psychiatric Association (APA). *Diagnostic and Statistical Manual of Mental Disorders,* 5th *Edition (DSM-5)*. Washington, D.C.: APA, 2013.

Andreasen, N. C. (1997). The evolving concept of schizophrenia: From Kraepelin to the present and future. *Schizophrenia Research, 28,* 105-109.

Andreasen, N. C. & Carpenter, W. T. (1993). Diagnosis and classification of schizophrenia. *Schizophrenia Bulletin, 19,* 199-214.

Bleuler, E. (1911/1950). *Dementia Praecox or the Group of Schizophrenias* (J. Zinkin, Trans.). New York: International Universities Press.

Bolinskey, P. K., & Gottesman, I. I. (2010). Premorbid personality indicators of schizophrenia-related psychosis in a hypothetically psychosis-prone college sample. *Scandinavian Journal of Psychology, 51,* 68-74. doi: 10.1111/j.1467-9450.2009.00730.x

Braff, D. L., Freedman, R. F., Schork, N. J., & Gottesman, I. I. (2007). Deconstructing schizophrenia: An overview of the use of endophenotypes in order to understand a complex disorder. *Schizophrenia Bulletin, 33,* 21-32. doi: 10.1093/schbul/sbl049

Braff, D. Schork, N. J. & Gottesman, I. I. (2007). Endophenotyping schizophrenia. *American Journal of Psychiatry, 164,* 705-707.

Butcher, J. N., Dahlstrom, W. G., Graham, J. R., Tellegan, A. & Kaemmer, B. (1989). *MMPI-2*: *Manual for Administration and Scoring.* Minneapolis: University of Minnesota.

Carlsson, A. & Lindqvist, M. (1963). Effect of chlorpromazine or haloperidol on formation of 3methoxytyramine and normetanephrine in mouse brain. *Acta Pharmacologica et Toxicologica, 20,* 140-144.

Chapman, L. J., & Chapman, J. P. (1985). Psychosis proneness. In M. Alpert (Ed.), *Controversies in schizophrenia: Changes and constancies: Proceedings of the 74th Annual Meeting of the American Psychopathological Association, New York City, March 1-3, 1984.* (pp. 157-172). New York: Guilford Press.

Chapman, L. J., Chapman, J. P., & Raulin, M. L. (1976). Scales for physical and social anhedonia. *Journal of Abnormal Psychology, 85* (4), 374-382. doi: 10.1037/0021-843x.85.4.374

Chapman, L. J., Chapman, J. P., & Raulin, M. L. (1978). Body-image aberration in schizophrenia. *Journal of Abnormal Psychology, 87* (4), 399-407. doi: 10.1037/0021-843x.87.4.399

Chapman, L. J., Chapman, J. P., Kwapil, T. R., Eckblad, M., & Zinser, M. C. (1994). Putatively psychosis-prone subjects 10 years later. *Journal of Abnormal Psychology, 103* (2), 171-183. doi: 10.1037/0021-843x.103.2.171

Colibazzi, T., Wexler, B. E., Bansal, R., Hao, X., Liu, J., Sanchez-Peña, J…. Peterson, B. S. (2013). Anatomical abnormalities in gray and white matter of the cortical surface in persons with schizophrenia. *PLOS One, 8,* 1-13.

Conklin, H. M., Curtis, C. E., Katsanis, J., & Iacono, W. G. (2000). Verbal working memory impairment in schizophrenia patients and their first-degree relatives: Evidence from the Digit Span Task. *The American Journal of Psychiatry, 157,* 275-277. doi: 10.1176/appi.ajp.157.2.275

Couture, S. M., Penn, D. L., & Roberts, D. L. (2006). The functional significance of social cognition in schizophrenia: A review. *Schizophrenia Bulletin,32,* S44-S63. doi: 10.1093/schbul/sbl029

Creese, I. Burt, D. R. & Snyder, S. H. (1976). Dopamine receptor binding predicts clinical and pharmacological potencies of antischizophrenic drug. *Science, 192,* 481-483.

Crow, T. J. (1980). Molecular pathology of schizophrenia: More than one disease process? *British Medical Journal, 280,* 66-68.

Crow, T. J. (1985). The two-syndrome concept: Origins and current status. *Schizophrenia Bulletin, 11,* 471-486.

Davis, K. L., Kahn, R. S., Ko, G., & Davidson, M. (1991). Dopamine in schizophrenia: A review. *Schizophrenia Bulletin, 1976,* 1474-1486.

Eack, S. M., Mermon, D. E., Montrose, D. M., Miewald, J., Gur, R. E., Gur, R. C., … Keshavan, M. S. (2010). Social cognition deficits among individuals at familial high risk for schizophrenia. *Schizophrenia Bulletin, 36,* 1081-1088. doi: 10.1093/schbul/sbp026

Eckblad, M., & Chapman, L. J. (1983). Magical ideation as an indicator of schizotypy. *Journal of Consulting and Clinical Psychology, 51* (2), 215-225. doi: 10.1037/0022-006x.51.2.215

Eckblad, M. B., Chapman, L. J, Chapman, J. P. & Mishlove, M. (1982). *The Revised Social Anhedonia Scale: Unpublished test.* University of Wisconsin at Madison. Madison, WI.

Ekman, P. & Friesen, W. V. (1975). *Unmasking the face*. Englewood Cliffs, New Jersey: Prentice-Hall.

Edwards, J., Jackson, H. J., & Pattison, P. E. (2002). Emotion recognition via facial expression and affective prosody in schizophrenia: A methodological review. *Clinical Psychology Review, 22,* 789-832.

Fett., A-K., J., & Maat, A. (2011). Social cognitive impariments and psychotic symptoms: What is the nature of their association? *Schizophrenia Bulletin, 39(1),* 77-85.

Forbes, N. F., Carrick, L. A., McIntosh, A. M., & Lawrie, S. M. (2009). Working memory in schizophrenia: A meta-analysis. *Psychological Medicine, 39*, 889-905. doi: 10.1017/s0033291708004558

Fox, H. A. (1978). Bleuler, Schneider and schizophrenia. *Journal of Clinical Psychiatry, 39*, 703-708.

Gard, D. E., Gard, M. G., Kring, A. M., & John, O. P. (2006). Anticipatory and consummatory components of the experience of pleasure: A scale development study. *Journal of Research in Personality, 40*, 1086-11012.

Gottesman, I. I. (1991). *Schizophrenia genesis: The origins of madness*. New York: W. H. Freeman.

Gottesman, I. I., & Gould, T. D. (2003). The endophenotype concept in psychiatry: Etymology and strategic intentions. *American Journal of Psychiatry, 160,* 636-645. doi: 10.1176/appi.ajp.160.4.636

Gur, R. E., Turetsky, B. I., Cowell, P. E., Finkelman, C., Maany, V., Grossman, R. I.,…Gur, R. C. (2000). Temporolimbic volume reductions in schizophrenia. *Archives of General Psychiatry, 57*, 769-775. doi: 10.1001/archpsyc.57.8.769

Harrison, P. J. (1999). The neuropathology of schizophrenia: A critical review of the data and their interpretation. *Brain: A Journal of Neurology, 122,* 593-624.

Howes, O. D. & Kapur, S. (2009). The dopamine hypothesis of schizophrenia: Version III - the final common pathway. *Schizophrenia Bulletin, 35,* 549-562.

Hunter, H. K. (2014). *Working memory and executive functioning deficits as possible endophenotypes in a psychometrically identified schizotypy population* (Unpublished doctoral dissertation). Indiana State University, Terre Haute, IN.

Hunter, H. K., Bolinskey, P. K., Novi, J. H., Hudak, D. V, James, A. V., Myers, K. R., & Schuder, K. M. (2014). Using the MMPI-2-RF to discriminate psychometrically-identified schizotypic college students from a matched comparison sample. *Journal of Personality Assessment, 96(6),* 596-603. doi: 10.1080/00223891.2014.922093

Hyler, S. E. (1994). *Personality Diagnostic Questionnaire – 4*. New York: New York State Psychiatric Institute.

Johnstone, E. C., Crow, T. J., Frith, C. D., Husband, J., & Kreel, L. (1976). Cerebral ventricle size and cognitive impairment in chronic schizophrenia. *Lancet, 2,* 924-926.

Kee, K. S., Horan, W. P., Mintz, J., & Green, M. F. (2004). Do the siblings of schizophrenia patients demonstrate affect perception deficits? *Schizophrenia Research, 67,* 87-94.

Keller, W. R., Fischer, B. A., & Carpenter, W. T. (2011). Revisiting the diagnosis of schizophrenia: Where have we been and where are we going? *CNS Neuroscience & Therapeutics, 17,* 83-88.

Kohler, C. G., Turner, T. H., Bilker, W. B., Brensinger, C. M., Siegel, S. J., Kanes, S. J., …Gur, R. C. (2003). Facial emotion recognition in schizophrenia: Intensity effects and error pattern. *American Journal of Psychiatry, 160,* 1768-1774. doi: 10.1176/appi.ajp.160.10.1768

Kohler, C. G., Turner, T. H., Gur, R. E., & Gur, R. C. (2004). Recognition of facial emotions in neuropsychiatric disorders. *CNS Spectrums, 9,* 267-274.

Kraepelin, E. (1971). *Dementia praecox and paraphrenia.* (R. M. Barclay, Trans.). In G. M. Robertson (Ed.). Huttington, NY: Krieger.

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

Kwapil, T. R., Barrantes-Vidal, N., & Silvia, P. J. (2008). The dimensional structure of the Wisconsin Schizotypy Scales: Factor identification and construct validity. *Schizophrenia Bulletin, 34* (3), 444-457. doi: 10.1093/schbul/sbm098

Lawrie, S. M. and Abukmeil, S. S. (1998). Brain abnormality in schizophrenia: A systematic and quantitative review of volumetric magnetic resonance imaging studies. *British Journal of Psychiatry, 172,* 110-120.

Lenzenweger, M. F. (2006). Schizotypy: An organizing framework for schizophrenia research. *Current Directions in Psychological Science, 15*, 162-166. doi: 10.1111/j.1467-8721.2006.00428.x

Lenzenweger, M. F. (2010). *Schizotypy and schizophrenia: The view from experimental psychopathology*. New York: Guilford.

Lenzenweger, M. F. (2013). Thinking clearly about the endophenotype-intermediate phenotype-biomarker distinctions in developmental psychopathology research. *Development and Psychopathology, 25*, 1347-1357.

Lenzenweger, M. F., & Dworkin, R. H. (1996). The dimensions of schizophrenia phenomenology: Not one or two, at least three, perhaps four. *The British Journal of Psychiatry, 168*, 432-440. doi: 10.1192/bjp.168.4.432

Lenzenweger, M. F., & Korfine, L. (1992). Confirming the latent structure and base rate of schizotypy: A taxometric analysis. *Journal of Abnormal Psychology, 101,* 567-571. doi: 10.1037/0021-843x.101.3.567

Leppänen, J. M., Niehaus, D. J. H., Koen, L., Du Toit, E., Schoeman, R., & Emsley, R. (2008). Deficits in facial affect recognition in unaffected siblings of Xhosa schizophrenia patients: Evidence for a neurocognitive endophenotype. *Schizophrenia Research, 99,* 270-273.

Mäkinen, J., Miettunen, J., Isohanni, M., & Koponen, H. (2008). Negative symptoms in schizophrenia – a review. *Nordic Journal of Psychiatry, 62,* 334-341. doi: 10.1080/08039480801959307

Matheson, S., & Langdon, R. (2008). Schizotypal traits impact upon executive working memory and aspects of IQ. *Psychiatry Research, 159,* 207-214. doi: 10.1016/j.psychres.2007.04.006

McGue, M., & Gottesman, I. I. (1989). A single dominant gene still cannot account for the transmission of schizophrenia. *Archives of General Psychiatry, 46,* 478-480.

Meehl, P. E. (1962). Schizotaxia, schizotypy, schizophrenia. *American Psychologist, 17,* 827-828.

Meltzer, H. Y. (1985). Dopamine and negative symptoms in schizophrenia: Critique of the Type I–II hypothesis. In M. Alpert (Ed.), *Controversies in schizophrenia: Changes and constancies: Proceedings of the 74th Annual Meeting of the American Psychopathological Association, New York City, March 1-3, 1984.* (pp. 110-136). New York, NY US: Guilford Press.

Minor, K. S., & Lysaker, P. H. (2014). Necessary, but not sufficient: Links between neurocognition, social cognition, and metacognition in schizophrenia are moderated by disorganized symptoms. *Schizophrenia Research, 159,* 198-204.

Minzenberg, M. J., Yoon, J. H., & Carter, C. S. (2011). Schizophrenia. In Hales, R. E., Yudofsky, S. C., & Gabbard, G. O. (Eds.). *Essentials of Psychiatry* (111-150). Washington, D.C.: American Psychiatric Publishing.

Moncrieff, J. (2009) A critique of the dopamine hypothesis of schizophrenia and psychosis. *Harvard Review of Psychiatry, 17,* 214-225.

Oldfield, R. C. (1971). The assessment and analysis of handedness: the Edinburgh inventory. *Neuropscyhologia, 9*, 97-113.

Owens, D. G. C., and Johnstone, E. C. (1980). The disabilities of chronic schizophrenia: Their nature and the factors contributing to their development. *British Journal of Psychiatry, 136*, 384-395.

Penn, D. L., Sanna, L. J., & Roberts, D. L. (2008). Social cognition in schizophrenia: An overview. *Schizophrenia Bulletin, 34,* 408-411.

Rado, S. (1953). Dynamics and classification of disordered behavior. *The American Journal of Psychiatry, 110*, 406-426

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

Sergi, M. J., Rassovsky, Y., Nuechterlein, K. H., & Green, M.F. (2006). Social perception as a mediator of influence of early visual processing on functional status in schizophrenia. *American Journal of Psychiatry, 163,* 448-454.

Sergi, M. J., Rassovsky, Y., Widmark, C., Reist, C., Erhart, S., Braff, D. L., …Green, M. F. (2007). Social cognition in schizophrenia: Relationships with neurocognition and negative symptoms. *Schizophrenia Research, 90,* 316-324. doi: 10.1016/j.schres.2006.09.028

Saddichha, S., Kumar, R., Sur, S., & Sinha, B. N. P. (2010). First rank symptoms: Concepts and diagnostic utility. *African Journal of Psychiatry, 13*, 263-266. doi: 10.4314/ajpsy.v13i4.61874

Snitz, B. E., MacDonald, A. W., III, & Carter, C. S. (2006). Cognitive deficits in unaffected first-degree relatives of schizophrenia patients: A meta-analytic review of putative endophenotypes. *Schizophrenia Bulletin, 32,* 179-194. doi: 10.1093/schbul/sbi048

Spitzer, R. L., Endicott, J., Robins, E. (1978). Research diagnostic criteria: Rationale and reliability. *Arch Gen Psychiatry, 35,* 774-782.

Tallent, K. A., & Gooding, D. C. (1999). Working memory and Wisconsin Card Sorting Test performance in schizotypic individuals: A replication and extension. *Psychiatry Research, 89,* 161-170. doi: 10.1016/s0165-1781(99)00101-8

Vita, A., De Peri, L., Silenzi, C., & Dieci, M. (2006). Brain morphology in first-episode schizophrenia: a meta-analysis of quantitative magnetic resonance imaging studies. *Schizophrenia Research, 82(1),* 75-88.

Wechsler, D. (2008a). *Wechsler Adult Intelligence Scale - Fourth Edition*. San Antonio, TX: Pearson Assessment.

Wechsler, D. (2008b). *Wechsler Adult Intelligence Scale - Fourth Edition: Technical and interpretive manual*. San Antonio, TX: Pearson Assessment.

Wechsler, D. (2009a). *Wechsler Memory Scale - Fourth Edition.* San Antonio, TX: Pearson Assessment.

Wechsler, D. (2009b). *Wechsler Memory Scale - Fourth Edition*: *Technical and interpretive manual*. San Antonio, TX: Pearson Assessment.

Williams, B. T., Henry, J. D., & Green, M. J. (2007). Facial affect recognition and schizotypy. *Early Intervention in Psychiatry, 1,* 177-182. doi: 10.1111/j.1751-7893.2007.00023.x

Woods, B. T., Yurgelun-Tood, D., Benens, F. M., Frankerburg, F. R. Pope, H. C., McSparren, J. (1990). Progressive ventricular enlargement in schizophrenia: Comparison to bipolar affective disorder and correlation to clinical course. *Biol. Psychiatry, 27,* 341-352.

World Health Organization (2001). *Mental health report 2001. Mental Health: New Understanding, New Hope.* World Health Organization, Geneva.

APPENDIX A: SCHNEIDERIAN FIRST RANK SYMPTOMS

1. Auditory hallucinations:
    a. Hearing thoughts spoken aloud
    b. Hearing voices referring to himself / herself, made in the third person
    c. Auditory hallucinations in the form of a commentary
2. Thought withdrawal, insertion and interruption
3. Thought broadcasting
4. Somatic hallucinations
5. Delusional perception
6. Feelings or actions experienced as made or influenced by external agents

APPENDIX B: DSM-5 CRITERIA FOR SCHIZOPHRENIA

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

   1. Delusions
   2. Hallucinations
   3. Disorganized speech
   4. Grossly abnormal psychomotor behavior, including catatonia
   5. Negative symptoms, e.g., diminished emotional expression or avolition

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

C. Continuous signs of the disturbance persist for at least 6 months. This 6-month period must include at least 1 month of symptoms (or less if successfully treated) that meet Criterion A (i.e., active-phase symptoms) and may include periods of prodromal or residual symptoms. During these prodromal or residual periods, the signs of the disturbance may be manifested by only negative symptoms or by an attenuated form

of two or more symptoms listed in Criterion A (e.g., beliefs perceived as odd, perceptual experiences described as out of the ordinary).

D. Schizoaffective Disorder and Depressive or Bipolar Disorder With Psychotic Features have been ruled out because either (1) no Major Depressive, Manic, or Mixed Episodes have occurred concurrently with the active-phase symptoms; or (2) if mood episodes have occurred during active-phase symptoms, their total duration has been less than half of the total duration of the active periods.

E. The disturbance is not due to the direct physiological effects of a substance (e.g., an abused drug, a medication) or a general medical condition.

F. If there is a history of Autistic Disorder or another Pervasive Developmental Disorder or other communication disorder of childhood onset, the additional diagnosis of Schizophrenia is made only if prominent delusions or hallucinations are also present for at least 1 month (or less if successfully treated).

APPENDIX C: DSM-IV CRITERIA FOR SCHIZOPHRENIA

A. Characteristic symptoms: Two (or more) of the following, each present for a significant portion of time during a 1-month period (or less if successfully treated):

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

Note: Only one Criterion A symptom is required if delusions are bizarre or hallucinations consist of a voice keeping up a running commentary on the person's behavior or thoughts, or two or more voices conversing with each other.

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

C. Duration: Continuous signs of the disturbance persist for at least 6 months. This 6-month period must include at least 1 month of symptoms (or less if successfully treated) that meet Criterion A (i.e., active-phase symptoms) and may include periods of prodromal or residual symptoms. During these prodromal or residual periods, the signs of the

disturbance may be manifested by only negative symptoms or two or more symptoms listed in Criterion A present in an attenuated form (e.g., odd beliefs, unusual perceptual experiences).

D. Schizoaffective and Mood Disorder exclusion: Schizoaffective Disorder and Mood Disorder With Psychotic Features have been ruled out because either (1) no Major Depressive, Manic, or Mixed Episodes have occurred concurrently with the active phase symptoms; or (2) if mood episodes have occurred during active-phase symptoms, their total duration has been brief relative to the duration of the active and residual periods.

E. Substance/general medical condition exclusion: The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition.

F. Relationship to a Pervasive Developmental Disorder: If there is a history of Autistic Disorder or another Pervasive Developmental Disorder, the additional diagnosis of Schizophrenia is made only if prominent delusions or hallucinations are also present for at least a month (or less if successfully treated).

Schizophrenia Subtypes

The subtypes of Schizophrenia are defined by the predominant symptomatology at the time of evaluation.

295.30 Paranoid Type

A type of Schizophrenia in which the following criteria are met:

A. Preoccupation with one or more delusions or frequent auditory hallucinations.

B. None of the following is prominent: disorganized speech, disorganized or catatonic behavior, or flat or inappropriate affect.

295.10 Disorganized Type

A type of Schizophrenia in which the following criteria are met:

A. All of the following are prominent:
    1. Disorganized speech
    2. Disorganized behavior
    3. Flat or inappropriate affect
B. The criteria are not met for Catatonic Type.

295.20 Catatonic Type

A type of Schizophrenia in which the clinical picture is dominated by at least two of the following:

1. Motoric immobility as evidenced by catalepsy (including waxy flexibility) or stupor
2. Excessive motor activity (that is apparently purposeless and not influenced by external stimuli)
3. Extreme negativism (an apparently motiveless resistance to all instructions or maintenance of a rigid posture against attempts to be moved) or mutism
4. Peculiarities of voluntary movement as evidenced by posturing (voluntary assumption of inappropriate or bizarre postures), stereotyped movements, prominent mannerisms, or prominent grimacing
5. Echolalia or echopraxia

295.90 Undifferentiated Type

A type of Schizophrenia in which symptoms that meet Criterion A are present, but the criteria are not met for the Paranoid, Disorganized, or Catatonic Type.

295.60 Residual Type

A type of Schizophrenia in which the following criteria are met:

A. Absence of prominent delusions, hallucinations, disorganized speech, and grossly disorganized or catatonic behavior.
B. There is continuing evidence of the disturbance, as indicated by the presence of negative symptoms or two or more symptoms listed in Criterion A for Schizophrenia, present in an attenuated form (e.g., odd beliefs, unusual perceptual experiences).

Classification of Longitudinal Course for Schizophrenia

These specifiers can be applied only after at least 1 year has elapsed since the initial onset of active-phase symptoms:

Episodic With Interepisode Residual Symptoms: This specifier applies when the course is characterized by episodes in which Criterion A for Schizophrenia is met and there are clinically significant residual symptoms between the episodes. With Prominent Negative Symptoms can be added if prominent negative symptoms are present during these residual periods.

Episodic With No Interepisode Residual Symptoms: This specifier applies when the course is characterized by episodes in which Criterion A for Schizophrenia is met and there are no clinically significant residual symptoms between the episodes.

Continuous: This specifier applies when characteristic symptoms of Criterion A are met throughout all (or most) of the course. With Prominent Negative Symptoms can be added if prominent negative symptoms are also present.

Single Episode In Partial Remission: This specifier applies when there has been a single episode in which Criterion A for Schizophrenia is met and some clinically significant residual symptoms remain. With Prominent Negative Symptoms can be added if these residual symptoms include prominent negative symptoms.

Single Episode In Full Remission: This specifier applies when there has been a single episode in which Criterion A for Schizophrenia has been met and no clinically significant residual symptoms remain.

Other or Unspecified Pattern: This specifier is used if another or an unspecified course pattern has been present.