

6-2017

Comparison of Implicit Thought and Learning in Individuals with Schizophrenia

Camilla Seippel

Antioch University Santa Barbara

Follow this and additional works at: <https://aura.antioch.edu/etds>



Part of the [Clinical Psychology Commons](#), and the [Cognitive Psychology Commons](#)

Recommended Citation

Seippel, Camilla, "Comparison of Implicit Thought and Learning in Individuals with Schizophrenia" (2017). *Dissertations & Theses*. 408.

<https://aura.antioch.edu/etds/408>

This Dissertation is brought to you for free and open access by the Student & Alumni Scholarship, including Dissertations & Theses at AURA - Antioch University Repository and Archive. It has been accepted for inclusion in Dissertations & Theses by an authorized administrator of AURA - Antioch University Repository and Archive. For more information, please contact dpenrose@antioch.edu, wmcgrath@antioch.edu.

**COMPARISON OF IMPLICIT THOUGHT AND LEARNING IN INDIVIDUALS
WITH SCHIZOPHRENIA**

A dissertation presented to the faculty of

ANTIOCH UNIVERSITY SANTA BARBARA

in partial fulfillment of
the requirements for the
degree of

DOCTOR OF PSYCHOLOGY
in
CLINICAL PSYCHOLOGY

By

CAMILLA SEIPPEL
JUNE 2017

COMPARISON OF IMPLICIT THOUGHT AND LEARNING IN INDIVIDUALS
WITH SCHIZOPHRENIA

This dissertation, by Camilla Seippel, has been approved by the committee members signed below who recommend that it be accepted by the faculty of Antioch University Santa Barbara in partial fulfillment of requirements for the degree of

DOCTOR OF PSYCHOLOGY

Dissertation Committee:

Henry V. Soper, PhD
Chairperson

Brett Kia-Keating, Ed.D.
Second Faculty

Bernice A. Marcopulos, PhD, ABPP
External Expert

© Camilla Seippel, 2017

Abstract

This investigation studied implicit learning differences in individuals with schizophrenia. Three implicit learning strategies were examined: priming, procedural, and incidental learning. Twenty-six participants with schizophrenia were recruited from various outpatient clinics and programs in Orange, CA to participate in this study. Participants were administered a psychological battery composed of tests to measure individual differences in implicit learning abilities within the group. Differences in crystallized and fluid knowledge abilities within the different implicit learning conditions were tested. Demographic information was also collected and where possible included for the purpose of accounting for demographic variations amongst participants. Demographic variables included the participant's age, gender, years of education, and ethnicity. Following data collection, raw scores were converted to T-scores (and normed when possible per demographic variations) and run through SPSS. The first analysis conducted was a path analysis to estimate the causal relationships amongst the variables. Following this initial analysis, six separate *t* tests were run through SPSS. Differences between individual learning conditions were identified, and three of the four hypotheses were found to be significant. The electronic version of this dissertation is accessible at the Ohiolink ETD center <http://www.ohiolink.edu/etd>

Acknowledgements

There are so many that I wish to thank and who's presence in life at some time have influenced my journey that led me to and through graduate school. There are so many of you who have shown me support in my life, some who are still with me on my journey, others who are not, and others that distance and time, has grown us apart. But in my heart and in my thoughts, I still carry you.

For the unconditional love and support throughout my career and life endeavors I express my deepest gratitude and appreciation for my father, mother, and both of my siblings. Without the love, patience, and understanding from my family I would not have reached my goals and experienced the successes I have.

To my mentors and advisors throughout my education and studies, and to other professionals in the field I have met along the way, your kindness and willingness to share your knowledge, experience, and guidance is nonpareil.

To my friends, thank you for being there in the roughest and most trying of times, your friendship and strength means everything.

Table of Contents

Abstract.....	iv
Acknowledgements.....	v
Table of Contents.....	vi
List of Tables.....	ix
Chapter I: Introduction.....	1
Background and Rationale.....	1
DSM-IV to DSM-5.....	2
Schizophrenia Characteristics.....	3
Positive Symptoms.....	4
Delusions.....	4
Hallucinations.....	5
Formal thought disorder.....	6
Negative Symptoms.....	6
Neurocognitive Symptoms.....	7
Positive, Negative, and Cognitive Systems.....	9
Why is it Important to Understand Neuropsychological Abilities in Schizophrenia?.....	9
Neuropsychology, Thought, and Cognition, and Rehabilitation Outcomes.....	11
Gaps in Research and Purpose.....	12
Chapter II: Review of the Literature.....	16
Models of Schizophrenia Disorder Categorization/ Etiology of Schizophrenia.....	16
Neurotransmitter and chemical networks.....	16
The dopamine hypothesis.....	16
The dopamine cholinergic theory.....	17
The glutamate hypothesis.....	17
Genetic models of schizophrenia.....	18
Model of delusions and hallucinations.....	19
Model of memory of schizophrenia.....	19
Model of medical conditions and metabolic syndrome in schizophrenia.....	20
Schizophrenia as a Neurodevelopment versus Neurodegenerative Disorder.....	22
Temporal changes.....	23
Changes during the stage of symptom onset.....	24
Cognitive progression.....	25
Neuropsychology Findings in Schizophrenia: What does the Literature Tell us about Neuropsychology and Neurocognition in Schizophrenia?.....	26
Areas of Spared Functioning.....	30
Neuropsychological Normalcy and Schizophrenia.....	33
Deficit and Nondeficit Schizophrenia.....	35
Top-Down and Bottom-Up Cognitive Processing.....	36
Explicit Learning/Declarative and Implicit/Nondeclarative Learning.....	38

Neuropsychology of explicit/declarative and implicit/nondeclarative learning	39
Explicit learning/declarative and implicit/nondeclarative learning in schizophrenia	40
Implicit Learning Strategies	41
Priming in schizophrenia	42
Procedural learning in schizophrenia.....	43
Incidental learning in schizophrenia	45
Learning and Skill Acquisition	46
Cattell-Horn-Carroll Theory of Intelligence.....	49
Crystallized and Fluid Knowledge in Schizophrenia.....	49
Cognition and Learning in Daily Living Skills.....	50
Relationships between Neurocognition and Functional Status in Schizophrenia.....	51
Neuropsychological Testing and Schizophrenia: NIMH Initiative and MATRICS.....	54
Medication and Cognition in Schizophrenia.....	55
Summary.....	57
Gaps in the Research.....	59
Research Questions.....	60
Chapter III: Research Design and Methodology	62
Design	62
Research Variables.....	63
Participant Recruitment	64
Instrumentation and Measures	65
Cognitive domains: MATRICS Consensus Cognitive Battery.....	66
Psychopathology: Presence of positive and negative symptoms.....	67
Declarative learning and memory: Crystallized.....	70
Non-declarative crystallized learning and memory: Priming strategy	71
Non-declarative fluid learning and memory: Procedural strategy	73
Non-declarative crystallized learning and memory: Incidental strategy ..	74
Non-declarative fluid learning and memory: Incidental strategy	76
Procedures.....	77
Data Collection Methods	79
Data Processing Techniques	85
Methodological Assumptions and Limitations.....	87
Ethical Assurances.....	88
Chapter IV: Results.....	91
Study Sample Selection and Characteristics.....	91
Analysis Results.....	92
Chapter V: Discussion	96

Implications for Preserved Functioning.....	100
Future Implications Directions	101
References.....	104
Appendix A: Informed Consent (Participant).....	119
Appendix B: Informed Consent (Conservator).....	122
Appendix C: Informed Assent (Participant)	125
Appendix D: Data Collection Forms	128
Appendix E: Participant Recruitment Material/Script.....	132
Appendix F: Sociodemographic Questionnaire	134
Appendix G: Ethical Assurances	136

List of Tables

Table 1. Neuropsychological Test Battery and Variables	66
Table 2. Test Order Administration for Groups 1-4.	81
Table 3. Sample Demographic Characteristics, Schizophrenia Participants <i>N</i> =26	92
Table 4. Prime and No-Prime Learning in Crystallized Knowledge	92
Table 5. Incidental Learning in Crystallized and Fluid Knowledge.....	93
Table 6. Incidental and Procedural Learning in Fluid Knowledge.....	94
Table 7. Priming and Incidental Learning in Crystallized Knowledge	95

Chapter I: Introduction

Background and Rationale

The Diagnostic and Statistical Manual-5 (DSM-5) identified the prevalence of schizophrenia across the lifetime to be between 0.3-0.7 percent (American Psychiatric Association [APA], 2013, p. 100). Schizophrenia is conceptualized as a chronic and persistent neurocognitive disorder that presents with diverse symptomology and heterogeneous levels of severity and functioning (Palmer, Heiby, Fujii, & Kameoka, 2008). Nearly one third of adult patients who received a schizophrenia diagnosis also experienced a psychotic break at an earlier point in their life, most commonly before the age of 19 (Mayoral et al., 2008; Wozniak, Block, White, Jensen, & Schulz, 2008).

In their review of the literature, Rabe-Jablonska, Kotlicka-Antczak, and Gmitrowicz (2000) discussed the difficulties of early, initial, diagnosis schizophrenia because there has been no clear-cut differentiation of prodromal symptoms and a schizophrenia diagnosis. They also indicated that it has been unclear if these symptoms are an indication of predisposition to the disorder, or if individuals will go on to develop schizophrenia. Additional difficulties that arise regarding prodromal schizophrenia and symptoms have included the deficit of existing studies, which assessed for neuropsychological and neurobiological markers for the prodromal symptoms of the disorder (Grimm, Kersting, Zink, & Gass, 2010). The literature around first-episode psychosis has suggested a high percentage of relapse rates during the initial phase and years of the onset of symptoms. Additionally, Wiersma, Nienhuis, Slooff, and Giel (1998) determined from their study that suicide and chronicity were high risk factors following this first-episode. A 2005 study by Chen et al. showed that first-episode

schizophrenia was associated with increased risk for relapse in the three years that followed the episode.

Schizophrenia is a severe and persistent mental illness, and developing our understanding of the neurocognitive deficits and clinical presentation of the disorder is essential to establish protective factors, and compensatory techniques. In their study, Ucok, Polat, Cakir, and Genk (2006) aimed to identify factors that predict clinical and functional outcome in this population, after first-episode schizophrenia. Participants were assessed on various outcome measures, following one year of their initial psychotic episode, including, relapse, employment, and symptom severity. Ucok et al.'s (2006) findings indicated that two factors, treatment compliance and premorbid social adjustment, were correlated with relapse. From these findings, the authors concluded that research in schizophrenia should place greater emphasis on understanding the etiology of schizophrenia as well as on psychosocial interventions (Ucok et al., 2006).

DSM-IV to DSM-5

The current research trend into identification is to move away from an approach that diagnoses schizophrenia based on presenting symptomology, and toward an approach that incorporates biology and cognition for valid diagnosis (Cuthbert & Insel, 2010; Insel, 2010). Prior to the implementation of the DSM-5, in the DSM-IV, schizophrenia was classified into distinct subtypes. One of the primary changes applied to schizophrenia diagnostic classification in the DSM-5 was the elimination of this sub-classification system (Heckers et al., 2013). The subtypes were discarded, based on their low validity, instability of treatment response, and limited long-term diagnostic stability. Instead, an approach that records symptom type and severity on a continuum was

adopted, thereby accounting for the heterogeneity of schizophrenia, was adopted (APA, 2013).

Despite changes to how schizophrenia is diagnosed, there continues to exist a deficit in the validity and utility of diagnosis. One of the arguments put forth by Heckers et al. (2013) was that despite the modifications made to diagnosing schizophrenia in the DSM-5, specifically the inclusion of the dimensionality of the disorder, the current diagnostic approach continues to present problems affecting how practitioners treat and prevent schizophrenia due to its continued use of categories of psychosis. The consensus underlying these continued problems in the diagnosis and treatment of schizophrenia is the gap in understanding that still exists in the research surrounding the pathophysiology of schizophrenia (Insel, 2010). This study parallels the changes implemented in the DSM-5 and emphasizes psychosis dimensions in schizophrenia, whilst placing greater importance on heterogeneity, symptom severity, and variability.

Schizophrenia Characteristics

Understanding of the underlying psychopathology of schizophrenia has progressed significantly since the time of Kraepelin (1899) and Bleuler (1911). Kraepelin (1899) emphasized the progressive worsening of a person's cognitive and emotional functions, dementia, and the progression of cellular degeneration presumed to underlie the nature of the syndrome (as cited in McCarley, Shenton, O'Donnell, & Nestor, 1993). Bleuler (1911) was the first author to apply the term schizophrenia to the disorder based on his understanding of the disorder as a splitting of the psyche resulting in significant "psychological disturbances" (as cited in McCarley et al., 1993, p. 37). Seidman (1983) provided a critical review of studies of schizophrenia as a brain disorder. Based on his

review, Seidman arrived at three primary conclusions: brain dysfunction has been identified in patients with a diagnosis of schizophrenia, there exist two or three patient subgroups that exhibit differences in brain irregularities and clinical presentations, and these brain dysfunctions are associated with symptom presentation, disorder trajectory, and overall outcome.

Positive Symptoms

Schizophrenia symptoms are currently described as three primary clusters: positive, negative, and cognitive. The positive symptoms of schizophrenia are described by Kandel, Schwartz, Jessel, Siegelbaum, and Hudspeth (2013) as “positive or psychotic symptoms include mental phenomena that do not occur in healthy people such as hallucinations and delusions” (p. 1390).

Delusions. In his review of the literature of organic delusions, Cummings (1985) stated that they were present in various disorders that impair brain processes. He further implied that delusions may be an indication of dysfunction in the central nervous system or may co-exist with other primary symptoms such as hallucinations.

Krishnan, Keefe, and Kraus (2009) proposed that neural network dysfunctions, due to impaired higher levels of perception and hierarchical temporal processing, contributed to the errors in perception and thought observed in individuals with schizophrenia. These authors suggested that impaired cortical intercommunication in the brain caused difficulties during initial memory formation for schematic representations at higher levels of cognition (Krishnan et al., 2009). Thus, faulty functioning of top-down processes to lower levels of cognition was hypothesized to result in erroneous percepts. These authors also indicated that it is through this repeated occurrence of erroneous

perception, as well as the emotional valence attached to the percepts, that individuals are likely to incorporate incorrect beliefs into their memory repertoire (Krishnan et al., 2009).

Hallucinations. A characteristic symptom of schizophrenia is hallucinations. Hallucinations may present themselves in the form of auditory hallucinations, visual hallucinations, or tactile hallucinations, with the former two being the most common. Hallucinations are experienced as perceptions through one of the previously indicated pathways, however, these perceptions are false and arise without any stimuli (APA, 2013; Waters et al., 2012). There are different proposed underlying causes for these experiences. In their review of the literature on the cognitive mechanism involved in auditory hallucinations, Waters et al. (2012) provided an integrated conceptual framework of auditory hallucinations. They proposed that auditory hallucinations arise because of the interaction of hyperactivity in the neural networks associated with auditory signals, top-down information specific to the individual's cognition and previous emotional experiences, and impaired executive function mechanisms, specifically, and frontal executive functions related to an individual's ability to "control and regulate thought and action" (Waters et al., 2012, p. 685). Specific executive functions thought to be involved were separated into distinct components: inhibition, attention and working memory, set-shifting, as well as perceptual and emotional quality components (Waters et al., 2012).

Another proposed cause for hallucinations, specifically auditory hallucinations, involves abnormalities in source monitoring of inner speech. McGuire et al. (1995) obtained findings associating the auditory hallucination predisposition with reduced activation of the middle temporal gyrus and the supplementary motor area, the former

associated with higher order processing of auditory information and the latter related to the individual's ability to imagine speech by another person. The latter area has also been directly related to an individual's ability to initiate the movement involved in articulation, and damage to this area has been associated with phenomena in which the individual may not recognize self-initiation. Thus, these authors hypothesized that damage to that region may cause patients to misattribute the nature and source from which the auditory information is originating (McGuire et al., 1995). Therefore, the presence of auditory signals, in combination with impaired neuropsychological functioning, and impaired cognitive functions, are prominent.

Formal thought disorder. The *APA Dictionary of Clinical Psychology* defines a formal thought disorder as “disruptions in the form or structure of thinking” (Van den Bos & American Psychological Association, 2013, p. 241). Some of the primary features of a formal thought disorder include the presence of derailment of thoughts, indicated by an individual's scattered connections of ideas that may be completely unrelated or related indirectly. These thought disruptions may be observed in an individual's speech or written process. Additionally, these loose associations often result in an individual's failure to arrive at the point of the subject (Van den Bos & American Psychological Association, 2013). A study by Sans-Sansa et al. (2013) obtained findings associating reduced brain volume amongst individuals with a chronic form of schizophrenia with formal thought disorder in the areas associated with speech and the orbitofrontal cortex.

Negative Symptoms

Defining negative symptoms of schizophrenia presents some difficulty in the literature due to the shared superficial characteristics that these symptoms share with the

cognitive symptoms of disorder. Andreasen, Olsen, Dennert, and Smith (1982) included symptoms of “impoverished speech and thinking (alogia), diminished emotional spontaneity and expression (affective flattening), loss of drive (avolition), loss of ability to experience pleasure (anhedonia), and impaired attention” (p. 198). More recently, Liemburg et al. (2013) introduced a two-factor model rather than the classical one-factor model of negative symptoms in schizophrenia. They proposed classifying negative symptoms into two primary factors: core negative symptoms and social emotive withdrawal. The Scale for the Assessment of Negative Symptoms (SANS) measures negative symptoms in this population on a five-factor model: “affective flattening or blunting, alogia, avolition-apathy, anhedonia-asociality, and inattention” (Sayers, Curran, & Mueser, 1996, p. 270). However, further examination of negative symptoms with a confirmatory factor analysis resulted in findings suggesting that a three-factor model for describing negative symptoms may be satisfactory. The three factors proposed by these authors were “diminished expression, inattention-alogia, and social amotivation” (Sayers et al., 1996, p. 269). This approach to the structure of negative symptoms may be more suitable due to issues of internal consistency of the five factors, and lack of correlation between some of the symptoms included in each category of the SANS (Keefe et al., 1992; Sayers et al., 1996). Some of the negative symptoms of schizophrenia present themselves as blunted emotional awareness and affect, reduced motivation, social withdrawal and isolation, and as poor thought and speech presentation (Kandel et al., 2013).

Neurocognitive Symptoms

Evidence from past research conducted on individuals with schizophrenia has

resulted in general agreement that the neurocognitive deficits resulting from schizophrenia are not specific to, or characteristic of, one domain, but instead generalize across most cognitive domains (Gold, Hahn, Strauss, & Waltz, 2009). Most current data surrounding cognition in schizophrenia has demonstrated that the most significant and severe impairments present in the neurocognitive domains of verbal and nonverbal working memory, functions of language production and comprehension, executive functioning, and processing speed. The latter, processing speed, has been found to be the most severely impaired (Aleman, Hijman, de Haan, & Kahn, 1999; Heinrichs & Zakzanis, 1998). However, impairments in this domain have been also been correlated with medication dose. Zabala et al. (2010) identified primary impairments in the areas of “attention, working memory, executive functioning, and verbal memory” (p. 230). Other authors (Brodeur, Pelletier, & Lepage, 2009; Palmer et al., 2008) indicated that cognitive symptoms of schizophrenia included varying levels of deficits in distinct domains, namely “attention, memory, processing speed, and executive functioning” (Brodeur et al., 2009, p. 1088).

Thus, most research surrounding schizophrenia dysfunction and its symptomology has investigated the neurological functions believed to contribute to working memory processes, namely frontal lobe activity. Schizophrenia causes profound negative impacts to the life of the individual who develops the symptoms. A primary goal of schizophrenia research should include furthering our understanding of the direct neurocognitive factors resulting in consequences across the various domains of functioning and the development of psychosocial rehabilitation techniques to assist in ameliorating the loss of functioning for daily living (Adcock et al., 2009).

Positive, Negative, and Cognitive Systems

A study by Addington, Addington, and Maticka-Tyndale (1991) investigated the correlations of positive and negative symptoms, and cognitive performance amongst individuals with schizophrenia. Assessment of symptoms and cognitive functioning was conducted at two separate times, the first during a period of patient hospitalization, and the second at a six-month follow-up. From this study, these authors concluded that negative symptoms were correlated with cognitive performance deficits on neuropsychological tests more than positive symptoms, and improved positive symptoms were associated with improved cognitive functions (Addington et al., 1991). In a study conducted in 2015 by Bagney et al., negative symptoms in patients with schizophrenia were found to be unrelated to cognitive functioning. These authors studied a sample of 80 stable outpatients using the Positive and Negative Syndrome Scale to evaluate negative symptoms and the MATRICS Consensus Cognitive Battery (MCCB) to measure cognition (Bagney et al., 2015).

Ventura et al. (2015) investigated links between theory of mind, neurocognition, negative symptoms, and positive symptoms. Results suggested an associated link between theory of mind and negative symptom severity, and ultimately affecting role functioning including work and school outcomes.

Why is it Important to Understand Neuropsychological Abilities in Schizophrenia?

Allardyce, Gaebel, Zielasek, and van Os (2007) recognized that a definition of schizophrenia that did not provide information on a real and valid construct would not be able to specify the actual underlying pathology and mechanisms by which psychosis arises. In their review of schizophrenia and psychosis classification, Allardyce et al.

(2007) indicated that although schizophrenia is a construct that has strong clinical utility, it fails to explain accurately the underlying nature of the disorder, and in turn results in its failure to be a valid construct. Thereby, this vague explanation of the underlying features of the disorder creates difficulty in a creating a unified diagnostic system, intervention and treatment. Shanks, Silverstein, Schenkel, Valone, and Nuernberger (1998) identified that the deficits in cognition were correlated with poor functional outcomes. Green, Kern, Braff, and Mintz (2000) provided an extensive review of the literature on the deficits in neurocognition and their relationship to functional outcomes, in schizophrenia. These authors divided functional outcomes into three domains: psychosocial skills, instrumental skills, and community skills (Green et al., 2000). In their study of predictors for cognitive remediation with persons with schizophrenia, Lindenmayer et al. (2017) supported the importance of identifying variations in cognition amongst patients for effective treatment and intervention and specifically regarding improvement in daily skills functioning. Green (1996) sought to identify the relationship of neurocognitive deficits in schizophrenia to domains of cognitive functioning, and found that deficits were related with functional outcomes and factors including the ability to benefit from psychiatric rehabilitation. Leifker, Bowie, and Howie (2009) also indicated from their findings that neuropsychological performance was in fact related to everyday outcomes. Thus, the evidence for the importance of understanding the neuropsychology underlying schizophrenia and its relationship to functional capacity in many domains of everyday functioning is essential.

This study seeks to expand awareness of the neurocognitive deficits present in persons with schizophrenia as a way of understanding the underlying pathology in the

context of real and valid constructs that may be considered in intervention and treatment, essential identifying cognitive factors that may increase patient ability to benefit from psychiatric and cognitive rehabilitation. Specifically, to the knowledge of this researcher this study differs to previous studies of neurocognition in this population in that it proposes to investigate implicit domains of cognitive functioning to identify potential strengths rather than deficits. Understanding neuropsychological ability, cognition, and thought amongst individuals with schizophrenia may provide insight into predicting functional outcomes, thereby providing information about neurocognitive abilities that may be capitalized on in rehabilitation efforts. This study contributes to this purpose. Some of these are discussed in the literature review.

Neuropsychology, Thought, and Cognition, and Rehabilitation Outcomes

The overall goal of this investigation is to contribute to the current understanding of neuropsychology and formal thought in schizophrenia, and apply this knowledge to functional outcomes. In their meta-review, Green et al. (2000) discussed the potential influence that this knowledge might have on psychosocial rehabilitation procedures. Two approaches proposed by these authors that may arise in the psychosocial rehabilitation setting as a result of better understanding of the underlying neurocognitive patterns included tailoring intervention methods to fit with the individual's neurocognitive profile and modifying the intensity of training delivery based on neuropsychological abilities (Green et al., 2000). Specifically, as concluded by Shanks et al. (1998), an understanding of, and ability to delineate, cognitive strengths and weaknesses in a group or in an individual enables the opportunity to tailor interventions to individual needs. Additionally, as stated previously, understanding the neuropsychology of the patient

population may facilitate rehabilitation efforts to ameliorate deficits in everyday functions in domains including social and vocational.

Gaps in Research and Purpose

As can be inferred from the discussion thus far, understanding behavior in schizophrenia in the context of neuropsychological processes and cognitive functions is important in order to develop a valid construct of schizophrenia. This may assist in creating valid and effective interventions that may be generalizable to the daily living contexts for individuals included in this population. Advancing research in this domain may facilitate developing tailored interventions and rehabilitation programs that are applicable to functional outcome domains, such as psychosocial and community integration. Understanding the processes that create change amongst these individuals is essential for functional applicability. “While the role of impaired cognition accounting for functional outcome in schizophrenia is generally established, the relationship between cognitive and functional change in the context of treatments is far from clear” (Penades et al., 2010, p. 41).

Fosshage (2011) implied that prior to implementing therapeutic interventions intended for adaptive functioning, which are grounded in our current knowledge of therapeutic technique and development, it is important to understand the interactions by which these changes occur. He maintained that it is essential that the clinician be cognizant and knowledgeable of their patient’s neurocognitive abilities, and their capacity benefit from specific rehabilitative interventions prior to applying cognitive intervention techniques. One approach used to identify this information is neuropsychological testing. Thus, understanding cognitive functions and capabilities in

specific populations is necessary prior to implementing treatments, and this information is attainable using empirically validated methods. For effective transformations in adaptive functioning, the underlying neurological and cognitive processes contributing to change in the patient must be foremost in a practitioner's knowledge and understanding (Fosshage, 2011). Hence, measuring neuropsychological processes in this population is necessary to understand cognitive functions that are impaired, or possibly spared, for adoption of targeted clinical and therapeutic interventions (Gold, 2004). Information about an individual's strengths or impairments can facilitate intervention by clarifying what cognitive techniques the individual may be able to benefit from, and which they will not.

This study contributes to the literature on the relationship between neurocognitive function and thought in schizophrenia. McGrath and Richards (2009) put forth a strong argument for developing models to clarify and learn about the underlying biological functions. They emphasized the importance of developing models to identify anomalies in neurological connections that can be observed early in the disorder. One of the objectives of this investigation is to expand the current research and understanding of neurobehavioral and cognitive processes that contribute to thought and learning in schizophrenia. This study investigated various domains of implicit learning and cognition in individuals with schizophrenia to contribute to a greater understanding of the processes and capabilities of learning in individuals with schizophrenia. This investigation focused attention on the deficit in findings for specific implicit/nondeclarative learning processes in this population, resulting from this domain being significantly understudied as compared to the domain of explicit/declarative learning. The need to explore and develop

the neuropsychological and physiological underpinnings of the disorder is essential for further development of cognitive rehabilitation approaches, to effectively result in greater reintegration into the daily living skills needed by this population (Brekke, Raine, Ansel, Lencz, & Bird, 1997). As reported previously in the introduction, Lindenmayer et al. (2017) indicated the importance of cognitive variations in patients for establishing effective treatment and improving in daily skills functioning. Leifker et al. (2009) also indicated also emphasized the importance of neuropsychology and its relationship to everyday outcomes.

Some examples of daily living skills include the ability to go grocery shopping independently, manage finances, ability to use public transportation independently, initiate and carry out recreational activities, and cook independently. Medalia and Choi (2009) discussed cognitive remediation in schizophrenia. They summarized that various investigations studying participant response to cognitive remediation have demonstrated variable response to remediation. They emphasized the importance of identifying factors that may be influencing this variability. One example of such factors hypothesized by these researchers believed to influence treatment outcomes in this population is patient variables. Medalia and Choi (2009) explained that cognitive remediation differs from other rehabilitative techniques, such as those included in the recovery model, by directly targeting underlying neuropsychological functions believed to contribute to thought control. They further differentiated psychiatric rehabilitation, and indicated that this approach connects functional outcome goals with the specific interventions included in cognitive remediation. Thus, this approach facilitates developing interventions that target specific underlying neuropsychological processes that are believed to influence cognition

in schizophrenia, and overall functional outcomes. Developing understanding of less studied neuropsychological processes in schizophrenia, specifically implicit cognition, may contribute to this.

Chapter II: Review of the Literature

Models of Schizophrenia Disorder Categorization/ Etiology of Schizophrenia

Three primary models have been proposed to explain schizophrenia: “(a) the categorical subtypes model, (b) the dimensional model, and (c) the unitary model.” (Andreasen, 1999, p. 909). The categorical subtypes model highlights the well-known Type I/Type II subtype differentiation, in which the positive vs. negative vs. mixed types are described based on symptom presentation. The dimensional model divides the disorder into three dimensions: two of the dimensions include the positive symptoms resulting in psychosis dimension and a disorganized dimension. The third dimension included in this model is made up of the negative symptoms. Alternatively, the unitary model describes schizophrenia as the result of faulty cognitive processing following abnormal neural development (Andreasen et al., 1999). As discussed, the current diagnostic trend in schizophrenia places emphasis on a dimensional continuum, consistent with the dimensional model presented by Andreasen et al. (1999). This model, in combination with current changes in approach to schizophrenia diagnosis implemented into DSM-5 correlates directly with the approach and direction of this study. Specifically, this study aimed to develop a more detailed understanding of learning and cognition in individuals with schizophrenia in the context of heterogeneity placement along the dimensional continuum (Cuthbert & Insel, 2010; Insel, 2010).

Neurotransmitter and chemical networks. *The dopamine hypothesis.* Various neurotransmitter activity models have been proposed to explain both the positive and negative symptoms of schizophrenia. One of the leading models is the dopamine hypothesis. This is a notoriously provocative model, primarily due to the mixed evidence

that exists. This model proposes that the positive and negative symptoms, as well as the cognitive and physical changes that arise in schizophrenia, can be explained through four pathways in which the dopamine activity has been shown to be as abnormal. These include the “mesolimbic pathway, the mesocortical pathway, the nigrostriatal pathway, and the tuberoinfundibular pathway” (Mueser & Jeste, 2008, p. 28). However, evidence from PET studies and postmortem studies have not fully supported the dopamine hypothesis (Mueser & Jeste, 2008).

The dopamine cholinergic theory. Another neurochemical model that exists to explain the fundamental symptomology characteristics of schizophrenia is the dopamine-cholinergic theory. Findings obtained in an investigation into the muscarinic cholinergic activity revealed increased levels amongst individuals with schizophrenia (Tandon et al., 1991). Consistent with other models of the dopamine-cholinergic activity, increased activity of the cholinergic system was associated to increased negative symptoms. Additionally, an inverse relationship between increased cholinergic activity and positive symptoms was observed (Tandon et al., 1991).

The glutamate hypothesis. A third neurochemical model to explain some of the symptoms observed in schizophrenia is the glutamate hypothesis. Basically, this model stems from the dominant role glutamate neurotransmitter plays in the central nervous system, and the deficient levels of this neurotransmitter detected in the spinal fluid of schizophrenics (Mueser & Jeste, 2008). The glutamate hypothesis is associated with the negative symptoms and cognitive deficits observed amongst individuals with schizophrenia. The basic premise states that there is a reduction in *N*-methyl-D-aspartate (NMDA) ionotropic receptor activity, one of the receptors to which glutamate binds,

resulting from decreased glutamate synthesis. The hypothesis further indicates that this hypoactivity in NMDA receptor activity results in a reduction in the GABA system activity, giving rise to the negative symptoms and cognitive deficits is observed in schizophrenia (Belforte et al., 2010; Gordon, 2010; Moghaddam & Javitt, 2012). The NMDA receptor is thought a key role in memory through means of long-term potentiation (Belsham, 2001).

Genetic models of schizophrenia. Extensive investigation into the underlying genetic influence on schizophrenia has resulted in the development of novel models for explaining the disorder. One of the leading, and most notorious, models for explaining human genetics is the *common disease-common alleles* model proposed by Chakravarti (1999) in his review of gene diversity in the population. This model essentially proposed that susceptibility to schizophrenia is the additive effective of common genetic variations, each exerting a small effect, their interaction with environmental factors that are shared within the population (Chakravarti, 1999; Gottesman & Shields, 1982). However, more recent genetic models of schizophrenia have proposed that the predisposing mutations are in fact rare and may be specific to a particular patient or family genetic makeup (McClellan, Susser, & King, 2007; Walsh et al., 2008). Additionally, based on the findings of their investigation, Walsh et al. (2008) suggested that the genetic disruptions caused by structural mutations were prominent in brain development pathways. Some examples of the pathways include “synaptic long-term potentiation, axonal guidance signaling, and glutamate receptor signaling” (Walsh et al., 2008, p. 540).

The most recent findings around genetics and their association to schizophrenia were discovered in the summer of 2014. A molecular genetic study directed by professor

Michael O'Donovan compared genetic samples from individuals from 30 different countries, with total comparisons from approximately 37,000 patients with 110,000 controls without the disease. Findings from this study revealed a total of 100 genes leading to increased susceptibility to schizophrenia. Based on the recollections of the authors reporting on this study, these genes were believed to regulate intrabrain communication and immune system (O'Donovan, 2015). Additional models that exist in the literature have attempted to explain some of the phenotypes of the disorder, specifically thought and cognition. Some of these are discussed in the following sections.

Model of delusions and hallucinations. Following their review of the various proposed hypotheses of schizophrenia that exist in the literature of models of schizophrenia, Krishnan et al. (2009) proposed that schizophrenia is a hierarchical processing disorder. These authors argued that schizophrenia symptoms result from impairments in reality monitoring, perceptions, and predictions based on memory (Krishnan et al., 2009). In their review, emphasis was directed toward memory as it pertains to recognition abilities and learning, through bottom-up and top-down processes. These authors argued that these combined impaired processes produce distorted constructs of the world amongst individuals with schizophrenia (Krishnan et al., 2009).

Model of memory of schizophrenia. Brébion, Gorman, Malaspina, and Amador (2005) proposed a model of verbal memory and clinical symptoms. These investigators divided memory impairments into two measurable categories, memory efficiency, and memory errors, that were measured through correct verbal responses and memory errors, respectively (Brébion et al., 2005). The findings of this investigation identified a positive correlation between memory errors, and no relationship between positive symptoms and

memory efficiency. Furthermore, avolition was related to memory efficiency impairment, but unrelated to memory errors; however, other negative symptoms such as withdrawal and blunted affect were inversely associated with memory errors. These findings are consistent with the previously discussed model by Krishnan et al. (2009) of delusions and hallucinations, attributing errors to source monitoring. Brébion et al. (2005) proposed that the observed inverse relationship between memory error and negative symptoms may be the lack of emotional valence placed on the verbal information, or the context of the interaction, consistent with such symptoms.

Model of medical conditions and metabolic syndrome in schizophrenia. In

their review of the literature, Stone and Hsi (2011) discussed declarative memory deficits, hippocampal abnormalities, and learning and memory deficits impairments in schizophrenia in the context of poor glucose regulation and the comorbidity of metabolic syndrome with schizophrenia. These authors presented information from various studies about the role of glucose in hippocampal functions such as learning and memory, as well as the high comorbidity of schizophrenia presence and metabolic syndrome which includes irregularities in glucose and insulin absorption as well as other conditions. From their review, these authors connected insulin and glucose to some of these features observed in schizophrenia (Stone & Hsi, 2011). They concluded that glucose irregularities contribute to the deficits in learning and memory observed in schizophrenia, specifically verbal declarative memory. Stone and Hsi (2011) also reviewed verbal declarative memory in non-psychotic biological relatives of patients, and indicated from their review that these deficits were also present to a milder degree and without the forgetting rates observed in patients. Furthermore, they reported that schizophrenia

patient relatives also exhibited higher rates of glucose and insulin irregularities and similarities in the presence of genes associated with susceptibility for these medical conditions. Thus, they concluded this to support their hypothesis that glucose and insulin irregularities and other conditions contribute to the etiology of schizophrenia (Stone & Hsi, 2011).

A study by Venkatasubramanian et al. (2007) investigated IFG-1, cortisol, and glucose levels in patients with schizophrenia as compared to healthy controls. Findings of this study revealed that the patient group had significantly lower IFG-1 levels which were also inversely correlated with hallucination scores, and scores on the Scale for the Assessment of Positive Symptoms (SAPS; Venkatasubramanian et al., 2007, p. 1558). Venkatasubramanian et al. (2007) also reported that patients presented with higher insulin resistance as compared to healthy controls.

Demirel, Demirel, Emül, Duran, and Ügur (2014) investigated the relationship between schizophrenia and metabolic syndrome and insulin-like growth factor-1 (IGF-1). Contrary to various previous studies, these authors did not find differences in IGF-1 in individuals with the disorder in comparison to controls. This study further aimed to discover the presence of metabolic syndrome in the patients, and the possible relationship of the use of antipsychotic medications to the various symptoms of metabolic syndrome. They did not obtain any significance results for this relationship either. Contrary to much of the existing research in this area, Demirel et al. (2014) did not obtain any significant findings regarding IGF-1 levels in each of the groups. The authors did, however, observe significant difference in insulin resistance between the experimental group and third group which consisted of patient siblings (Demirel et al., 2014).

Schizophrenia as a Neurodevelopment Versus Neurodegenerative Disorder

The source of the structural abnormalities hypothesized to govern the schizophrenia profile has been a significant debate in the history of schizophrenia pathogenesis. The initial cause and progression of schizophrenia symptoms has been hypothesized to be both the result of neurodevelopmental mechanisms, and contrarily, also a neurodegenerative process. Until the last two decades, it was believed that schizophrenia was a neurodegenerative disorder, characterized by the term coined by Kraepelin: “dementia praecox.” Kraepelin (1919, as cited in McCarley et al., 1993) speculated that schizophrenia symptoms resulted in impaired performance of the temporal and frontal lobes of the brain caused by a neurodegenerative disease similar to that observed in dementia. The current evidence that exists in the literature has revealed that schizophrenia can be more accurately described as a neurodevelopmental disorder. The pioneering author to conceptualize schizophrenia as a neurodevelopmental disorder as opposed to neurodegenerative was Weinberger in 1987 (McCarley et al., 1993).

Neuropathological studies analyzing brain functioning of individuals with a schizophrenia diagnosis have suggested ectopic brain changes. From these findings, it was inferred that these neurobiological abnormalities occurred during the developmental stage of cell migration, indicating these changes occurred prior to birth. Postmortem studies have identified cells that may have been displaced during the primary phases of neuronal migration, evidenced by their presence or absence in distinct brain regions (Bearden & Cannon, 1998). Another hypothesis of the epidemiology of schizophrenia researched in the literature has to do with the time at which disturbances occur during development. For example, Mednick, Machon, Huttunen, and Bonett (1988) investigated

the hypothesis of a correlation between adult schizophrenia and fetal exposure to viral infection and the last two trimesters of development. These researchers used the Type A2 influenza epidemic of 1957, the rates of schizophrenia births and individuals who were admitted the psychiatric hospital presenting with schizophrenia as the variables in their study. They observed what they termed “the second-trimester effect” (Mednick et al., 1988, p. 189). The findings suggested an increased incidence of schizophrenia amongst individuals who were exposed to the viral infection during the second trimester of fetal development (Mednick et al., 1988).

As indicated previously under the glutamate neurotransmitter hypothesis, NMDA receptor hypoactivity is presumed in schizophrenia. This primary receptor for glutamate, NMDA, also plays an important role in an individual’s early development. This receptor is crucial for guiding early axonal migration, and it also plays a key role during early brain pruning stages, all of which occur during gestation (Mueser & Jeste, 2008). Belsham (2001) discussed glutamate as it pertains to psychiatric illness and the prominent role of glutamate in neuronal development and plasticity during developmental stages for critical neural connections. Thus, reduced glutamate concentration observed in the cerebral spinal fluid has been presumed to result in NMDA hypofunctionality, receptor upregulation, and has been associated with symptoms of schizophrenia (Belsham, 2001).

Temporal changes. Further evidence supporting schizophrenia being the result of neurodevelopmental processes was proposed following discoveries of abnormal nerve cell migration to brain regions such as the temporal area as well as the frontal area associated with speech (Bearden & Cannon, 1998; Jakob & Beckmann, 1986). Another line of evidence supporting the origins of schizophrenia as a neurodevelopmental

disorder arises from the postulation of the presence of early dysfunctions in the temporolimbic systems. These early dysfunctions negatively impact connections between the temporal-limbic and prefrontal brain area, ultimately causing increased release of dopamine in the striatum (Pankow, Knobel, Voss, & Heinz, 2011). This results in a deregulation in the processes of excitatory glutamate projections to the central brain regions where dopamine is created (Pankow et al., 2011). These abnormalities have been believed to produce the negative and cognitive symptoms observed amongst individuals with schizophrenia.

Changes during the stage of symptom onset. An investigation by Borgwardt et al. (2008) identified abnormalities in the cortex amongst participants identified to be at-risk for developing schizophrenia based on genotype. Other outcomes of this investigation resulted in identification of various cortical changes that appeared to occur during the phase in which individuals develop psychosis. Some of the identified areas in which transformations were observed in this study included the frontal, temporal, and parietal regions (Borgwardt et al., 2008).

Francis et al. (2013) analyzed MRI scans for 46 “adolescents or young adult offspring (OS) of schizophrenia probands” (p. 188). This investigation compared high risk offspring with controls who were matched for age. They discovered altered white matter volume in various areas of the brain, specifically reduced levels of white matter in both hemispheres as well as in the left parietal cortex (Francis et al., 2013). Neural synapses in the white matter are the source of inter- and intra- brain region connectivity. Interconnection impairments could contribute to many the symptoms observed in the schizophrenia disorder (Francis et al., 2013, p. 191).

Kandel et al. (2013) discussed factors occurring during adolescence that may contribute to schizophrenia. The authors explained that brain development factors, including synaptic pruning, and changes in the neurotransmission of dopamine at this stage of life, supported the hypotheses suggesting these factors as contributors of the disorder. They indicated that enlarged ventricles and abnormalities observed in the cortex suggested the presence of irregular neural activity prior to individual's displaying symptoms (Kandel et al., 2013). Thompson et al. (2001) used brain mapping to study brain patterns during developmental stages of schizophrenia. These investigators observed progressive grey matter losses across distinct brain regions amongst adolescents with early onset schizophrenia during the development stages of the disorder. The subjects in this study satisfied the DSM-III-R diagnostic criteria for schizophrenia and were assessed on the SAPS, SANS, and BPRS (Thompson et al., 2001). Boksa (2012) reviewed previous findings in the literature correlating synaptic pruning that occurred during the early stages of development with schizophrenia, and decreased brain volumes amongst individuals with schizophrenia, following the initial onset of psychosis. Palmer, Dawes, and Heaton (2009) reported consistent findings in the literature indicating that the average reduction in IQ during initial symptom onset in first-episode schizophrenia ranged between five and ten points.

Cognitive progression. Rajji et al. (2013) observed that individuals with schizophrenia, as compared to normal controls, demonstrated a similar cognitive decline in aging. These researchers recognized that although these individuals experienced global cognitive deficits, these deficits were comparable both early and late in life. Thus, presenting further evidence that schizophrenia is, in fact, neurodevelopmental as opposed

to neurodegenerative such as is the case with dementia (Rajji et al., 2013). As stated by Grilli, Toninelli, Uberti, Spano, and Memo (2003), dementia of the Alzheimer's type is believed to arise as a consequence of neuronal degeneration that occurs in the cerebral cortex, as well as in specific areas of the limbic cortex. Rajji et al. (2013) also noted that they used the MCCB, and that all of the cognitive domains, except for the social cognition domain, were accounted for (p. 109).

Neuropsychology Findings in Schizophrenia: What does the Literature Tell us about Neuropsychology and Neurocognition in Schizophrenia?

One of the most widely cited meta-analyses discussing neurocognition in schizophrenia was completed by Heinrichs and Zakzanis in 1998. In their analysis, the researchers sought to answer four questions about neurocognitive functions between individuals with schizophrenia and normal controls, as well as within group information in the patient population:

1. Does neurocognitive testing provide reliable evidence of impairment in schizophrenia and what is the average magnitude of difference between patients and healthy controls?
2. Do tests of specific neurocognitive functions (e.g., memory, language, attention) reveal similar magnitudes of difference between patients and controls or some aspects of neurocognitive performance spared in the illness?
3. Are there relationships between neurocognitive impairment and clinical and demographic attributes of patients and controls?
4. Are there relationships between specific neurocognitive functions and more general tests of ability (e.g. IQ)? (Heinrichs & Zakzanis, 1998, p. 426)

Heinrichs and Zakzanis (1998) found that individuals with schizophrenia exhibited deficits across many neurocognitive domains, and no single construct or specific task deficits specific to the population. Palmer, Heaton, Paulsen, Kuck, and Braff (1997) obtained similar findings: Patients who presented in the normal range as measured in global neuropsychological ratings demonstrated impaired learning when compared to healthy controls.

Findings by Heinrichs and Zakzanis (1998) also revealed that the largest effect sizes of patient's performance compared to controls were in the domains of "global verbal memory, performance, full-scale IQ, Continuous Performance scores and word fluency," and the smallest were observed on tasks of "block design (WAIS-R), Vocabulary, and non-WAIS-R-IQ" (p. 434).

A second meta-analysis frequently referred to in the research was led by Mesholam-Gately, Guiliano, Goff, Faraone, and Seidman (2009), who observed significant deficits across ten domains of cognitive functioning amongst first-episode schizophrenia. The authors included forty-three studies in their analysis, and participant mean age across the studies was twenty-five and a half years of age. The mean effect sizes observed in the ten neurocognitive domains ranged from, -.64 to -1.20, and greatest effect sizes were observed in the domains of immediate verbal memory, and processing speed (Mesholam-Gately et al., 2009). A study by Dickinson, Goldberg, Gold, Elevag, and Weinberger (2009) examined cognitive performance amongst schizophrenia patients, siblings of patients, and normal controls. Their observations indicated that schizophrenia patients demonstrated greater variability in cognitive performance, and that performance was not domain specific as was observed in the control and sibling groups (Dickinson et

al., 2009). The findings across these studies point to a somewhat clear consensus that persons with schizophrenia appear to exhibit widespread neurocognitive deficits not specific to a single construct, but particular impairments have been observed in verbal and processing speed tasks. Therefore, the key to advancing our understanding of how to best serve this population lies in identifying neurocognitive abilities that are less compromised within the population rather than comparing them to only typical populations.

A study by Faraone et al. (1995) assessed neuropsychological functioning in schizophrenic patient relatives as compared to typical controls. The authors discovered impairments in the schizophrenia relatives in the domains of abstraction, verbal memory, and auditory attention, which they concluded to be potential risk factors for identifying schizophrenia (Faraone et al., 1995). They suggested that these results corroborated the hypothesis that there is a genetic factor that results in these impairments, and that this factor may also be a factor that predisposes an individual to schizophrenia. Thus, it was Faraone et al.'s (1995) tentative summary that neuropsychological measures may be effective in determining individuals carrying the schizophrenia genotype, especially in cases where these individuals may to be identifiable by more traditional psychiatric assessments.

A study by Palmer et al. (2010) researched possible intra-person, as opposed to the traditional inter-group studies, cognitive profile differences between a group of schizophrenia outpatients, and matched healthy controls. The authors indicated that their basis for studying cognition at an intra-person level was to bypass the limitations that arose when attempting to develop core cognitive profiles for this population as a result of

the heterogeneity with which these patients present. The authors' goal for this study was to investigate the cognitive differences between these groups specifically, crystallized intelligence measured by the verbal comprehension index (VCI) compared to the other five factors from the Wechsler Adult Intelligence Scale, third edition (WAIS-III) and Wechsler Memory Scale, third edition (WMS-III) six-factor model. Confirming some of their original hypotheses, their findings suggested significantly higher functioning as compared to the other five domains. Compared with healthy controls, the patient group presented with significantly greater discrepancies between the visual episodic working memory domain and the other domains (Palmer et al., 2010). Contrary to the hypotheses of the authors, the auditory episodic working memory and working memory domains did have significant discrepancies with other domains in the patient group compared with the healthy control group (Palmer et al., 2010). Thus, it may be understood that persons would perform better on tasks based in crystallized knowledge and those tasks that apply auditory and working memory than those based in fluid knowledge and visual memory. One of the comparisons included in this current study directly compared these two domains in an incidental learning condition.

Marcopulos and Kurtz (2012) discussed various neurocognitive findings in the schizophrenia literature across the cognitive domains of cognition using Cohen's *d* effect size standards. Overall, they demonstrated moderate to low deficits in attention, between moderate to large deficits in "verbal and nonverbal episodic memory," and large "deficits in verbal and nonverbal working memory." The largest deficits were in processing speed which were in the severe range, large deficits in executive function, and moderate deficits in the sensory processing domain (Marcopulos & Kurtz, 2012, p. 6). In their study

comparing differential impairments in cognitive profiles between a group of outpatients with schizophrenia, Palmer et al. (2010) revealed that patients presented with more processing speed impairment than their previously hypothesized greater impairment in working memory, as compared to other ability domains. Knowles, David, and Reichenberg (2010) indicated that the severe deficits observed in processing speed were likely associated with antipsychotic exposure and dose size. Marcopulos and Kurtz (2012) also included the findings by Knowles et al. (2010). From these findings, it is expected that persons with schizophrenia will perform worse on verbal and non-verbal working memory tasks. One of the learning tasks of this current study that was used to measure incidental learning based in a fluid learning task relied heavily on working memory (digit-symbol coding). Therefore, it is possible based on the findings of the cited literature that participants would perform worse on this task based on its heavy dependence on working memory.

Areas of Spared Functioning

Investigations into cognition in schizophrenia have found preserved cognitive functioning across certain cognitive domains. Gold et al. (2009) performed a meta-analysis and concluded there are some domains that may in fact, be relatively spared despite the currently presumed generalized cognitive impairment. Some of these domains included “aspects of attention, procedural memory, and emotional processing...” (Gold et al., 2009, p. 294). In 2006, Gold et al. investigated attention control through five different experiments that assessed selective attention and its role in working memory. Their findings revealed that participants’ ability to apply selective attention processes for the purpose of encoding and storing target data remained unaffected. In this study, the

investigators identified a preserved ability to use both abstract and peripheral cues for target information selection (Gold et al., 2006). The implications of these findings could be tremendously important for tailoring effective cognitive rehabilitation and functional skills learning. Tailoring interventions to monopolize on these relatively spared cognitive processes could improve how this population benefits from interventions. Gold et al. (2006) claimed that specific aspects of selective attention, for the purpose of storing information in visual working memory, might be intact amongst individuals with schizophrenia. The researchers, and many reviewers of this investigation, recognized the possible limitation of this study and the problem of the power because of the small sample size. Gold et al. (2006) also correctly explained that this observation should not be generalized to different attention processes involved in other cognitive domains. They also implied that these findings, in addition to future advancements in our knowledge of schizophrenia, would contribute to models of cognitive impairment in schizophrenia (Gold et al., 2006). Additionally, developing this theoretical model may facilitate identification of strengths and weaknesses that may guide individualized and tailored models for treatment.

Huddy et al. (2009) also identified that motor inhibitory processes during conscious tasks were impaired, but in tasks involving non-conscious motor inhibitory control processes were unaffected in participants with first-episode psychosis. In the use of the term motor inhibitory processes, these researchers were referring to “inhibition of activated motor responses” (Huddy et al., 2009, p. 914). Schizophrenia participants recruited for Huddy et al.’s (2009) study were between the ages of 15 and 50 with no previous history of psychotic symptoms or episodes. The researchers indicated that at

one-year follow-up, following contact with participants and review of diagnosis, 27 of the 33 participants had received a schizophrenia diagnosis and the remaining six had been diagnosed with schizoaffective disorder (Huddy et al., 2009). As discussed in the introduction, cognition has been significantly associated with functional outcomes and performance in the rehabilitation setting (Bowen et al., 1994). Thus, identification of preserved or relatively spared cognitive functions as measured by task performance may provide insight into cognitive functions upon which individuals with schizophrenia could capitalize in rehabilitation.

Significant variation in intellectual ability is present amongst individuals with schizophrenia. Despite this heterogeneity, Badcock, Dragovic, Waters, and Jablensky (2005) found that processing speed was uniformly compromised across patients with varying levels of intelligence, including those higher functioning patients with more preserved intelligence. Moreover, comparing non-patient individuals to individuals with varying types and severity levels of schizophrenia impairment resulted in findings identifying higher functioning schizophrenics who displayed impairment in various domains as compared to those without schizophrenia (Badcock et al., 2005). In summary, preserved cognitive functioning amongst higher-functioning patients with schizophrenia as compared to lower-functioning patients with schizophrenia was observed at a level of significance; however, cognitive processing appeared to be impaired amongst all patient groups irrelevant of general intellectual functioning. Thus, the investigators concluded that processing speed was equally impaired between individuals in both preserved and compromised groups of participants with schizophrenia (Badcock et al., 2005).

However, Badcock et al.'s (2005) findings may not accurately reflect intellectual ability in individuals with schizophrenia. Weickert et al. (2000) obtained findings that indicated variability in neurocognitive patterns amongst individuals with schizophrenia. These researchers used results from the WRAT-R reading scores to obtain participant premorbid intellectual functioning, which was compared to current intellectual functioning to measure intellectual decline. Significant intellectual decline was observed in only half of the participants of their study. Approximately 25% of the participants in this study displayed spared cognitive abilities and intellectual functioning; however, this preserved group did display impaired functioning in attention and executive functioning (Weickert et al., 2000).

Neuropsychological Normalcy and Schizophrenia?

In their review of the literature, Palmer et al. (2009) reported that within the heterogeneity of cognitive profiles and symptoms observed in the schizophrenia patient population, approximately a quarter of patients present with typical neurocognitive profiles. Wilk et al. (2005) investigated neurocognitive profiles on individuals with schizophrenia based on the premise that individuals that present with schizophrenia may be neuropsychologically typical, as suggested in some of the studies covered in the literature. In their study, these authors compared neuropsychological profiles of schizophrenia patients with healthy controls who were based on their full-scale IQ scores that were matched within a three-point range, education, and age. Healthy controls and schizophrenia participants were then administered the WAIS-III and WMS-III to measure neuropsychological domains. Findings from this study revealed that schizophrenia patients were more impaired on tests measuring "processing speed, working memory,

immediate memory, and delayed memory” (Wilk et al., 2005, p. 781). They also observed better performance by patients with schizophrenia than the control group without schizophrenia on measures of verbal and performance tests. Wilk et al. (2005) confirmed that these results aligned with the majority of previous literature findings, and further suggested that the patients with schizophrenia were able to attain matching full scale intelligence quotient (FSIQ) scores with different neurocognitive patterns and overall profiles. Wilk et al. (2005) also concluded that based on the fact that verbal abilities remained relatively stable and were an accurate measure of premorbid IQ, the cognitive decline observed in the previously stated domains demonstrated neuropsychological function decline (Wilk et al., 2005). Thus, patients who closely matched healthy controls on overall scale measures may, in fact, have had a premorbid IQ that was masking the overall decline in cognitive functions that they experienced as a result of schizophrenia (Wilk et al. 2005, p. 784).

Heinrichs et al. (2008) investigated various characteristics of schizophrenia patients with superior verbal abilities. Explicitly, these investigators studied how these patients compared to healthy controls with superior verbal functions across other cognitive domains, if both healthy controls and patients with superior verbal abilities deviated significantly in functional skills and real-life outcomes, and if there existed significant differences in clinical symptom profiles between schizophrenics with superior level verbal functioning and patients with conventional impairments. The major findings of this study suggested that verbally superior patients approached similar outcomes as superior normal controls in other cognitive domains, and for skills of daily life, and the two groups were found to diverge significantly in community support and assistance

needs (Heinrichs et al., 2008). Thus, the authors suggested that superior verbal ability in this population may, in fact, mitigate the degree of impairment in daily living skills experienced by these patients, but does not change the level of support they require as compared to patients with typical impairments (Heinrichs et al., 2008).

As is apparent from the discussion thus far, observations of neuropsychological functions in patients with schizophrenia have resulted in significant consensus in specific domains. However, many studies have presented mixed findings with regard to apparent less impaired cognitive functions, specifically particular patients with superior abilities.

Deficit and Nondeficit Schizophrenia

Carpenter, Heinrichs, and Wagman (1988) contrasted deficit schizophrenia with nondeficit schizophrenia. They indicated that the deficit symptoms referred to the negative symptoms that were enduring traits that remained present regardless of positive symptom severity or psychotic episodes (Carpenter et al., 1988). Patients included in the nondeficit type of schizophrenia subtype may still present with negative symptoms; however, these may not present as the primary symptoms, and demonstrate variable severity and endurance. Many previous studies in the literature investigating the relationship between deficit- and nondeficit-schizophrenia, and neuropsychology have produced varied results. Cascella et al. (2008) obtained findings demonstrating similar neurocognitive profiles between the two subgroups. They also identified some differences in symptom severity and nature of deficits between the groups, with the most notable difference observed in patient ability to initiate ideas, or ideational fluency. The deficit schizophrenia group demonstrated greater difficulty, and deficit, within this domain. These investigators obtained findings consistent with the majority of the

literature, associating deficit schizophrenia with more impoverished performance in the verbal fluency domain (Casella et al., 2008). This is consistent with the finding obtained by Heinrichs et al. (2008).

Top-Down and Bottom-Up Cognitive Processing

Bottom-up processing refers to activation of the senses and receptors by stimuli in the observer's environment. Top-down processing involves the knowledge that a person has about the perceptual stimuli, often involved in the perceptual experience. The separate and connected neurocognitive functions work both independently and in unison to guide the individual to consolidate his or her perceptual experience into one entire picture of the world. These cognitive constructs, bottom-up and top-down processing, have been hypothesized to direct the functions of attention and guide the information filtration processes after perception of information. During information filtration processes, bottom-up and top-down processes assist in determining what environmental information enters working memory, as well as what transfers into long-term storage memory. This information eventually either becomes stored into existing schemas, or new ones are created (Gold et al., 2006; Goldstein, 2005). The ability to create and maintain reality depends on higher cognition processes that work to combine top-down and bottom-up information to produce an image of reality (Keefe, Arnold, Bayen, McEvoy, & Wilson, 2002).

The functions of bottom-up and top-down processing, in combination with attention and perception mechanisms, contribute to the individual's overall perceptual experience of their environment. Combined, these activities facilitate the ability to acquire information from the environment and create a unified perceptual experience that

contributes to the creation of thoughts and memories (Keefe et al., 2002). The neurological processes that contribute to the aforementioned cognitive functions are believed to be interrupted and impaired amongst individuals with schizophrenia. Specifically, it is presumed that the ability to perceive novel information, and to apply top-down knowledge from an existing prototype or schema of a similar experience for the purpose of integrating this new information is absent or impaired amongst individuals with schizophrenia. Krishnan et al. (2009) emphasized the role of these processes as potential contributors to some of the clinical features of the disorder, specifically delusions and hallucinations.

All of these components, metacognition, top-down processing, and bottom-up processing, are involved in how an individual assimilates his or her environment, as well as how he or she learns to interact in it. The role of individual learning types and processes regarding how they contribute to the acquisition and development of skills for daily living is the major theme of this study. Cognitive and processing abilities play a primary role in how individuals interact with the environment daily, their adaptability, and ultimately their functional success. Learning a new skill is a part of schizophrenia rehabilitation and intervention that assist to increase functional competency as it relates to everyday functions. Learning capitalizes on both processes, top-down and bottom-up. Therefore, better understanding top-down and bottom-up processes amongst persons with schizophrenia may help to not only explain neurocognitive functions and how they impact learning, but how rehabilitation and skill training should be tailored to increase outcomes.

Explicit Learning/Declarative and Implicit/Nondeclarative Learning

This study aimed to contribute to the existing literature on the relationships between the clinical symptoms in schizophrenia, particularly thought and cognition. Learning and memory can be divided into two categories: declarative or explicit learning, and nondeclarative or implicit learning. The former is characterized by learning that requires the learner to put forth a conscious effort toward encoding and learning the material. Another characteristic of explicit learning is that the material being learned is often, if not always, dependent on some explicit rule or set of rules (MacWhinney, 1997; Xie, Gao, & King, 2013). Russeler, Kuhlicke, and Munte (2003) further added to the definition of explicit learning when they indicated that explicit learning involves the individual gaining insight as to how their behavior is influenced by application of the learning that has been acquired.

The second category of learning is implicit learning. Horan et al. (2008) defined implicit learning as the process of acquiring information such as skill, habit, and knowledge through processes outside of conscious cognitive awareness (p. 606). Within the declarative domain there exists semantic and episodic categories, the former is made up of learning and memory for facts, and the latter encompasses information that an individual is able to remember about an event that he or she experienced personally, including temporal and contextual information about that event. Within the nondeclarative domain, the types of learning include priming, procedural learning primarily for skill acquisition, associative learning that includes classical and operant conditioning functions, and nonassociative learning that occurs in the cases of habituation and sensitization to stimuli (Kandel et al., 2013; Van den Bos & American Psychological

Association, 2013). One of the key characteristics differentiating explicit and implicit learning is the information learning, verbal or non-verbal, that takes place in the absence of the learner's conscious awareness. The person involved in implicit learning is unaware of the effect of the learning experience on their behavior and acquired learning (Danion, Meulmans, Kauffmann, & Vermaat, 2001; Horan et al., 2008; Russeler et al., 2003).

Explicit learning occurs during conscious learning activities, and entails error monitoring and hypothesis testing mechanisms (Russeler et al., 2003; Weinert, 2009). Explicit learning occurs through conscious control and effortful mental processes. One key distinction proposed by MacWhinney (1997) in his description of explicit learning lies in the distinct cognitive processes underlying explicit instruction and explicit learning. MacWhinney (1997) claimed that explicit learning does not necessarily occur in the presence of explicit instruction, and conversely explicit instruction does not have to take place for explicit learning mechanisms to occur. The significance of this distinction for this investigation is to realize that for explicit learning to have occurred, the individual will have applied conscious, rule-construction learning through error-monitoring mechanisms (MacWhinney, 1997; Russeler et al., 2003; Weinert, 2009).

Neuropsychology of explicit/declarative and implicit/nondeclarative learning. The neurocognitive and neurophysiological mechanisms underlying the processes of declarative and nondeclarative learning are currently areas of great interest and debate in the literature. An investigation by Gureckis, James, and Nosofsky (2011) into implicit and explicit neural pathways revealed a unitary processing system. In accordance with previous research conducted on this topic, the findings did not provide evidence of a dual learning system of processing when learning categorical information.

Implicit perceptual learning was preserved amongst individuals with localized damage incurred primarily to the hippocampus. These findings were thus inferred to imply that hippocampus activity does not play a role in implicit learning (Manns & Squire, 2001).

Explicit learning/declarative and implicit/nondeclarative learning in schizophrenia. In an investigation conducted by Huron et al. (2005), individuals with schizophrenia displayed impairments in their ability to recall consciously previously learned information through explicit channels. From these findings, Huron et al. (2005) concluded that these participants experienced deficits in their ability to create learning associations through conscious channels. These functions were hypothesized to depend on purposeful information manipulation and organization (Huron et al., 1995).

Another investigation comparing explicit and implicit learning task performance amongst individuals with schizophrenia revealed findings indicating that they were impaired on explicit learning tasks, but not on implicit tasks (Gras-Vincedon et al., 1994). An investigation conducted by Schwartz, Rosse, and Deutsch (1993) showed preserved implicit learning functions amongst individuals with schizophrenia. Their investigation of implicit and explicit learning and memory measures in the population produced results indicating typical implicit learning and impairments in explicit tasks (Schwartz et al., 1993). Investigation into implicit learning mechanisms of individuals with schizophrenia conducted by Horan et al. (2008) found that these individuals exhibited heterogeneous abilities when completing “cognitive implicit learning tasks” (p. 613). Other findings revealed that participants with schizophrenia exhibited impaired functioning on explicit learning tasks. These participants demonstrated noteworthy impairment on tasks primarily dependent on learning through feedback and reinforcement mechanisms and

those requiring conscious learning and awareness (Horan et al., 2008, p. 614).

Activities of daily living are dependent upon the overlap and interaction between implicit and explicit learning mechanisms (Sun, Slusarz, & Terry, 2005). Various investigations have suggested complete separation in the activation of these mechanisms regarding distinct cognitive functions and behavioral activities. However, complete distinction is not easily accomplished, suggesting that some overlap will exist (Tilborg, Kessels, & Hulstijn, 2011). This suggests that impairment in one of these functions is likely to cause difficulty in activities of daily living and the ability to carry out daily life skills despite the relatively intact processes of the other. The components of the brain, although distinct and separate, work together as an integrated whole (Brain Injury Association of America, 2009). The research on declarative learning in schizophrenia has been plentiful, and well discussed in the literature. However, nondeclarative learning in schizophrenia has been far less researched.

Implicit Learning Strategies

Some of the mechanisms included in the implicit learning domain include procedural learning and repetition priming (Gras-Vincedon et al., 1994; Tilborg et al., 2011, p. 639). A third mechanism of implicit learning is incidental learning. These mechanisms were the primary focus of this current study. Repetition priming occurs when an individual's response to a stimulus is more effective, and occurs with more accuracy because of previous exposure to the stimulus. These mechanisms facilitate response to previously encountered stimuli when the initial learning occurred outside of the learner's conscious awareness. Thus, an individual's response to a stimulus is modified as a result of previous exposure to the stimulus. Priming can also occur when

previous contact with a stimulus influences a similar stimulus, not only an identical stimulus (APA, 2013). Procedural learning, also classified as a form of implicit learning, involves developing procedural memory for a cognitive or motor skill or skill set. Thus, repetition priming and procedural learning are employed in cognitive and motor skill acquisition and development, in the absence of conscious awareness (Goldstein, 2005).

Another form of implicit learning includes incidental learning. Incidental learning occurs through mechanisms in which reinforcement contingencies are non-applicable, but instead occur independent of awareness that the learning is occurring. As opposed to explicit learning, incidental learning does not occur through processes of error-feedback or reinforcement. This form of learning typically occurs outside the learner's conscious awareness, through observation and exposure to situational feedback (Doeller & Burgess, 2008, p. 5913). Thus, it may be inferred that incidental learning occurs in many aspects of our lives. Through incidental learning mechanisms, people are able to acquire information about their surroundings.

Priming in schizophrenia. Minzenberger, Ober, and Vinogradov (2002) conducted a meta-analysis of the effects of priming in semantic memory in schizophrenia participants. Based on their review, these authors presented four primary conclusions. First, they differentiated controlled processing from automatic processing, and concluded that changes to both forms of processing were characteristic in individuals with schizophrenia. Second, they concluded that the spreading activation in the automatic processing condition was heterogeneous in individuals with schizophrenia depending on length of history of medication, variations in cause and developmental course of schizophrenia, specifically, thought disorder, semantic memory retrieval and

configuration, and other “factors identifiable at physiological, neuropsychological, and clinical levels of analysis” (Minzenberger et al., 2002, p. 709). Third, they reported that patient’s performance in the controlled processing condition for semantic information was homogenously impaired in the schizophrenic group as compared to controls. They reported that this impairment seemed to be related losses in attention and organization processes. Fourth, they suggested that these two processes underlying semantic networks might be independent but related (Minzenberger et al., 2002).

A study by Bressi et al. (1998) sought to investigate implicit learning functions amongst individuals with schizophrenia. In their study, the authors compared performance by twenty participants with schizophrenia with significant deficits in explicit abilities and twenty healthy control participants on a lexical-semantic priming task and a sequence-skill learning task (Bressi et al., 1998). The primary objectives of this study were to address if and how priming and procedural learning are affected in patients as compared to individuals without schizophrenia. Findings from this study revealed that individuals with schizophrenia demonstrated implicit priming effects that were typical, although they completed fewer words than controls on the word-stem task. These differences were found to be non-significant. Significant differences were, however, observed on the explicit learning recognition portion of the trial. Additionally, a lack of observed significance on the sequence-learning task in the patient group resulted in these authors concluding that individuals with schizophrenia exhibited deficits in sequence procedural learning abilities (Bressi et al., 1998).

Procedural learning in schizophrenia. Fitts (1964) and Anderson (1982) described three critical steps in procedural learning. The first of these steps was the

cognitive stage, which they proposed to be characterized by an individual's efforts toward carrying out a novel skill based on a set of concrete instructions, or modeled examples. The individual will often engage in rehearsal of the new skill, and can characterize the task verbally. The second step was the associative stage, during which the learner is able to perform the new skill more smoothly and with fewer mistakes, often discontinuing the use of rehearsal strategies. The final step was the autonomous stage, in which the individual masters the skill and can perform the skill with negligible or no cognitive involvement (Anderson, 1995).

In a study of procedural learning in un-medicated patients with first episode schizophrenia, Purdon, Waldie, Wiman, Woodward, and Tibbo (2011) required patients to demonstrate reaction time and task accuracy in a serial reaction time task. There were also no significant impairments observed either between the patient and healthy control group, or within the patient group. From these findings, these authors concluded that procedural learning skills in this patient population were somewhat intact (Purdon et al., 2011). Gomar et al. (2011) investigated procedural learning abilities in patients and non-schizophrenic controls. The findings of this study indicated that there were differences in procedural learning abilities in this population depending on whether the task involved was a motor or cognitive procedural task. Results indicated that patients presented normal learning on motor tasks, and impaired learning on a prediction task, but also demonstrated normal learning on a motor task that was classified as a cognitive rather than motor task (Gomar et al., 2011).

A study by Kumari et al. (2002) investigated neural activity in patients using functional magnetic resonance imaging (fMRI) while the patients were engaged in a

procedural learning task. Participants were administered a non-verbal sequence-learning task from which findings demonstrated that the patient group did not benefit from procedural learning. The authors indicated that compared to controls, participants demonstrated deficit activity in brain areas associated with procedural learning, namely “the striatum, cerebellum, thalamus, cingulate gyrus, precuneus, and sensorimotor regions” (Kumari et al., 2002, p. 106). Some limitations to this study included the fact that only six male patient subjects were included. The authors also indicated another limitation in their findings; it was not clear if the observed diminished procedural learning in the patient group was the result of antipsychotic medication, or true consequences of the disease (Kumari et al., 2002). However, in another fMRI study by Woodward, Tibbo, and Purdon (2007) investigated the neural correlates associated with a serial reaction time task in siblings of patients as compared to control subjects. Observations revealed similar neural deficits in the sibling group as compared to healthy controls, specifically in the “regions of the PFC, the left angular gyrus, and basal ganglia” (Woodward et al., 2007, p. 312).

Incidental learning in schizophrenia. Incidental learning has not been researched in depth, and this is especially true in relation to schizophrenia, resulting in a significant lack of knowledge and understanding in this domain. Incidental learning is preserved amongst individuals with schizophrenia (Danion et al., 2001). A second investigation by Aaronson, Sugerman, and Hafetz (1966) in which incidental learning abilities amongst individuals with schizophrenia was compared to a group of alcoholics and a group of individuals without alcoholism or schizophrenia demonstrated these abilities to remain unaffected in the group with schizophrenia. In their discussion, the

authors claimed that individuals with schizophrenia demonstrated intact incidental learning functions; however, they also demonstrated reduced sensitivity to cues in their environment (Aaronson et al., 1966). This confirms the previously discussed deficits in conscious learning amongst individuals with schizophrenia, and suggests an area of preserved functioning.

Canan, White, and Bingham (2009) also found evidence for preserved incidental learning functions amongst individuals with schizophrenia. They found unimpaired incidental learning functions amongst youth in two distinct groups: young individuals with psychosis and young individuals with attention deficit hyperactivity disorder. No significant deficiencies in incidental sequence learning were observed amongst individuals in either group (Canan et al., 2009).

Learning and Skill Acquisition

An investigation by Russeler et al. (2003) researching the differences between individuals who learned through explicit means as compared to those who learned through implicit pathways established that those who learned through explicit means acquired more sequence knowledge. Sequence knowledge plays a significant role in the acquisition of skills and aptitudes involved in daily living skills. This study's findings demonstrated that individuals in the explicit learning skill group were better able to learn sequence knowledge through verbal channels (Russeler et al., 2003). Verbal channels contributed to their behavior monitoring proficiencies. This supports the notion that explicit learning is a valuable technique for behavioral learning through internal mental feedback and adaptation for individuals with normal, non-impaired explicit learning abilities. Furthermore, these findings have positively correlated compromised explicit

learning mechanisms with skills acquisition. This could explain some of the deficits in learning of daily living skills observed in people with schizophrenia.

Danion, Rizzo, and Bruant (1999) investigated recognition abilities amongst individuals with schizophrenia. Participant's recognition performance was measured for level of functioning during conscious recognition tasks as well as recognition tasks occurring outside the participant's conscious awareness. Results obtained in an investigation by Danion et al. (2001) revealed findings demonstrating that patients exhibited significant impairment on explicit recognition tasks. The findings of this study also demonstrated unimpaired performance by participants with schizophrenia on an implicit learning task. In this study, implicit learning was measured using an implicit grammar learning task. This negatively impacted their potential for psychosocial rehabilitation and independence through explicit learning techniques; however, these findings also identified cognitive functions that appeared less, or unimpaired amongst individuals in this population.

As suggested in Horan and colleagues (2008), explicit learning and memory functioning abilities paralleled the individual's possibility for success with daily experiences and the challenges of daily living in their environment and community. In their investigation, the researchers compared performance of 59 participants with schizophrenia to 43 controls in the domains of procedural learning, incidental learning, and explicit learning and memory, as measured by their performance on various tasks. Horan et al. (2008), consistent with previous findings in the literature, observed impaired performance by patients on explicit learning tasks. However, when the investigators compared performance by patients on two separate implicit learning tasks, one that

consisted of gradual learning through provided feedback and another that provided no feedback, subjects performed better on the implicit learning task that did not provide trial-by-trial feedback (Horan et al., 2008). Based on the findings across these studies it could be hypothesized that individuals with schizophrenia exhibit greater difficulties in knowledge and skill acquisition when the material is presented or taught through explicit learning means, thus, directly relating to difficulties in sequential knowledge acquisition and ultimately daily and community skill development. The current study observed implicit learning abilities in crystallized and fluid knowledge domains, which bypasses explicit and conscious learning mechanisms, as these have been found to be less impaired in individuals with schizophrenia.

Wessel, Haider, and Rose (2012) explained that the importance of implicit learning is its unique ability to allow “humans to adapt to regularities and contingencies” (p. 153) with which they encounter and interact in their regular environments. This statement emphasizes the inherent necessity of implicit learning functions for the adequate achievement of capabilities and adaptations required for daily living skills. Tilborg et al. (2011) proposed that rehabilitative techniques that activate implicit learning processes could be used as alternative forms of intervention for improving daily living satisfaction amongst individuals presenting with intact implicit learning functions. Thus, rehabilitative interventions capitalizing on implicit learning abilities should be further explored for this population. The method of strategy deployment of an intervention could be designed, taking into account the person’s strengths by, for example, using peripheral or abstract cues in the environment to facilitate learning and recall.

Cattell-Horn-Carroll Theory of Intelligence

Over the last two centuries, various theories of intelligence have been suggested and developed. One accepted theory of intelligence in psychology is the Cattell-Horn-Carroll (CHC) model of intelligence. Under this model, there exists three primary interconnected levels of cognition, called *stratums*. The first stratum (Stratum I) is composed of eighty specific and narrow abilities such as verbal reasoning (*K0*) and quantitative reasoning (RQ). The second stratum is made up of sixteen broad and general cognitive categories such as fluid intelligence (*Gf*) and crystallized intelligence. The model states that each category in the second stratum symbolizes a particular characteristic of cognition that can be further broken down into specific abilities, Stratum I abilities. This model also proposes that stratum involves a *g*-factor which is presumed to represent an individual's general cognitive ability, and includes all the first and second stratum factors (Flanagan & Dixon, 2013; McGrew, 2009).

Crystallized and Fluid Knowledge in Schizophrenia

Cattell (1963) developed a dichotomous theory of intelligence in the 1940's in which he identified two forms of intelligence named crystallized intelligence (*Gc*) and fluid intelligence (*Gf*). Cattell's definition for the former, crystallized intelligence, includes abilities that apply cognitive functions utilizing previously learned skills and knowledge not exclusive to a unitary learned ability, but including a collection of abilities and information obtained over time which is usually influenced by culture and the environment. He defined the second construct, fluid intelligence, as a general ability that loads on cognitive functions associated with problem solving in new contexts and situations (Cattell, 1963; Thorsen, Gustafsson, Cliffordson, 2014). Previously discussed

in this review of the literature, Palmer et al. (2010) conducted a study that compared crystallized learning abilities with five other factors from the WAIS-III/WMS-III six-factor model. Palmer et al. (2010) reported better functioning for crystallized intelligence than the remaining factors. The current study observed participant performance on a series of tasks based on different implicit learning methods: priming, procedural, and incidental. Some of these tasks were based on crystallized intelligence and some on fluid. Incidental learning can be applied for learning both crystallized and fluid knowledge, thus they were compared in order to test the specificity of this learning technique and how it related to these two intelligence types.

Cognition and Learning in Daily Living Skills

In the literature presented by Fischer, Holland, Subramaniam, and Vingogradov (2010), they identified cognitive remediation techniques believed to promote significant cognitive gains amongst individuals with schizophrenia. The researchers indicated that delivery of these techniques employs functions from specific cognitive domains including, but not limited to, working memory, and verbal, spatial, and episodic memory (Fischer et al., 2010). Thus, greater understanding of correlational factors between neural activity changes and cognitive and learning functions amongst individuals with schizophrenia should be considered foremost in schizophrenia research.

Individuals apply both implicit and explicit learning techniques for daily living skills. Some tasks that individuals must learn to accomplish in recovery from mental illness include learning to go to the grocery store independently, understanding how to use public transportation, and learning everyday social skills. Current research has provided substantial support indicating that cognitive deficits have significant impact on

an individual's skills for daily living, social functioning, and overall life satisfaction (Cohen, Forbes, Mann, & Blanchard, 2006). In discussing incidental learning as it applies to acquisition of skills for daily life, it has been suggested that the primary mechanisms for this type of learning include spatial and contextual processes. As noted in the previous pages, these learning processes have been hypothesized to occur in the absence of reinforcement contingencies (Doeller & Burgess, 2008, p. 5909). Individuals with schizophrenia have shown more deficits in tasks that depended on explicit learning processes rather than implicit learning. Therefore, investigating implicit and explicit domains of cognition amongst individuals with schizophrenia will further contribute to the literature on cognition in schizophrenia. Such contributions will contribute to improvements in functional domains amongst individuals with schizophrenia.

Relationships between Neurocognition and Functional Status in Schizophrenia

In their one-year longitudinal investigation into the relationship between neurocognitive skills and functional status among individuals with schizophrenia who received cognitive and psychosocial rehabilitation, Kurtz, Wexler, Fujimoto, Shagan, and Seltzer (2008) discovered a number of interesting results. Verbal learning abilities were determined to predict functional change amongst three individuals post-one year. However, the researchers indicated that, after one year, "positive and negative symptoms, crystallized verbal ability, sustained visual vigilance, problem-solving, and processing speed were not related to change in functional status after outpatient rehabilitation" (Kurtz et al., 2008, pp. 308-309). One major limitation of this study was that it only examined a few neurocognitive domains.

Another study by Bowen et al. (1994) identified a significant relationship between “cognitive functioning, interpersonal skills, and performance in elemental skills training tasks” amongst individuals with schizophrenia (p. 298). In this study, vigilance level (a type of attention) was identified to be considerably associated with elemental skills performance, and was identified as the cognitive skill most associated with interpersonal skills. Immediate recall ability was also identified as a cognitive skill substantially associated with elemental skills performance. The relationship between immediate recall ability and elemental skill performance was found to occur independently of the effects of vigilance. Thus, the authors concluded that cognitive impairments amongst individuals in this population were associated with decreased overall functioning and the relevance of individual cognitive functions, specifically in this case vigilance and recall memory, in the rehabilitation setting (Bowen et al., 1994).

As indicated previously, Green et al. (2000) published a meta-review of the association of between neurocognitive functions and outcomes in schizophrenia. Reliable findings revealed that secondary verbal memory was related to all of the functional outcome domains they measured: psychosocial skills, instrumental skills, and community and daily activities. Additional findings revealed that immediate memory related to psychosocial skill achievement, executive functioning, and verbal fluency related to community and daily activities effects, and sustained attention was associated with instrumental skills execution (Green et al., 2000). Amongst several limitations to the study, Green et al. (2000) acknowledged that the amount of participants included in each of these studies was widely variable, and thus they could not provide accurate allocation of degree of significance to the findings of each study.

Consistent with the conclusions made by Green et al. (2000), Tramley et al. (2002) also observed strong correlations between performance based skills and neuropsychological domains of memory, attention, executive functioning, and learning. Additionally, the researchers observed positive correlations between UPSA scores and each of the cognitive domains measured in their study, such as initiation/perseveration and psychomotor ability. Tramley et al. (2002) further examined individual UPSA scales independently, revealing associations between each of the subscales and many of the cognitive domains included in this study (Tramley et al., 2002). Cognitive impairments may or may not be directly related to overall clinical outcomes and the patient's quality of life, and identifying and targeting these cognitive factors could create changes in these variables (Gold, 2004).

Bowie, Reichenberg, Patterson, Heaton, and Harvey (2006) studied the relationship between three presumed interconnected variables—neuropsychological, performance, and real time skills—in 78 ambulatory schizophrenic participants between the ages of 50-85. Their findings revealed strong correlations between neuropsychological functions, as measured with a comprehensive test battery, and real-world functional outcome made up of three domains: interpersonal skills, community activities, and vocational skills. When Bowie et al. (2006) included functional capacity as a factor, they reported that there was a sporadic relationship between neuropsychological performance and functional domains. They also reported that relationship between the latter was mediated by functional capacity. Bowie et al. (2006) indicated that real-world functional skill performance was further affected by disease symptoms such as negative symptoms. In a later follow-up study by Bowie et al. (2008), specific relationships

between cognitive functions and social, functional, and real-world skills were observed. The findings further delineated distinct skills necessary for skill acquisition in some domains, and skill deployment in others. Some of their findings between verbal memory and executive functioning were important for functional competence but not for social skill competence, and a relationship between working memory, attention, and processing speed with both domains of competence was identified. In conclusion to their findings, the authors emphasized the critical need to identify relationships between specific cognitive functions and specific skills so they could be applied to specific treatment targets (Bowie et al., 2008).

As indicated previously, understanding of the underlying neuropsychology of individuals with schizophrenia as measured by neuropsychological test batteries can provide information about cognitive functions, impairments, and spared domains amongst individuals from this population. For this study, the Expanded Brief Psychiatric Rating Scale (BPRS-E) was used to measure symptom severity at time of assessment primarily for diagnostic purpose, and the WRAT-4 Word Reading subtest was included to assess grade reading level (per inclusionary criteria. Gender and age were also accounted for on the demographic questionnaire and in calculating T-scores where existing norms were being used in analysis.

Neuropsychological Testing and Schizophrenia: NIMH Initiative and MATRICS

The National Institute of Mental Health (NIMH) proposed the MATRICS initiative to develop a standardized measure for the purpose of measuring cognition, and changes in cognition, over time. One of the leading considerations during the development of the consensus battery by the NIMH was that the battery would serve to

not only demarcate distinct and distinguishable cognitive functions but also the different causes for the cognitive impairments. Specifically, the consensus battery was developed with the primary goal of creating a standardized battery for measuring and improving cognition in schizophrenia through its use in clinical trials. Following an extensive process by various experts in the field, ten tests were selected and included in the final MATRICS consensus cognitive battery. The cognitive domains measured in the battery include “speed of processing, verbal learning, working memory, reasoning and problem solving, visual learning, social cognition, and attention” (Nuechterlein et al., 2004, p. 29).

Medication and Cognition in Schizophrenia

Previous investigations have revealed the following findings regarding medication regimens among individuals with schizophrenia. Riedel et al. (2010) reported that a quantifiable amount of studies produced findings demonstrating the advantages of second-generation antipsychotics (atypical) over the first-generation typical antipsychotics on cognitive functions among individuals with schizophrenia. They also indicated that more recent studies that involved larger sample sizes have revealed more moderate effects. Selva-Vera et al. (2010) set forth on a study to understand the differences in cognitive functions following the use of either typical or atypical antipsychotics over time, as well as studying changes in cognitive functions following antipsychotic treatment over time. Findings from Selva-Vera et al.’s (2010) study revealed mixed results with regard to cognitive functioning change over time; however, they also observed significant results. The researchers concluded from their findings that atypical as opposed to typical antipsychotics did not produce results indicating cognitive benefit from the first over the later (Selva-Vera et al., 2010).

Canan et al. (2009) stated that typical antipsychotics block more of the dopamine receptors in the brain than other antipsychotics. Their findings revealed that this difference affected sequence-learning deficits, resulting in different sequence-learning abilities between individuals taking typical neuroleptics and patients taking atypical neuroleptics (Canan et al., 2009). Selva-Vera et al. (2010) proposed that the differences in neurocognitive effects of the two classes might have been due to the reduced neurological side effects of atypical antipsychotics, thereby supporting the individual's adherence to pharmacotherapy. Another proposed cause of worsened cognitive performance amongst individuals taking typical antipsychotics was the use of anticholinergic drugs to mediate the extrapyramidal symptoms caused by this older generation of drugs (Selva-Vera et al., 2010). An investigation conducted by Harvey, Rabinowitz, Eerdeken, and Davidson (2005) observed improvements in cognitive functioning, following treatment with Risperidone and Haloperidol, amongst patients presenting with early psychosis. These investigators further identified significantly higher scores on domains of verbal fluency and recall among participants being treated with Risperidone (Harvey et al., 2005). Similar findings were identified for long-term use of antipsychotics in the study conducted by Selva-Vera et al. (2010).

In addition to the observed associations between different medications and cognition that was introduced earlier in the introduction, one of the aims of the study by Demirel et al. (2014) was to study the possible existence of an association between atypical antipsychotic medication and metabolic syndrome. These authors obtained findings consistent with other research indicating the lack of association between the two factors (Demirel et al., 2014).

Participant medication type, dosage, and duration of treatment were not studied as primary variables in the current investigation. However, information about the participant's current medication, dose, socioeconomic status, and ethnicity was collected, as well as other factors that impacted abilities. This is discussed further under "Confounds" in the section titled "Data Processing Techniques" of the Methods chapter.

Summary

Mixed results about cognitive functions in individuals with schizophrenia have been established. As can be inferred from this review of the literature, some cognitive functions have been observed to be more impaired than others in individuals with schizophrenia. For example, numerous studies have determined verbal functions to be more impaired than non-verbal functions amongst individuals with schizophrenia (Cascella et al., 2008; Faraone et al., 1995; Heinrichs et al., 2008; Heinrichs & Zakzanis, 1998; Marcopulos & Kurtz, 2012; Mesholam-Gately et al., 2009; Stone & Hsi, 2011; Zabala et al., 2010).

Understanding the relationship between symptoms of the disorder and cognition may provide insight into the schizophrenic condition. As indicated previously, studies comparing cognitive functioning between individuals presenting with deficit and those with nondeficit-schizophrenia have resulted in observations indicating significant variability between these groups. However, some studies, including Cascella et al. (2008), have resulted in observations indicating homogenous neurocognitive profiles between these groups. As may be inferred from the extensive research in schizophrenia, most studies have focused on explicit knowledge and intelligence in schizophrenia. Although there do exist studies of implicit processes in schizophrenia, these are limited in

that there is not a study, to my knowledge, that combines research of various implicit mechanisms of thought into one comprehensive study.

Understanding the neurocognitive strengths and weaknesses in this population may further contribute to the current literature that exists about the association of cognitive dysfunction and symptom severity in schizophrenia. Cognitive remediation, and rehabilitation are essential for improving daily life functioning and satisfaction. An essential part of rehabilitation includes skill development that depends on cognitive functions and learning. Thus, furthering current understanding cognitive ability as it relates to thought is especially important for strengthening treatment interventions and efficacy. Insight into areas of spared and compromised cognition and heterogeneous cognitive abilities as they relate to symptoms and thought process amongst individuals with schizophrenia may provide valuable information about pathways for effective delivery of rehabilitation techniques. Better understanding of specific cognitive domains amongst individuals with schizophrenia may inform rehabilitation interventions that are specific, tailored to an individual's abilities, and increase overall daily living skills. A study by Shanks et al. (1998) obtained findings relating various cognitive functions to distinct functional outcome domains of psychiatric rehabilitation. These researchers discussed that knowledge of the cognitive predictors of behaviors that are associated with target outcomes of rehabilitation could provide the opportunity for developing tailored cognitive and behavioral interventions for the individual. The work by Bowie and colleagues (2008, 2006) further exemplifies the need to understand how specific neuropsychological domains are impacted in this disease, so that they may be paired with functional and performance outcomes that apply to real-world settings and treatment.

Gaps in the Research

The purpose of this research was to further understand implicit learning and memory processes amongst individuals with a diagnosis of schizophrenia. Research of implicit learning and memory strategies in individuals with schizophrenia may contribute to a more informed understanding of the relationship between thought and cognition in schizophrenia. As suggested in previous studies, the need to control for other factors that may influence neurocognition or deployment of performance skills should be accounted for. Their variance must be included in findings so that treatment is targeted more skillfully (Bowie et al., 2008; Bowie et al., 2006)

In this current study, I aimed to understand implicit learning processes in individuals with schizophrenia, namely, priming, procedural, and incidental learning, in the context of both crystallized and fluid knowledge. This study was designed to investigate aspects of cognition in schizophrenia that have not been researched, namely implicit learning and thought, and to combine the study of various aspects of schizophrenia into one comprehensive study. Additionally, this study examined the potential impact of many other variables that may influence implicit learning potential in individuals with schizophrenia.

To my knowledge, no studies have specifically examined priming, procedural, and incidental learning strategies in the context of fluid and crystallized knowledge, and no previous studies have accounted for potential influence of other factors, such as symptoms severity. Thus, this study examined implicit learning in schizophrenia within the design described. My purpose was to contribute to current understanding of neurocognitive factors in schizophrenia. Future studies could examine relationships

between the specific implicit learning strategies upon which this study focused, and other functional and performance competence domains that are critical targets of treatment for schizophrenia.

Research Questions

The purpose of this study was to identify if differences exist in different implicit learning tasks for crystallized and fluid based knowledge in individuals with schizophrenia. One of the objectives was to differentiate strengths and weaknesses in implicit learning abilities in this population. Differences in crystallized and fluid knowledge aptitude was also observed. This study sought to answer four questions:

1. Will priming crystallized knowledge amongst individuals with schizophrenia facilitate improved recall?
2. Will individuals with schizophrenia perform better on a task based in crystallized or fluid knowledge?
3. Will participants perform better on an incidental learning task or a procedural learning task?
4. Will participants perform better on a priming task or on an incidental learning task?

To answer these questions, the following primary hypotheses were developed and tested using empirically validated psychological tests and procedures. The first condition tested performance by schizophrenic participants in a primed compared to non-primed learning condition in a crystallized knowledge based task. To test this hypothesis participants were administered two verbal production category tests that were either primed or not primed. It was hypothesized that participants would produce more verbal

responses in the primed condition than the non-primed condition. The second condition tested was differences in participant performance in a task of crystallized knowledge compared to fluid knowledge. It was hypothesized that participants would perform better on tasks based in crystallized knowledge than tasks based in fluid knowledge. To test this, participants performed two distinct incidental learning tasks, one based in crystallized knowledge and one based in fluid knowledge. A third condition that was tested compared participant performance between an incidental and procedural learning task to test the hypothesis that participants' abilities related to incidental learning would be better than the procedural learning condition. To test this, participants were administered two different tests based in a fluid knowledge: an incidental task and a procedural task. The last condition that was tested compared participant performance between tasks learned through priming compared to incidental learning methods in crystallized knowledge based tasks. It was hypothesized that participants would perform better in the priming than the incidental learning condition.

Chapter III

Research Design and Methodology

Design

This quantitative cross-sectional relationship study of implicit learning in schizophrenia was designed to obtain findings to contribute to the current literature on the neuropsychology and cognition in schizophrenia. Discussed extensively in the literature review, cognition is directly related to rehabilitation and functional outcomes. Thus, complete understanding of underlying cognition and thought in schizophrenia contributes to functional outcomes amongst individuals with schizophrenia. The cognitive domain that was investigated and measured in this study was implicit learning. Specifically, abilities in three types of implicit learning were studied: priming, procedural, and incidental learning. Additionally, these learning types were studied in the context of two types of knowledge: crystallized knowledge and fluid knowledge. Prior to data collection, demographic characteristics were collected, including psychosis symptoms and symptom severity (measured by BPRS-E), IQ (measured by Wide Range Achievement Test, 4th edition [WRAT-4] Reading Test), years of education (reported in the demographic questionnaire), age (reported in the demographic questionnaire), and gender (reported in the demographic questionnaire). This information and variations were considered when converting raw data to T-scores.

Measures of a participant's ability to apply certain implicit learning strategies were compared by contrasting summed and averaged raw scores that were converted to age-corrected T-Scores for all the implicit learning conditions: the priming condition, the procedural condition, and the incidental learning condition. Please refer to the "Data

Collection Methods” section that follows for further details on test battery and data collection methods.

Research Variables

A paired *t* test (paired-samples *t* test or dependent *t* test) was used for analyzing all research variables to test the hypotheses. The first hypothesis stated that priming crystallized knowledge would improve the recall among individuals with schizophrenia. This hypothesis was tested using the mean word production as measured by the Controlled Oral Word Association Test (COWAT; converted to a T-score) as the dependent variable (continuous) measured in the two conditions prime and no-prime (categorical).

The second hypothesis stated that the performance of patients with schizophrenia on tasks based in crystallized knowledge would be better than tasks based in fluid knowledge. The test for the second hypothesis included one dependent variable item production on either the Boston Naming Test – 15-Item-Incidental Trial (BNT-15-I) or the Digit-Symbol-Coding test converted to a T-score. The two related but different conditions were crystallized and fluid learning. Testing this hypothesis was further broken down into two analyses; in the first item, production and recall was elicited through pairing during retrieval of information on the Digit-Symbol-Coding test and in the second through free-recall.

The third hypothesis stated that participant performance would be better in the incidental learning than procedural learning condition. The test included one dependent variable, T-scores scores on the Digit-Symbol-Coding Incidental (Paired) Test Finger Tapping Test. The independent variables were incidental learning and procedural

learning. As in testing Hypothesis 2, two separate analysis were also run for Hypothesis 3. The first performance on the Finger Tapping Test (Paired) for the participant's dominant hand was compared to performance on the Digit-Symbol-Coding Incidental Test, and in the later performance with non-dominant hand was conducted. The two categories were incidental condition and procedural condition.

The fourth hypothesis stated that participants would perform better in the priming than the incidental learning condition. The test for this included the mean word production as (converted to a T-score) as measured by either the COWAT categories test or the BNT-15-I test (both continuous) and the two conditions were priming and incidental, both conditions were based in crystallized knowledge.

Additional variables measured in this study included reading level, symptomology, and symptom severity (for diagnostic purposes), age, gender, and years of education. Some of these variables were used during initial screening for determining inclusion and exclusion criteria of the potential participant, others were used for calculating norm adjusted T-scores for analysis where norms were available.

Participant Recruitment

The participant sample used in this study was obtained from various clinics including County of Orange Behavioral Health-Adult Mental Health Services, Clubhouses, Orange County Wellness Centers, and through referral from community clinicians through the Orange County Psychological Association Listserv. IRB approval was obtained from the IRB at Antioch University and from the IRB at County of Orange Health Care Agency Human Subjects Review Committee. Following initial contact, I arranged a meeting time and location with a potential participant. Participants met me in

a private room at either the primary clinic where they received services, in their day program, or at the education center located adjacent to the day program.

On the assigned testing date and time, prior to administering the test battery, participants were informed of the intended procedures and were provided with an informed consent form. Participants were encouraged to ask any question they had about the informed consent and the study, bring up any concerns they may have about the study procedures, and were provided the option to decline participation in the study. The informed consent was explained to the participants, and each participant was asked to sign the informed consent/assent form. These forms are in Appendices A, B, and C. If the participant was on conservatorship, they were informed that consent would need to be obtained from their conservator first, and this was done prior to proceeding with their participation in the study (Appendix B). The participants were reminded that participation was voluntary and would have no effect on treatment services that the participant was receiving from their clinic at that time.

Instrumentation and Measures

Table 1 presents a list of some of the tests that were used to collect data on the independent and dependent variables. These instruments and how they were used to test the hypotheses is discussed in further detail in this chapter.

Table 1

Neuropsychological Test Battery and Variables

Construct Knowledge	Dependent Variable Test Measure
Word Reading	Wechsler Test of Adult Reading
Processing speed	Trails A & B
Crystallized knowledge	Controlled Oral Word Association Test (Categories: animals/clothing)
Crystallized knowledge	Controlled Oral Word Association Test (Categories: animals/clothing)
Crystallized knowledge	Boston Naming Test (BNT; 15-Item-Incidental Trial)
Fluid knowledge	Digit-Symbol-Coding Incidental Trial
Fluid knowledge	Finger Taping Test

Cognitive domains: MATRICS Consensus Cognitive Battery. The NIMH developed a consensus battery to carry out clinical trials. The primary intended purpose of the consensus cognitive battery was to be for assessing cognitive-enhancing drugs as well as developing interventions for addressing cognitive symptoms in schizophrenia. A final cognitive battery was determined by a panel of experts, and included ten neuropsychological tests to measure seven cognitive domains. The seven cognitive domains included in the MCCB are “speed of processing, attention/vigilance, working memory, verbal learning, reasoning and problem solving, and social cognition” (Nuechterlein et al., 2008, p. 203). The domains of cognition that were selected to be measured in this study included some of the domains included in the MCCB as well as some additional domains not included in the MCCB.

The cognitive domains included in this research deviated from those proposed in the MCCB because the primary focus of this study was to investigate implicit learning abilities in schizophrenia. The implicit learning strategies that were the focus of this study included priming, procedural learning, and incidental learning. Additionally, subject learning when using these strategies for crystallized and fluid knowledge was differentiated. The priming variable had two levels, prime and non-prime. Some of the current literature that exists on implicit learning abilities in the schizophrenia population was briefly described in the literature review, as well as what the research has shown about these individual implicit learning domains in the population.

Psychopathology: Presence of positive and negative symptoms. The original Brief Psychiatric Rating Scale (BPRS), introduced by Overall and Gorham (1962), is a test made up of 18 items that measures the presence and the severity of both positive and negative symptoms. The BPRS is used to measure and classify psychopathology, and has been used extensively in research for measuring therapeutic change and classifying patients into subgroups based on pathology and symptom severity. In this study, the BPRS-Extended (BPRS-E; Lukoff, Nuechterlein, & Ventura, 1986) version was used. The BPRS-E includes 24 items that are similar to the original 18-item BPRS as well as six additional items in the following categories: “bizarre behavior, suicidality, self-neglect, motor hyperactivity, distractibility, and elevated mood” (Dingemans, Liszen, Lenior, & Smeets, 1995, p. 265).

Ventura, Green, Shanner, and Liberman (1993) discussed the “quality assurance program” that was developed by the University of California, Los Angeles for increasing inter-rater reliability. They identified the four main changes made to the BPRS

administration, including a manual, structured interview questions, rater training, and quality assurance. As a result of the changes, high inter-rater reliabilities were obtained. In their appendices, Ventura et al. (1993) included the manual and administration guidelines for administering the BPRS-E. To increase reliability of the use of the BPRS in this study, the structured questions developed for the BPRS-E manual, as well as the anchor points for the 24 items were used in this study. Based on the scoring guidelines, the participants received a score between one and seven on each of the items, according to the presence and the severity of the symptom. Total scores for each individual participant were calculated, and total scores ranged between 24 and 168. The BPRS-E was administered immediately after the neuropsychological tests.

Hedlund and Vieweg (1980) indicated that the original BPRS was used extensively for the purpose of assessing patient psychopathology to develop predictions about treatment expectations of distinct treatment modalities. They indicated that inter-rater agreement was the best approach for obtaining reliability measures for the BPRS and showed that 13 studies reported inter-rater reliabilities (Hedlund & Vieweg, 1980). Of the thirteen studies, ten of the studies reported reliability coefficients of $r = .80$ or higher. Additionally, two of the three studies that reported reliability coefficients below $.80$ used populations with specific restrictions and variable forms of the test. They stated that one of these studies included performance of non-psychotic, alcoholic patients on an extended form of the BPRS made up of 21-items. The second study included a homogenous population of individuals with schizoaffective disorder. Based on pre-screening findings, participants that presented with more manic or schizophrenic symptoms were excluded. These authors reported validity coefficients between the BPRS

and the Multidimensional Scale for Rating Psychiatric Patients (MSRPP) of .93 for the purpose of measuring treatment change, and moderate correlations of .58 to .61 between scores on the anxiety, depressed mood, and motor retardation items on the BPRS with specific MMPI composite scores on BPRS anxiety, depressed mood, and motor retardation items (Hedlund & Vieweg, 1980). They also reported validity coefficients of .79 and .73 between the activation and hostility-suspicious constructs of the BPRS, respectively, and the Clinical Global Impressions Scale (CGI; Hedlund & Vieweg, 1980). Ventura et al. (1993) reported that previous research with the BPRS on normal controls has demonstrated that these individuals scored two points or lower on the items that measure psychosis on the BPRS (p. 230).

For the BPRS-E, Lukoff et al. (1986) indicated that as a result of the added six items, the test became more inclusive for a broader range of patients, specifically those who are not hospitalized as opposed to the original BPRS; thus, it was a good fit for the population in this study (Ventura et al., 1993). For this study, the BPRS-E was a much more appropriate test to assess symptom severity. Dingemans et al. (1995) conducted an analysis of the BPRS-E. Four out of the five components were identified in the factor and component analysis; however, only four of these five component solutions showed internal consistencies in the acceptable and good range: “positive symptoms ($\alpha=.74$), depression ($\alpha=.75$), negative symptoms ($\alpha=.76$), mania ($\alpha=.64$)” (Dingemans et al., 1995, p. 265). Participants were given a total score between 24 and 168 per the scoring criteria based on the anchor points provided in the BPRS-E manual (Ventura et al., 1993). Scores and BPRS-E performance were used to address symptom severity and for diagnostic clarification.

Declarative learning and memory: Crystallized. For the purpose of this investigation, the Wide Range Achievement Test – Fourth Edition (WRAT-4; Wilkinson & Robertson, 2006) was used to measure grade level efficiency and results were used to identify that participants had the minimum 8th grade reading level required to participate in the study. In their discussion, Marcopulos and Fujii (as cited in Marcopulos & Kurtz, 2012) identified some key studies, both challenging and supporting the use of premorbid IQ tests in populations presenting with a developmental disorder. Some of the specific authors identified in their discussion included Dennis et al. (2009) and Ravheim et al. (2006). The WRAT first came into use in the 1930s, developed by Dr. Joseph F. Jastak for assessing academic codes in addition to cognitive processes being measured by other tests. The Wide Range Achievement Test, Third Edition (WRAT-3) was published in 1993. Minimal changes were made to the word reading subtests between the WRAT-3 and WRAT-4: three items were eliminated and 29 items were added, resulting in 55 items instead of 44 items on the subtest. By age, median alpha subtest reliability coefficients ranged between .87 and .93. External evidence of validities for the word reading subtests were .77 with reading on the WRAT-E, .80 with the Wechsler Individual Achievement Test – Second Edition (WIAT-II) reading comprehension, and .70 with broad reading on the Woodcock-Johnson – Third Edition (WJ III). Test-retest reliability for the word reading was found to be .86. Reading recognition ability has remained stable in the context of cognitive decline in normal aging and brain disturbance, and the test has been normed according to age and education grade level (Marcopulos & Kurtz, 2012). Participants in the current study received a total score out of a potential maximum of 70, which was then converted to a T-score which was used to assess reading level.

Non-declarative crystallized learning and memory: Priming strategy. The COWAT (Benton, Hamsher, & Sivan, 1994) is a test of verbal fluency made up of phonemic, semantic, and written fluency tasks. Priming for crystallized knowledge was measured with the COWAT, which is one of the independent variables. Additionally, three self-created paragraphs, two category priming paragraphs and one neutral category paragraph, were included. As described in the literature review, priming, specifically repetition priming, results in stimulus response that is facilitated by previous exposure to the stimulus or a similar stimulus, otherwise known as the priming stimulus (Goldstein, 2005). Strauss, Sherman, and Spreen (2006) reported that the test-retest reliability coefficients for semantic fluency tests have been determined to be in the range of .70 and above as determined by various authors, including “Basso et al., 1999, Dikmen et al., 1999, Harrion et al., 2000, Levine et al., 2004, Ross, 2003” (p. 514). Correlations between distinct categories on the semantic category fluency tests were determined to be in .66-.77 range, indicating moderate to high inter-category validity. Moderate correlations have been identified between semantic fluency tests and the BNT in the range of .57-.68, which Strauss et al. (2006) concluded is substantially indicative of semantic memory (crystallized knowledge).

In this study, the priming stimulus (either the animal category or clothing category paragraph) was presented in the prime/no-prime condition, this consisted of a short paragraph presented to participants. These paragraphs used were self-constructed and can be reviewed in Appendix D - Data Collection Forms. A total of three paragraphs were constructed, two priming paragraphs, one for animals and one for clothing items, and a neutral paragraph (based on furniture) that were read to the participant. There were

two priming paragraphs, the first paragraph described contextual information relating to animals, but did not actually name any animals, and was read to half of the participants. The second half of participants were read a paragraph that described clothing contexts, without naming any articles of clothing. This condition, prime/no-prime compared participants' abilities to name either animals or clothing items as a measure indicating whether verbal production of this crystallized knowledge base had been facilitated by the priming paragraph for each of the two categories.

Participants in each of the two groups completed two trials of verbal production (one for animals and one for clothing). Each participant had to produce words from the category of the paragraph they received, the priming paragraph, (animals or clothing, depending on their group number) and items from the opposite group category for which they were not read the associated priming category paragraph, but instead the neutral (furniture) unrelated paragraph. The neutral paragraph to be read in each group was identical. Each of the two groups were further split, half of the participants in each group were read the priming paragraph and asked to recall words for that category first, then the neutral paragraph and asked to recall words from the non-associated paragraph. The other half of each group was presented the neutral condition first followed by the priming stimulus second. This was done to counterbalance test order effects. Thus, participants were placed in four separate groups randomly, labeled Group 1 and Group 2, who were read the animal category priming paragraph and neutral furniture paragraphs, and Group 3 and Group 4, who were read the clothing category priming paragraph and neutral furniture paragraph.

The participants obtained a total word score for words produced on the COWAT in the prime condition, and a total word score in the no-prime condition. Each of these raw scores were converted to T-score, which was representative of how many standard deviations the score fell from the mean. The mean and standard deviation were estimated from a population mean taken from previous studies with larger samples. The T-scores for each participant on each of the conditions were then compared utilizing a paired *t* test in the data analysis stage.

Non-declarative fluid learning and memory: Procedural strategy. The Finger-Tapping Test (FTT; Reitan, 1996) was one of the tests introduced and included in the test battery developed by Halstead in 1947. The FTT is used as a part of neuropsychological evaluations for the purpose of measuring an individual's motor and cognitive functions. In this study, the FTT was used to measure procedural learning in the fluid procedural learning condition. Procedural learning and procedural memory are often used interchangeably. Procedural learning describes learning of a skill that can be cognitive, a motor skill, or a skill set, described previously in the literature review (APA, 2013). In a study by Da Silva et al. (2012), the FTT was used to measure procedural learning. These authors indicated that use of this test for measuring procedural learning was vindicated due to procedural learning being defined as “implicitly learning a motor or cognitive procedure by repetition, up to the point of automation” (Da Silva et al., 2012, p. 235). In their study, Da Silva et al. (2012) defined procedural learning as the learning slope across ten trials in which the number of taps on the trial was the outcome.

Participants were administered five to ten consecutive trials on each hand until five scores for each hand were collected with each hand that lie within five taps of each

other, first with the dominant hand and then the other hand per standardized instructions developed by Reitan and Wolfson (1985). The five scores were then averaged and converted to demographically adjusted scaled scores utilizing the revised comprehensive norms for an expanded Halstead-Reitan Battery (Heaton, Taylor, & Manly, 2003). Scaled scores were then converted to T-scores.

Non-declarative crystallized learning and memory: Incidental strategy. The BNT-15-I (Kaplan, Goodglass, & Weintraub, 1983) was used to measure implicit learning in the crystallized knowledge incidental learning condition. Doeller and Burgess (2008) described incidental learning as a form of implicit learning which takes place outside the awareness of the individual resulting from their encounter with environmental stimuli. Implicit learning was a third independent variable of interest in this study. Lezak, Howieson, and Loring (2004) described the mechanism for measuring incidental learning observing learning as it occurs in the learners naturally. In this study, the digit-symbol coding test from the WAIS-III included an incidental trial that was used to measure incidental learning for fluid knowledge.

A study by Bryan and Luszcz (2000) used a 15-item version of the Boston Naming Test to measure incidental memory, as well as a digit-symbol-coding incidental trial. For the Boston Naming Trial, the participants received 15 individual items from the test. Participants were asked to name each of the 15 items, and semantic or phonemic cues were provided to the participant if they were unable to name the object in the picture. Bryan and Luszcz (2000) also indicated that examiners provided participants with the name of the object if they were still unable to name the picture after the cues were provided, thus ensuring that the participant was exposed to the word. Following this

initial phase, participants were asked to produce as many of the picture words as they could remember, thus obtaining a measure for incidental learning. These authors also indicated that the use of cues, and providing names by the examiner, may change the total words recalled by the participant; thus, this was factored in as a covariate (Bryan & Luszcz, 2000).

The two short, 15-item forms of the BNT that exist are the Mack SF4 and the CERAD, which have been determined to have test-retest reliability correlations in the .49-.84 and .36-.83 ranges, respectively. As indicated previously, this test has been determined to demonstrate inter-test validity with the COWAT in the range of .57-.68 (Strauss et al., 2006). High inter-test validity has also been observed between the Visual Naming Test of the Multilingual Aphasia Examination, with correlations in the range of .76-.86, also described by Straus et al. (2006) as a language test. Mack, Freed, Williams, and Henderson (1992) developed four shortened versions of the original BNT 15-item version which showed significant correlations with each other, as well as with the complete BNT. Correlations between the four new versions were in the range of .79 to .98, and correlations between the four new versions and the complete (60-item) BNT were in the range of .97 to .98. These authors also discovered in their comparative analysis that the items included in the CERAD version appeared to be less challenging than the four 15-item versions they developed (Mack et al., 1992).

This study used the 15-item version of the BNT developed by Mack et al. (1992). Each correctly recalled item out of 15 previously shown picture items was recorded and totaled. The total score was then converted to a T-score representative of how many standard deviations the score fell from the mean, which was estimated from a population

mean taken from previous studies with larger samples.

Non-declarative fluid learning and memory: Incidental strategy. For this investigation, the WAIS-III Digit Symbol-Coding subtest (Wechsler, 1997) was administered as one of the tests in the battery to measure participant implicit learning and memory abilities in the fluid incidental learning condition. Incidental learning was one of the independent variables of interest in this study. This WAIS-III subtest includes a procedure for assessment specifically for measuring incidental learning. Lezak et al. (2004) briefly described the procedures used to develop this technique. To summarize, the Digit Symbol-Coding subtest was administered as directed in the WAIS-III Administration and Scoring Manual. Following this, the participant was provided a set of numbers that were paired with a design. The participant must later recall the design that pairs with the number stimulus. Following the recall pairing activity, the participant was required to depict through pairing and free-recall, on a separate piece of paper, as many symbols as possible (Lezak et al., 2004; Strauss et al., 2006). Individuals were given a total correct score determined by their responses on the test. Participants received two scores on this test, a score for pairing a number with a symbol (P), and a score for free recall (FR).

Reliability of the Digit Symbol-Coding subtest was calculated using the test-retest method. Reliability coefficients for individuals between the ages of 16 to 89 were obtained, and were in the range of .81 to .87. Intercorrelation validity values for the Digit Symbol-Coding subtest for all age groups were averaged, and were in the range of .33 to .65. Correlations of the Digit Symbol-Coding subtest with the Wechsler Intelligence Scale for Children, Third Edition (WISC-III) was determined at .77, as inferred from the

performance of a sample consisting of 184 subjects who were 16 years old. Incidental verbal learning, an implicit learning construct, was measured with the incidental trial.

The WAIS-III subtest included procedure instructions, and normative data for assessment specifically for measuring incidental learning in individuals between the ages of 16 and 89. There are two incidental measures for this test: pairing and free recall. Participants completed the Digit-Symbol Coding trial which was then followed by the Digit Symbol-Incidental Learning Pairing trial and then the Free-Recall trial (per standardized instructions). A raw score of up to 18 points was recorded for the Paired trial and up to 8 points for the Free-Recall trial. Each participant's raw scores on each of these two conditions was converted to a T-Score that was utilized later for data analysis and comparison with the other learning conditions.

Procedures

During my initial contact with the potential participants, I provided participants with information about the research study. Participants were informed of the background and purpose of the research, what was expected of them should they choose to participate, including the basic structure of the protocol, and the short and long term benefits of participation. To ensure a coercion-free environment, the voluntary nature of the study was explained. Participants were informed that their decision to participate in the study would have no impact on the current or future mental health services they may receive. Participants had to meet the inclusionary and exclusionary criteria specified in the following paragraph to be considered for participation in the study.

All the potential participants had to have received a prior diagnosis of schizophrenia as specified in the DSM-IV-TR or DSM-V criteria for schizophrenia by

either a psychiatrist or a licensed psychologist. When possible this was verified with the clinic where they received services. Also, to meet inclusion criteria, participants had to have a minimum of an 8th grade education, and ability to read at an 8th grade level to complete the tests included in the battery. This was assessed through self-report by the participant on the demographic questionnaire, participants also completed the Wide Range Achievement Test –Fourth Edition (WRAT-4) to account for this potential covariate and assess for a minimum of an 8th grade reading level (part of inclusion criteria). Individuals with a history of brain injury, or any other medical disorder or neurological condition affecting cognitive abilities were excluded from participating in the study. Also, to be included in this study, participants' primary language had to be English, all others were excluded from participating. Medication information was also collected. All this information was collected through a demographic questionnaire; this information is specified in detail in Appendix E – Participant Recruitment.

Once the potential participant expressed an interest to participate in the study, I obtained informed consent or assent from the participant, depending on their conservatorship status. Included in the informed consent/assent forms, located in Appendices A, B, and C is the participant's consent to participate in the study. In cases in which the participant had a conservator, informed consent was obtained from the conservator and informed assent from the participant (Appendices B and C, respectively). Following this initial agreement, I set up a date and time with the participant to meet for the study trial, which took place in a reserved room at either the participant's regular clinic or education center located near the wellness center in Orange, CA. Participants received bus passes as needed for any transportation required to and from the trial on

their scheduled testing date.

On the date of the trial, I administered the sociodemographic questionnaire and WRAT-4-Word Reading, and if the participant met the inclusion criteria, I proceeded with administering the test battery. Participants who met one or more elements of the exclusionary criteria were thanked and explained that they did not meet criteria to be included in the study. Participants who met all the inclusion criteria were administered the complete trial, and were rewarded with a \$20 health and wellness Walmart gift card at the completion of all or most of the subtests. Participants with a conservator had their gift card mailed directly to their conservator.

Data Collection Methods

Data was collected at the time of test battery administration. Informed consent was recorded on the informed consent form. Informed consent forms were separated from the assessment data collected, and were stored in a separate lock box. Sociodemographic data provided by participants was recorded on the sociodemographic questionnaire, and responses provided by participants on the neurobehavioral test battery were recorded on designated response forms for the individual tests. To protect participant privacy, each participant was assigned an individual code number. All informed consent and data collection forms were stored by the researcher in a safe location, in separate lock boxes, per confidential participant data storage guidelines. The lock boxes were stored in a locked room.

The sociodemographic questionnaire was administered to participants first, followed by the neuropsychological tests. The BPRS was used to measure participant symptom severity, and assess current symptom presentation and diagnosis, and the

WRAT-4 was used to measure participant pre-morbid IQ. The tests were administered in the following order: (a) the COWAT (Phase I), (b) the FTT, (c) the COWAT (Phase II), (d) the Digit-Symbol-Coding incidental trial, (e) the BNT-15, (f) Trails A & B, (g) the WRAT-4 Word Reading, and (h) the BPRS. All tests were administered following the administration guidelines in the manual for each of these tests. I applied test administration and data collection techniques in accordance with the standardized and normed rules for each individual test being administered.

Prior to administering the tests, participants were assigned to one of four groups through random assignment. Each participant was administered the test protocol corresponding to the group to which they were assigned. Table 2 presents the order of test administration for each of these groups. Test procedures were designed to minimize test interference, and account for potential test order effects. For example, the FTT was administered between Phase I and Phase II of the COWAT to minimize interference. The first test to be administered was the COWAT (Phase I). Participants in Group 1 were administered the Animals Prime, followed by the Animal Fluency Task. Group 2 were administered the Neutral Prime followed by the Furniture Fluency Task. Group 3 were administered the Furniture Prime followed by the Furniture Fluency Task. Group 4 were administered the Neutral Prime followed by the Animal Fluency Task. The purpose of this order was to determine if a priming effect had occurred, and to counterbalance test order administration of the COWAT and eliminate any test order variability.

Table 2

Test Order Administration for Groups 1-4

Order of Administration	Group 1	Group 2	Group 3	Group 4
WRAT-4	WRAT-4	WRAT-4	WRAT-4	WRAT-4
COWAT (Phase I)	Animals Prime Condition	Neutral Prime Condition	Furniture Prime Condition	Neutral Prime Condition
	Animals Fluency Test	Furniture Fluency Test	Furniture Fluency Test	Animals Fluency Test
Finger Taping Test (FTT)				
COWAT (Phase II)	Neutral Prime	Animal Prime	Neutral Prime	Furniture Prime
	Furniture Fluency Test	Animals Fluency Test	Animals Fluency Test	Furniture Fluency Test
Digit-Symbol Coding (WAIS-III)				
BNT-15-I	BNT-15-I	BNT-15-I	BNT-15-I	BNT-15-I
BPRS-E	BPRS-E	BPRS-E	BPRS-E	BPRS-E

In Phase II of the COWAT, the remaining tests were administered. After the COWAT Phase I, the FTT was administered to all participants. Participants were administered five to ten consecutive trials on each hand until five scores for each hand were collected with each hand that lie within five taps of each other, first with the dominant and then the non-dominant per standardized instructions developed by Reitan and Wolfson (1985). Participants were given a practice trial first, and each test trial lasted ten seconds. Total taps were recorded by the examiner, and the five scores were

converted to T-scores and included in SPSS analysis.

Following administration of the FTT, participants were administered Phase II of the COWAT. Group 1 was administered the Neutral Prime followed by the Furniture Fluency Task. Group 2 was administered the Animal Prime followed by the Animal Fluency Task. Group 3 was administered the Neutral Prime followed by the Animal Fluency Task. Group 4 was administered the Furniture Prime followed by the Furniture Fluency Task. The number of words produced by the participant was recorded for each participant, converted into T-scores, and entered into SPSS for analyses.

Digit-Symbol Coding was administered to all participant groups after administration of the COWAT Phase II. For this test, participants were asked to pair numbers with specific marks according to a reference key. Instructions for this test were administered following the test administration guidelines in the WAIS-III Administration and Scoring Guidelines (Wechsler, 1997). Participants completed the initial encoding portion of this task for 120 seconds, at which point the examiner stopped the participant. The examiner then administered the two tasks of the incidental learning trial of the Digit-Symbol Test. For the first task, pairing, participants were provided a sheet of paper with two rows containing nine numbers each but not marks. Participants received one point for every correct pairing and received a score out of 18 points (maximum number of points possible). Participants were asked to pair the marks with each of each of their corresponding numbers that they encoded during the initial encoding trial. In the second retrieval task of the incidental learning task participants were provided with a blank sheet and asked to produce as many marks as possible from memory (without the cued/paired number). A maximum possible score for this task was nine points, one point per correct

recall.

The Boston Naming 15-item Incidental Test was administered next. Participants were administered the 15-item Version 1 (Mack et al., 1992). The examiner administered this test per the standardized original BNT-2 instructions, and essentially replicated the administration format used by Bryan and Luszcz (2000), described previously in “Instrumentation and Measures” section of this Chapter. Following the initial presentation of the pictures during the encoding phase, the participant was asked to produce as many picture-words as the participant could recall. Participants received one point for each correctly recalled picture-word. Picture-word scores were recorded for each participant and converted into a T-scores based on the BNT-15-I norms by Mack et al. (1992) and included in the final SPSS analysis.

The next test to be administered is the WRAT-4 word reading test. The WRAT-4 word reading test was administered and scored according to the WRAT-4 Manual (Wilkinson & Robertson, 2006)). This test was administered in accordance with the administration instructions provided in the manual. Participants were provided with the word reading form (from the blue test form set) and asked to read the words. Participant responses and totals were scored according to the instructions set forth in the manual.

The final administration was the BPRS-E. The BPRS-E was administered and scored using the structured question and the scoring anchor points in the BPRS-E manual (Ventura et al., 1993). At the conclusion of the BPRS-E administration, each raw score was entered into SPSS analyses as a covariate.

At the conclusion of the testing, the forms were transported in a locked box to the secured data storage location indicated previously. Individual participant informed

consent forms were stored separately from the code numbered response forms assigned to each individual participant. Data was transferred into encrypted excel documents before being run through the statistical analysis. For the demographic questionnaire, participants were asked questions, and then recorded the responses, which were transferred into Excel documents and applied to calculating appropriate norms.

All data was screened and prepared before conducting data analysis to ensure that the data collected was accurate, to address any issues with missing data, identify any extreme values, otherwise known as outliers, to prevent misrepresentation of final statistical results, and to ensure all T-scores were correctly calculated (Mertler & Varnatta, 2010). These assumptions are discussed further under the section of methodology limitations and assumptions. Mahalanobis distance was used to identify potential outliers, and was estimated with a chi-square statistic.

The test administration sequence was developed to minimize inter-test interference. Tests were arranged in a format so that no individual test interfered with the participant's performance or scores on subsequent tests. For example, the COWAT verbal test was followed by the FTT non-verbal test. Additionally, the orders of test administration were varied between the four participant groups to counterbalance administration effects and avoid potential systematic variation effects caused by test order administration (Field, 2009). The test sequence was also developed to consider the administration time of individual tests, and time required between separate trials of individual tests. At the conclusion of the protocol administration, participants were thanked and awarded with a \$20 health and wellness Walmart gift card. The card was awarded to the participant directly or mailed to the participant's conservator if the

participant was conservatorship.

Data Processing Techniques

Participant responses and data obtained during the test battery administration were transferred from data collection forms into an Excel data form. A participant code number was recorded along with all the data collected for each dependent measure on one row in separate columns. Once all the data was collected, each analysis was run to test each one of the four individual hypotheses. Prior to independent analyses, data was screened with SPSS frequencies for assessing the frequency distributions and accuracy of data. As indicated previously, the first step in the analysis was screening data to ensure that the data collected was accurate, to address any issues with missing data, identify any extreme values, otherwise known as outliers, to prevent misrepresentation of final statistical results (Mertler & Varnatta, 2010). These assumptions are discussed further under the section of methodology limitations and assumptions. Mahalanobis distance was used for identifying potential outliers, and was estimated with a chi-square statistic.

To test the first hypothesis, priming crystallized knowledge would improve recall among schizophrenics in the crystallized knowledge condition, a paired *t* test was run comparing data COWAT primed condition data to the COWAT non-primed condition data for each participant. The independent variable was entered as a categorical variable, with two categories, the prime and no-prime condition. There were two dependent variables, the total word score obtained in the primed trial and the total word score obtained on the COWAT in the no-prime condition. One score in the prime condition and one score in the no-prime condition was entered for each participant, and each raw score was converted to T-scores and compared utilizing a paired sample *t* test.

To test the second hypothesis, participant performance on a task based in crystallized knowledge would be better than performance based in fluid knowledge in an incidental learning condition (the constant) in schizophrenic participants, two separate paired *t* test analysis were conducted. Both analyses included one categorical independent variable with two categories, crystallized knowledge and fluid knowledge. The first analysis included two continuous dependent variables, participant T-scores on the BNT-15-I and participant T-scores on the Digit-Symbol Search Incidental Task pairing trial. In the second analysis, the two continuous dependent variables that were entered into the analysis for comparison were participant T-scores on the BNT-15-I and participant T-scores on the Digit-Symbol Search Incidental Task free-recall trial. T-scores were obtained for each as described previously under description of measures. T-scores for each condition were then entered into SPSS and compared utilizing a paired sample *t* test.

To test the third hypothesis, that participants would perform better on the incidental learning than on the procedural condition, a paired *t* test analysis was used. There was one categorical independent variable with two categories, incidental learning and procedural learning. Two continuous dependent variables that were compared were individual participant T-scores on the Digit-Symbol Search Incidental Task pairing trial and participant T-scores on the FTT. Participant T-scores in each of these conditions were compared in SPSS utilizing a paired samples *t* test.

The final hypotheses that was tested, applied a paired *t* test analysis to determine if participants performed better in a primed learning condition than an incidental learning condition. In this analysis, one categorical independent variable (learning condition) was analyzed, the two categories were primed compared to incidental. The two continuous

dependent variables that were compared were scores obtained by participants on the COWAT-prime learning condition converted to T-scores and T-scores obtained for each participant on the BNT-15-I trial. These scores were compared using a sample paired t test.

Methodological Assumptions and Limitations

Mertler and Varnatta (2010) indicated four primary methodological assumptions in using a paired sample t test in statistics. The first of these assumptions is that the observations that are made for each sample in the study are random and are not associated with each other, hence, independent. The second assumption is approximate normal distribution of the dependent variable, and the third assumption presumes that the dependent variable does not contain outliers. The fourth assumption made in this type of analysis is that the dependent variable is a continuous variable.

A limitation to this study was with the ability to generalize these findings to all individuals with schizophrenia. The individuals included in this study were outpatients from various mental health care programs, thus the findings from this study may not generalize to patients who do not live in the community and are instead institutionalized.

Some possible confounds, defined as “a variable that is conceptually distinct but empirically inseparable from one or more other variables” (Van den Bos & American Psychological Association, 2013, p. 132) that were screened for during the interview and by participant responses on the sociodemographic questionnaire, were medication type, level of education and reading level (assessed using the WRAT-4), symptom severity (assessed with the BPRS), and a history of head injury. To address and minimize these confounds, participants provided responses on the sociodemographic questionnaire

(Appendix F, Form A) and these were factored out. Some of the confounds, such as education level, age, and gender, could be accounted for by obtaining standardized scores from empirically derived norms. This was only possible for data for which norms existed.

Ethical Assurances

Participants were informed that the services provided by the county to them as a result of their being county clients were completely separate from the activities of this investigation. To avoid coercion, participants were informed that participation in this study would not affect the services they currently were receiving at the County of Orange, or their eligibility for future services. Participants were informed at the beginning of the meeting that their choice to participate in this study was completely voluntary. Participants were informed that they would receive \$20 Walmart gift card in exchange for their participation. To further safeguard participant vulnerability, they were informed that each participant's decision to participate, not participate, or drop out of the trial would remain confidential. Furthermore, only I had knowledge of the identity of the participant. The research committee shared information about any participant who may not have completed the full test protocol; however, they only had access to the numerical identification code previously assigned to the participant.

Prior to commencing the trial, informed consent was requested from participants, and participants were further reminded that their participation was voluntary. Due to the vulnerable nature of this population, for participants who were on conservatorship or had a guardian, informed consent was requested from their conservator or guardian and informed assent was explained and requested from the participant at the beginning of the testing session.

Each participant's data was collected in a test response packet made up of the individual answer and response forms. In order to safeguard confidentiality of the participant's identifiable information, and their anonymity, participants were assigned a code number at the beginning of the testing meeting. This code number was used collectively on the sociodemographic questionnaire, and for identifying all test forms and results for that participant. The code number was placed in the upper right corner of each response form. At the end of each session I transported the test response packet containing the informed consent, sociodemographic questionnaire, and the test battery response forms in a locked box to a second locked box for permanent storage. The informed consent forms were removed and stored in a locked box in a separate file from the sociodemographic and test battery response forms. All forms and testing materials will be stored in a locked box for five years. At the conclusion of five years, the paper copies will be shredded and destroyed.

At the conclusion of each testing session, data was transferred into electronic form. This information, and responses to the sociodemographic questionnaire was stored on a flash drive. The uploaded demographic material and testing data were contained in separate folders on the flash drive. Each individual data set and information were identifiable only through assigned coded numbers. The flash drive was stored in the locked box that also contained the sociodemographic and test battery response forms. The electronic data developed during the process of data integration was stored in encrypted documents. These documents were password protected, and stored on the flash drive protected with a password. Only I (not the committee) have access to the passwords required to access the flash drive and the confidential data files. All material contained in

these locked boxes were kept locked and stored throughout the duration of the study, and will be for an additional five years. Furthermore, the files and information on the two flash drives, used during data collection and storage, will be deleted five years after conclusion of this study.

Chapter IV: Results

Data collected from the study was analyzed using the SPSS. The level of significance adopted for all the statistical comparisons reported was set at $p < .05$. Within group differences (of the sample of schizophrenia participants) of the obtained participant scores for each of the implicit learning conditions were performed using a series of paired t tests. To examine whether significant differences existed between performance of crystallized and fluid knowledge in differing incidental learning conditions in individuals with schizophrenia, subsequent analyses utilized a series of paired t tests to compare implicit learning abilities among a sample of individuals with schizophrenia. Priming, procedural, incidental implicit learning abilities were compared – as individual independent variables.

Study Sample Selection and Characteristics

Twenty-five outpatient participants with a diagnosis of schizophrenia per previous diagnosis by a psychiatrist or psychologist per the DSM (4th or 5th edition) were included in the sample study. Participant sample demographic characteristics are presented in Table 3. Of the sample, 60% ($N=15$) were male and 40% ($N=10$) were female. The mean age of the sample was 42.29, the mean years of education was 12.06, and the mean score on the WRAT-4 on the Word Reading test was 57.24. The sample mean score on the BPRS-E was 61.31.

Table 3

Sample Demographic Characteristics, Schizophrenia Participants N=26

Demographic characteristics	Measurement
Male/Female	15/10
Mean age in years	42.29
Year of education	12.06
Mean WRAT-4 (Word Reading) score	57.24
BPRS-E mean	61.31 ($N=17$)

Analysis Results

Results of the analysis of the first hypothesis, priming crystallized knowledge will improve the recall among schizophrenics as compared to no-prime, suggested that participants obtained a higher mean score on the COWAT Primed trial (73.18; $SD = 16.15$) than the COWAT No Prime trial (65.91; $SD = 10.98$); $t(21) = 2.935, p=.008$. This is shown in Table 4.

Table 4

Prime and No-Prime Learning in Crystallized Knowledge

Test	Hypothesis 1		
	Mean	SD	n
T-score for COWAT (Primed)	73.18	16.15	22
T-score for COWAT (No-Prime)	65.91	10.98	22

The second hypothesis stated that performance on a task based in crystallized knowledge would be better than performance on a task based on a fluid knowledge in schizophrenic individuals. Results of the analysis for this hypothesis indicated that participant mean was significantly lower on the incidental task based in crystallized knowledge as measured by the BNT-15 item test (49.24; $SD = 4.59$) than on the incidental task based in fluid learning task in the paired retrieval condition (66.86; $SD = 20.90$); $t(23) = -4.315$, $p = .000$. In a second analysis, no significant difference was found between participant mean participant performance on the incidental task based in the crystallized and the overall mean of the incidental task based in a fluid learning task in the free recall retrieval condition $t(23) = 1.018$, $p = .319$. Table 5 shows the results for this analysis.

Table 5

Incidental Learning in Crystallized and Fluid Knowledge

Test	Hypothesis 2		
	Mean	<i>SD</i>	<i>n</i>
T-score for BNT-15	49.24	4.59	24
T-score for Digits Span Incidental (Paired)	66.86	20.90	24
T-score for BNT-15	49.24	4.59	24
T-score for Digits Span Incidental (Free Recall)	46.69	2.39	24

No significant differences were identified between participant performance on an incidental task as compared to a procedural learning task in the crystallized knowledge condition as measured by the digit span incidental task (Paired) and the Finger Tapping

Test - Dominant Hand $t(21) = -1.085, p = .290$ or Non-Dominant Hand $t = -.687, p = .500$, respectively. Table 6 displays these results.

Table 6

Incidental and Procedural Learning in Fluid Knowledge

Test	Hypothesis 3		
	Mean	SD	<i>n</i>
T-score for Digits Span Incidental (Paired)	68.77	22.61	22
T-score for Finger Tapping (Dominant Hand)	73.77	17.77	22
Non-Dominant Hand			
T-score for Digits Span Incidental (Paired)	68.77	22.61	22
T-score for Finger Tapping (Non-Dominant Hand)	72.22	15.90	22

An analysis comparing participant performance in a primed learning task, as measured by the COWAT category test, compared to an incidental learning task, as measure by the BNT-15 item test, based in crystallized knowledge produced the following results (Table 7). Participants obtained a higher mean score in the primed learning condition (75.22; $SD = 16.20$) than the incidental learning condition (49.69; $SD = 4.14$); $t(22) = 8.178, p = .000$.

Table 7

Priming and Incidental Learning in Crystallized Knowledge

Test	Hypothesis 4		
	Mean	<i>SD</i>	<i>n</i>
T-score for COWAT (Primed)	75.22	16.20	23
T-score for BNT-15	49.69	4.14	23

Chapter V

Discussion

This study examined differences in implicit learning abilities in individuals with schizophrenia, and their differences in tasks based in fluid and crystalized knowledge and intelligence. To my knowledge, this is the first study that sought to understand how these individual factors relate to each other in this population. The existing research in schizophrenia has already demonstrated that individuals with schizophrenia are less compromised in tasks of implicit learning than those utilizing explicit learning strategies. In a study by Danion et al. (1999), discussed earlier in this dissertation, recognition abilities amongst individuals with schizophrenia were researched, comparing performance during conscious recognition tasks and recognition tasks occurring outside conscious awareness. Findings from Danion et al.'s (1999) study revealed significant impairment on the explicit recognition tasks and unimpaired performance on the implicit learning task. Horan and colleagues (2008) compared performance of persons with schizophrenia to controls in the domains of procedural learning, incidental learning, and explicit learning and memory, and, consistent with previous findings, they identified impaired performance on explicit learning tasks. They also found that the schizophrenic group performed better on an implicit learning task that provided corrective feedback than on an implicit learning task that did provide corrective feedback for gradual learning. In a study by Huddy et al. (2009) they found that motor inhibitory processes during conscious tasks were impaired but non-conscious tasks were unaffected in first-episode psychosis.

In this study, only implicit forms of learning were studied, and specifically three types of implicit learning strategies were studied; priming, incidental, and procedural. These learning mechanisms were studied in the context of the type of knowledge and intelligence the task was set in: crystallized or fluid. This study had four main hypotheses. The first prediction was that priming crystallized knowledge would improve participant word production. The results obtained in this study supported this hypothesis. Participants who were read a category paragraph (the prime) prior to being asked to produce words in that category performed significantly better than participants who were read a neutral paragraph (no prime) prior to being asked to produce words from a separate category. This is consistent with results obtained by Bressi et al. (1998) that demonstrated that individuals with schizophrenia showed implicit priming effects that were normal. However, differences between their control group and the experimental group were found to be non-significant. This study did not seek to compare the experimental group to a control group but rather to understand within-sample differences using a paired sample *t* test, between priming and not priming for crystallized knowledge. This study applied a self-created category priming paragraph instead of a word-stem task, which was used by Bressi et al. (1998), for priming crystallized knowledge.

The second prediction of this study was that participant performance on tasks based in crystallized knowledge would be better than performance on tasks based in fluid knowledge in schizophrenic patients. Performance on two separate tasks were compared; both tasks applied an incidental learning strategy. Two separate analyses were conducted to test this hypothesis, the first compared performance in the crystallized knowledge condition with performance in the fluid learning condition in which a paired (cued)

retrieval strategy was applied during the retrieval phase of the latter condition. The second analysis also compared performance on the crystallized condition with performance on the fluid learning condition but free-recall was used during the fluid learning retrieval condition in this second analysis. In the study by Palmer et al. (2010), they found that their experimental group with schizophrenia were significantly higher functioning on a measure of crystallized intelligence (measured by the Verbal Comprehension Index (VCI) compared to the other five factors from the WAIS-III/WMS-III six-factor model. In this study, two sets of analyses were conducted to test the hypothesis. Findings from the first analysis were only partially consistent with the original prediction: Results showed that fluid knowledge performance was significantly better than crystallized knowledge performance in tasks employing an incidental learning strategy when a paired (cued) retrieval strategy was used to facilitate recall during the retrieval phase of the incidental task based in fluid knowledge. This is consistent with the conclusions made by Gold et al. (2009) in their meta-analysis, that a possible preserved use of abstract and peripheral cues for target information selection and that specific aspects of selective attention used for storing information in visual working memory may be intact in this population. The second analysis performed to test this hypothesis also compared performance between the task based in crystallized knowledge and the task based in fluid knowledge.

In the meta-analysis by Gold et al. (2009) they also concluded there are some domains that may in fact be relatively spared despite the currently presumed generalized cognitive impairment. Some of these domains included “aspects of attention, procedural memory, and emotional processing” (Gold et al., 2009, p. 294). The results for this study

found that procedural learning was not significantly better or worse compared to a task employing an incidental learning strategy; both tasks were based in fluid knowledge. Two separate analyses were also run to test this hypothesis. The first analysis compared incidental learning with procedural learning when participants used their dominant hand in the finger tapping test, and in the second analysis participant performance in incidental learning was compared to participant performance in procedural learning using their non-dominant hand. Procedural learning was compared to the incidental learning task that employed a pairing (cued) retrieval strategy. The researchers of the Gold et al. (2009) study and many reviewers of this investigation identified the problem of the power as a possible limitation because of the small sample size. Findings in the study by Bressi et al. (1998) comparing performance by persons with schizophrenia to non-schizophrenia control participants on a lexical-semantic priming task and a sequence-skill learning task comparing priming and procedural learning failed to observe any significant differences on the sequence-learning task in the schizophrenic group (Bressi et al., 1998). The fourth prediction that participants would perform better in the priming than the incidental learning condition in tasks based in crystallized knowledge was supported. As predicted, participants performed better in the primed learning condition than in the incidental learning condition.

Gold et al. (2006) implied that their findings, in addition to future advancements in our knowledge of schizophrenia, would contribute to models of cognitive impairment in schizophrenia, and in developing this theoretical model may facilitate identification of strengths and weaknesses that may guide individualized and tailored models for treatment. This current study contributes to future advancements in knowledge of

cognitive functions in schizophrenia and theoretical models of schizophrenia.

Implications for Preserved Functioning

Wessel et al. (2012) emphasized that implicit learning is distinct in that it facilitates the changes in how people adapt to their environment through interacting with it. Therefore, there exists an inherent necessity of implicit learning functions for the adequate achievement of capabilities and adaptations required for daily living skills.

Findings from this study as well as previous literature has shown that some aspects of implicit learning are more preserved than others, and there has been significant evidence in the literature showing more preserved implicit than explicit functions.

Impaired functional outcome and deficits in skills in domains such as social and vocational have been well established and observed in individuals with schizophrenia.

The evidence for a relationship of neuropsychology and cognitive factors to functional outcomes in individuals with schizophrenia is also overwhelming, and is discussed extensively in the research. This study presented some of this literature: Shanks et al. (1998) found associations of cognition with poor functional outcomes, Green et al. (2000) presented findings of deficits in neurocognition and their relationship to functional outcomes in the schizophrenic population, and Lindenmayer et al. (2017) suggested the importance of identifying variations in cognition amongst patients for effective treatment and improving daily skills functioning. Green (1996) found that neurocognitive deficits were correlated with deficits in functional outcomes and the ability for patients to benefit from psychiatric rehabilitation, and Leifker et al. (2009) found that neuropsychological performance was associated with everyday outcomes. Implicit learning has been significantly correlated with learning and functioning in domains of daily skills. In

findings by Gold et al. (2006), participants' ability to apply selective attention processes for the purpose of encoding and storing target data was found to remain intact, specifically their abilities to use both abstract and peripheral cues for target information selection.

Top-down and bottom-up processing work together to guide the individual to consolidate his or her perceptual experience, learning, and creating schemas from which he or she constructs the world. This information eventually either becomes stored into existing schemas, or new ones are created (Gold et al., 2006; Goldstein, 2005; Keefe et al., 2002). Learning capitalizes on both processes, top-down and bottom-up, so naturally a better understanding of this function in persons with schizophrenia may help to explain neurocognitive functions and how they impact learning, and ultimately how rehabilitation and skill training efforts may be modified to fit these demands. Understanding the full extent of how implicit learning and thinking plays a role in psychiatric rehabilitation and its relationship to functional outcomes is beyond the scope of this study however, this would seem like the next direction for this research.

Future Implications Directions

Tilborg et al. (2011) proposed that rehabilitative techniques that activate implicit learning processes could be used as alternative forms of intervention for improving daily functioning. The findings of the current study may enlighten and facilitate how these alternative interventions are structured and implemented. For example, practitioners could capitalize on the propensity of individuals with schizophrenia to better apply crystallized intelligence within a context in which priming is used. This may be in the context of utilizing cues to facilitate learning of information needed to succeed in the academic or

vocational arena. Thus, modifying learning intervention and environments that apply techniques that rely on priming for both teaching and eliciting desired responses. Furthermore, in line with the findings of Gold et al. (2009), setting up rehabilitative environments that include either naturally occurring or staged peripheral and target cues in the rehabilitation environment may strengthen learning and ultimately daily functioning for individuals with schizophrenia. Fluid knowledge performance was in fact, significantly better than crystallized knowledge performance in tasks employing an incidental learning strategy. Another area of learning that may be considered when developing future rehabilitation is selecting the technique that is best suited for the type of knowledge/intelligence in which the functional outcome is based. For example, if the skill is based in fluid knowledge, such as sequential daily living tasks, the findings of the current study indicate that this skill would be best rehabilitated implementing an incidental learning modality, and that using a procedural learning technique to learn the same skill may not prove as effective.

As indicated by Penades et al. (2010), the relationship of impaired cognition and functional outcome is clear in schizophrenia but how cognition relates to functional change in treatment is not. As noted in the investigation by Russeler et al. (2003), learning through explicit means resulted in greater acquisitions of sequence knowledge, which is thought to play a significant role in developing skills necessary for daily living activities. Future research could focus on implementing specific implicit learning techniques in crystallized and fluid intelligence and knowledge building and measuring how this correlates with functional outcomes.

As discussed in the introduction, cognition has been significantly associated with functional outcomes and performance in the rehabilitation setting (Bowen et al., 1994). Thus, identification of preserved or relatively spared cognitive functions as measured by task performance may provide insight into cognitive functions upon which individuals with schizophrenia could capitalize in rehabilitation. Future directions of studies could begin to examine the potential relationship that may exist between implicit cognitive and training abilities and social and occupational functioning.

References

- Aaronson, B.S., Sugeran, A.A., & Hafetz, M.R. (1966, September). Incidental learning in chronic schizophrenics, alcoholics, and normals. *Proceedings of the Seventy-Fourth Annual Convention of the American Psychological Association*, 181-182.
- Adcock, R.A., Dale, C., Fisher, M., Aldebot, S., Genevsky, A., Simpson, G.V., ... Vinogradov, S. (2009). When top-down meets bottom-up: Auditory training enhances verbal memory in schizophrenia. *Schizophrenia Bulletin*, 35, 1132-1141. doi:10.1093/schbul/sbp068
- Addington, J., Addington, D., & Maticka-Tyndale, E. (1991). Cognitive functioning and positive and negative symptoms in schizophrenia. *Schizophrenia Research*, 5, 123-134. Retrieved from <http://www.schres-journal.com/>
- Allardyce, J., Gaebel, W., Zielasek, J., & van Os, J. (2007). Deconstructing Psychosis Conference February 2006: The validity of schizophrenia and alternative approaches to the classification of psychosis. *Schizophrenia Bulletin*, 33(4), 863-867. doi:10.1093/schbul/sbm051
- Aleman, A., Hijman, R., de Haan, E.H., & Kahn, R.S. (1999). Memory impairments in schizophrenia: A meta-analysis. *American Journal of Psychiatry*, 156, 1358-1366. Retrieved from <http://ajp.psychiatryonline.org/>
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed., text rev.). Washington, DC: Author.
- Anderson, J.R. (1982). Acquisition of cognitive skill. *Psychological Review*, 89(4), 369-406. doi:10.1037/0033-295X.89.4.369
- Anderson, J.R. (1995). *Learning and memory: An integrated approach*. New York, NY: Wiley.
- Andreasen, N.C., Nopoulos, P., O'Leary, D.S., Miller, D.D., Wassink, T., & Flaum, M. (1999). Defining the phenotype of schizophrenia: Cognitive dysmetria and its neural mechanisms. *Biological Psychiatry*, 46(7), 908-920. Retrieved from <http://www.journals.elsevier.com/biological-psychiatry>
- Andreasen N.C., Olsen S.A., Dennert J.W., & Smith M.R. (1982). Ventricular enlargement in schizophrenia: Relationship to positive and negative symptoms. *American Journal of Psychiatry*, 139(3), 297-302. Retrieved from <http://ajp.psychiatryonline.org/>
- Badcock, J.C., Dragovic, M., Waters, F.A., & Jablensky, A. (2005). Dimensions of intelligence in schizophrenia: Evidence from patients with preserved, deteriorated and compromised intellect. *Journal of Psychiatric Research*, 39, 11-19. doi:10.1016/j.jpsychires.2004.05.002

- Bagney, A., Dompablo, M., Santabarbara, J., Moreno-Ortega, M., Lobo, A., Jimenez-Arrieto, M.A., ... Rodriguez-Jimenez, R. (2015). Are negative symptoms really related to cognition in schizophrenia? *Psychiatry Research*, *230*(2), 377-82. doi:10.1016/j.psychres.2015.09.022
- Bearden, C.E., & Cannon, T.D. (1998). Neurodevelopmental origins of schizophrenia. In D. Routh & R.J. DeRubeis (Eds), *The science of clinical psychology: Accomplishments and future directions* (pp. 23-51). Washington, DC: American Psychological Association.
- Belforte, J.E., Zsiros, V., Sklar, E.R., Zhihong, J., Gu, Y., Li, Y., ... Nakazawa, K. (2010). Postnatal NMDA receptor ablation in corticolimbic interneurons confers schizophrenia-like phenotypes. *Nature Neuroscience*, *13*, 76-83. doi:10.1038/nn.2447
- Belsham, B. (2001). Glutamate and its role in psychiatric illness. *Human Psychopharmacology: Clinical and Experimental*, *16*(2), 139-156. doi:10.1002/hup.279
- Benton, A.L., Hamsher, K. deS., Sivan, A.B. (1994). *Multilingual Aphasia Examination*. Iowa City, IA: AJA Associates.
- Borgwardt, S.J., McGuire, P.K., Aston, J., Gschwandtner, U., Pfluger, M.O., Stieglitz, R.D., ... Riecher-Rossler, A. (2008). Reductions in frontal, temporal and parietal volume associated with the onset of psychosis. *Schizophrenia Research*, *106*, 108-114. doi:10.1016/j.schres.2008.08.007
- Boksa, P. (2012). Abnormal synaptic pruning in schizophrenia: Urban myth or reality? *Journal of Psychiatric Neuroscience*, *35*, 75-77. doi:10.1503/jpn120007
- Bornstein, R.A., Baker, G.B., & Douglass, A.B. (1987). Short-term retest reliability of the Halstead-Reitan Battery in a normal sample. *The Journal of Nervous and Mental Disease*, *175*, 229-232. doi:10.1097/00005053-198704000-00007
- Brébion, G., Gorman, J.X., Malaspina, D., & Amador, X. (2005) A model of verbal memory impairments in schizophrenia: Two systems and their association with underlying cognitive processes and clinical symptoms. *Psychological Medicine*, *35*, 133-142. doi:10.1017/S0033291704002879
- Brekke, J.S., Raine, A., Ansel, M., Lencz, T., & Bird, L. (1997). Neuropsychological and psychophysiological correlates of psychosocial functioning in schizophrenia. *Schizophrenia Bulletin*, *23*(1), 19-28. Retrieved from <http://schizophreniabulletin.oxfordjournals.org/>

- Bressi, S., Miele, L., Bressi, C., Vita, A., Astori, S., Gimosti, E., ... Zirulia, G. (1998). Sequence skill learning and semantic priming in schizophrenia. Evidence for differential impairment of the implicit memory system. *Trends in Clinical and Experimental Psychiatry*, 14(4), 179-188. Retrieved from http://www.scielo.br/scielo.php?script=sci_serial&pid=2237-6089&lng=en&nrm=iso
- Brodeur, M.B., Pelletier, M., & Lepage M. (2009). Memory for everyday actions in schizophrenia. *Schizophrenia Research*, 114, 71-78. doi:10.1016/j.schres.2009.06.023
- Bowen, L., Wallace, C.J., Glynn, S.M., Nuechterlein, K.H., Lutzker, J.R., & Kuehnel, T.G. (1994). Schizophrenic individuals' cognitive functioning and performance in interpersonal interactions and skill training procedures. *Journal of Psychiatric Research*, 28, 289-301. Retrieved from <http://www.journalofpsychiatricresearch.com/>
- Bowie, C.R., Leung, W.W., Reichenberg, A., McClure, M.M., Patterson, T.L., Heaton, R.K., & Harvey, P.D. (2008). Predicting schizophrenia patients' real-world behavior with specific neuropsychological and functional capacity measures. *Biological Psychiatry*, 63, 505-511. doi:10.1016/j.biopsych.2007.05.022
- Bowie, C.R., Reichenberg, A., Patterson, T.L., Heaton, R.K., & Harvey, P.D. (2006). Determinants of real-world functional performance in schizophrenia subjects: Correlations with cognition, functional capacity, and symptoms. *American Journal of Psychiatry*, 163(3), 418-425. doi:10.1176/appi.ajp.163.3.418
- Bryan, J., & Luszcz, M.A. (2000). Measures of fluency as predictors of incidental memory among older adults. *Psychology and Aging*, 15, 483-489. doi:10.1037//Q882-7974.15.3.483
- Burwell, R.D., Saddoris, M.P., Bucci, D.J., & Khested, A.W. (2004). Corticohippocampal contributions to spatial and contextual learning. *Journal of Neuroscience*, 24, 3826-3836. doi:10.1523/JNEUROSCI.0410-04.2004
- Canan, K., White, T., & Bingham, C. (2009). Incidental and intentional sequence learning in youth-onset psychosis and attention-deficit/hyperactivity disorder (ADHD). *Neuropsychology*, 23, 445-459. doi:10.1037/a0015562
- Carpenter, W.T., Heinrichs, D.W., & Wagman, A.M. (1988). Deficit and nondeficit forms of schizophrenia: The concept. *American Journal of Psychiatry*, 145(5), 578-583. Retrieved from <http://ajp.psychiatryonline.org/>
- Cascella, N.G., Testa, S.M., Meyer, S.M., Rao, V.A., Diasz-Asper, C.M., Pearlson, G.D., & Schretlen, D.J. (2008). Neuropsychological impairment in deficit vs. non-deficit schizophrenia. *Journal of Psychiatric Research*, 42, 930-937. doi:10.1016/j.psychires.2007.10.002

- Cattell, R.B. (1963). Theory of fluid and crystallized intelligence: A critical experiment. *Journal of Educational Psychology*, *54*(1), 1-22. doi:10.1037/h0046743
- Chakravarti, A. (1999). Population genetics-making sense out of sequence. *Nature Genetics*, *21*(1 Suppl), 56-60. Retrieved from <http://genetics.nature.com>
- Chen, E.Y., Hui, C.L., Dunn, E.L., Miao, M.Y., Yeung, W., Wong, C., ... Tang, W. (2005). A prospective 3-year longitudinal study of cognitive predictors of relapse in first-episode schizophrenic patients. *Schizophrenia Research*, *77*, 99-104. doi:10.1016/j.schres.2005.02.020
- Cohen, A.S., Forbes, C.B., Mann, M.C., & Blanchard, J.J. (2006). Specific cognitive deficits and differential domains of social functioning impairments in schizophrenia. *Schizophrenia Research*, *81*, 227-238. doi:10.1016/j.schres.2005.09.007
- Cummings, J.L. (1985). Organic delusions, phenomenology, anatomical correlations, and review. *British Journal of Psychiatry*, *146*, 184-197. doi:10.1192/bjp.146.2.184
- Cuthbert, B.N., & Insel, T.R. (2010). Toward new approaches to psychotic disorders: The NIMH research domain criteria project. *Schizophrenia Bulletin*, *36*, 1961-1962. doi:10.1093/schbul/sbq108
- Da Silva, F.N., Irani, F., Richard, J., Brensinger, C.M., Bilker, W.B., Gur, R.E., & Gur, R.C. (2012). More than just tapping: Index finger-tapping measures procedural learning in schizophrenia. *Schizophrenia Research*, *137*, 234-230. doi:10.1016/j.schres.2012.01.018
- Danion, J.M., Meulemans, T., Kauffmann-Muller, F., & Vermaat, H. (2001). Intact implicit learning in schizophrenia. *American Journal of Psychiatry*, *158*, 944-948. doi:10.1176/appi.ajp.158.6.944
- Danion, J.M., Rizzo, L., & Bruant, A. (1999). Functional mechanisms underlying impaired recognition memory and conscious awareness in patients with schizophrenia. *Archives of General Psychiatry*, *56*, 639-644. doi:10.1001/archpsyc.56.7.639
- Demirel, A., Demirel, O.F., Emül, M., Duran, A., & Ügur, M. (2014) Relationship between IGF-1, schizophrenia, and treatment of metabolic syndrome. *Comprehensive Psychiatry*, *55*, 1391-1397. doi:10.1016/j.comppsy.2014.04.008
- Dennis, M., Francis, D.J., Cirino, P.T., Schanshar, R., Barnes, M.A., & Fletcher, J.M. (2009). Why IQ is not a covariate in cognitive studies of neurodevelopmental disorder. *Journal of the International Neuropsychological Society*, *15*, 331-343. doi:10.1017/S1355617709090481

- Dickinson, D., Goldberg, T.E., Gold, J.M., ELEVAG, B., & Weinberger D.R. (2011). Cognitive factor structure and invariance in people with schizophrenia, their unaffected siblings, and controls. *Schizophrenia Bulletin*, *37*(6), 1157-1167. doi:10.1093/schbul/sbq018
- Dikmen, S.S., Heaton, R.K., Grant, I., & Temkin, N.R. (1999). Test-retest reliability and practice effects of expanded Halstead-Reitan Neuropsychological Test Battery. *Journal of the International Neuropsychological Society*, *5*, 346-356. doi:10.1017/s1355617799544056
- Dingemans, P.M., Linszen, D.H., Lenior, M.E., & Smeets R.M. (1995). Component structure of the Expanded Brief Psychiatric Rating Scale (BPRS-E). *Psychopharmacology*, *122*, 263-267. doi:10.1007/BF02246547
- Doeller, C.F., & Burgess, N. (2008). Distinct error-correcting and incidental learning of location relative to landmarks and boundaries. *PNAS*, *105*, 5909-5914. doi:10.1073/pnas.0711433105
- Dominguez, M.G., Viechtbauer, W., Simons, C.J., van Os, J., & Krabbendam, L. (2009). Are psychotic psychopathology and neurocognition orthogonal? A systematic review of their associations. *Psychological Bulletin*, *135*, 157-171. doi:10.1037/a0014415
- Faraone, S.V., Seidman, L.J., Kremen, W.S., Pepple, J.R., Lyons, M.J., & Tsuang, M.T. (1995). Neuropsychological functioning among the nonpsychotic relative of schizophrenic patients: A diagnostic efficiency analysis. *Journal of Abnormal Psychology*, *104*(2), 286-304. doi:10.1037/0021-843X.104.2.286
- Fischer, M., Holland, C., Subramaniam, K., & Vinogradov, S. (2010). Neuroplasticity-based cognitive training in schizophrenia: An interim report on the effects 6 months later. *Schizophrenia Bulletin*, *36*, 869-879. doi:10.1093/schbul/sbn170
- Fitts, P.M. (1964). Perceptual-motor skill learning. In A.W. Melton (Ed.), *Categories of human learning* (pp. 243-285). New York, NY: Academic Press.
- Flanagan, D.P., & Dixon, S.G. (2013). The Cattell-Horn-Carroll theory of cognitive abilities. In C.R. Reynolds, K.J. Vannest, & E. Fletcher-Janzen (Eds.), *The encyclopedia of special education* (pp. 369-382). New York, NY: John Wiley.
- Fosshage, J.L. (2011). How do we “know” what we “know?” And change what we “know?” *Psychoanalytic Dialogues*, *21*, 55-74. doi:10.1080/10481885.2011.545328
- Francis, A.N., Bhojirah, T.S., Prasad, K.M., Montrose, D., Eack, S.M., Rajarethianam, R., ... Keshavan, M.S. (2013). Alterations in the cerebral white matter of genetic high risk offspring of patients with schizophrenia spectrum disorder. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, *40*, 187-192. doi:10.1016/j.pnpbp.2012.08.003

- Gold, J.M. (2004). Cognitive deficits as treatment targets in schizophrenia. *Schizophrenia Research*, 72, 21-28. Retrieved from <http://www.journals.elsevier.com/schizophrenia-research/>
- Gold, J.M., Fuller, R.L., Robinson, B.M., McMahon, R.P., Braun, E.L., & Luck, S.J. (2006). Intact attentional control of working memory encoding in schizophrenia. *Journal of Abnormal Psychology*, 115, 658-673. doi:10.1037/0021-843X.115.4.658
- Gold, J.M., Hahn, B., Strauss, G.P., & Waltz, J.A. (2009). Turning it upside down: Areas of preserved cognitive function in schizophrenia. *Neuropsychology Review*, 19, 294-311. doi:10.1007/s11065-009-9098-x
- Goldstein, E.B. (2005). *Cognitive psychology: Connecting mind, research, and everyday experience* (2nd ed.). Belmont, CA: Thompson Wadsworth.
- Gomar, J.J., Porarol-Clotet, E., Sarró, S., Salvador, R., Myers, C.E., & McKenna, P.J. (2011). Procedural learning in schizophrenia: Reconciling the discrepant findings. *Biological Psychiatry*, 69, 49-54. doi:10.1016/j.biopsych.2010.07.013
- Gordon, J. (2010). Testing the glutamate hypothesis of schizophrenia. *Nature Neuroscience*, 13(1), 2-4. Retrieved from <http://www.nature.com/neuro>
- Gottesman, I.I., & Shields, J. (1982). *Schizophrenia, the epigenetic puzzle*. New York, NY: Cambridge University Press.
- Gras-Vincedon, A., Danion, J.M., Grangé, D., Miloslav, B., Willard-Schroeder, W., Sichel, J.P., & Singer, L. (1994). Explicit memory, repetition priming and cognitive skill learning in schizophrenia. *Schizophrenia Research*, 13(2), 117-126. Retrieved from <http://www.schres-journal.com/>
- Green, M.F. (1996). What are the functional consequences of neurocognitive deficits in schizophrenia. *American Journal of Psychiatry*, 153, 221-330. Retrieved from <http://ajp.psychiatryonline.org/>
- Green, M.F., Kern, R.S., Braff, S.L., & Mintz, J. (2000). Neurocognitive deficits and functional outcome in schizophrenia: Are we measuring the “right stuff”? *Schizophrenia Bulletin*, 26(1), 119-136. Retrieved from <http://schizophreniabulletin.oxfordjournals.org/>
- Grilli, M., Toninelli, G.F., Uberti, D., Spano, P.F., & Memo, M. (2013). Alzheimer’s disease linking neurodegeneration with neurodevelopment. *Functional Neurology*, 18(3), 145-148. Retrieved from <http://www.functionalneurology.com/>
- Grimm, O., Kersting, J.M., Zink, M., & Gass, P. (2010). FMIR in prodromal schizophrenia. *The German Journal of Psychiatry*, 13, 57-60. Retrieved from <http://www.gjpsy.uni-goettingen.de/>

- Gureckis, T.M., James, T.W., & Nosofsky, R.M. (2011). Re-evaluating dissociations between implicit and explicit category learning: An event-related fMRI study. *Journal of Cognitive Neuroscience*, *23*(7), 1697-1709. doi:10.1162/jocn.2010.21538
- Harvey, P.D., Rabinowitz, J., Eerdeken, M., & Davidson, M. (2005). Treatment of cognitive impairment in early psychosis: A comparison of risperidone and haloperidol in a large long-term trial. *The American Journal of Psychiatry*, *162*(10), 1888-1895. doi:10.1176/appi.ajp.162.10.1888
- Heaton, R.K., Taylor, M.K., & Manly, J. (2003). Demographic effects and demographically corrected norms with the WAIS-III and WMS-III. In D.S. Tulsky, R.K. Saklofske, G.J. Chelune, R.K. Heaton, R.J. Ivnik, R. Bornstein, ... M.F. Ledbetter (Eds.), *Clinical interpretation of the WAIS-III and WMS-III* (pp. 181-210). San Diego: Academic Press.
- Heckers, S., Barch, D.M., Bustillo, J., Gaebel, W., Gur, R., Malaspina, D., ... Carpenter, W. (2013). Structure of the psychotic disorders classification in DSM-5. *Schizophrenia Research*, *150*, 11-14. Retrieved from <http://www.schres-journal.com/>
- Hedlund, J., & Vieweg, B.W. (1980). The brief psychiatric rating scale (BPRS): A comprehensive review. *Journal of Operational Psychiatry*, *11*, 48-65. Retrieved from <http://missouri.edu/>
- Heinrichs, R.W., Miles, A.A., Smith, D., Zargarian, T., Vaz, S.M., Goldberg, J.O., & Ammari, N. (2008). Cognitive, clinical, and functional characteristics of verbally superior schizophrenia patients. *Neuropsychology*, *22*, 321-328. doi:10.1037/0894-4105.22.3.321
- Heinrichs, R.W., & Zakzanis, K.K. (1998). Neurocognitive deficit in schizophrenia: A quantitative review of the evidence. *Neuropsychology*, *12*(3), 426-445. doi:10.1037/0894-4105.12.3.426
- Horan, W.P., Knowlton, B.J., Wynn, J.K., Green, M.F., Mintz, J., & Nuechterleing, K.H. (2008). Impaired implicit learning in schizophrenia. *Neuropsychology*, *22*, 606-617. doi:10.1037/a0012602
- Huddy, V.C., Aron, A.R., Harrison, M., Barnes, T.R., Robbins, T.W., & Joyce, E.M. (2009). Impaired conscious inhibitory processing in recent onset schizophrenia. *Psychological Medicine*, *39*, 907-916. doi:10.1017/S0033291708004340
- Insel, T.R. (2010). Rethinking schizophrenia. *Nature*, *468*, 187-193. doi:10.1038/nature09552
- Jakob, H., & Beckmann, H. (1986). Prenatal development disturbances in the limbic allocortex in schizophrenics. *Journal of Neural Transmission*, *65*, 154-161. doi:10.1007/BF01249090

- Kandel, E.R., Schwartz, J.H., Jessel, T.M., Siegelbaum, S.A., & Hudspeth, A.J. (Eds.). (2013). *Principles of neural science* (5th ed.). New York, NY: McGraw Hill.
- Kaplan, E., Goodglass, H., & Weintraub, S. (1983). *The Boston Naming Test*. Philadelphia, PA: Lea & Febiger.
- Keefe, R.S.E., Arnold, M.C., Bayen, U.J., McEvoy, J.P., & Wilson, W.H. (2002). Source monitoring deficits for self-generated stimuli in schizophrenia: Multinomial modeling analysis. *Psychological Medicine*, 26(4), 903-914. Retrieved from <https://www.cambridge.org/core/journals/psychological-medicine>
- Keefe, R.S., Harvey, P.D., Lenzenweger, M.F., Davidson, M., Apter, S.H., Schmeidler, J., ... Davis, K.L. (1992). Empirical assessment of the factorial structure of clinical symptoms in schizophrenia: Negative symptoms. *Psychiatry Research*, 44, 153-165. Retrieved from <http://www.psy-journal.com/>
- Knowles, E.E., David, A.S., & Reichenberg, A. (2010). Processing speed deficits in schizophrenia: Re-examining the evidence. *American Journal of Psychiatry*, 167(7), 828-835. doi:10.1176/appi.ajp.2010.09070937
- Krishnan, R.R., Keefe, R., & Kraus, M. (2009). Schizophrenia is a disorder of higher order hierarchical processing. *Medical Hypotheses*, 72, 740-744. doi:10.1016/j.mehy.2008.12.039
- Kumari, V., Gray, J.A., Honey, G.D., Soni, W., Bullmore, E.T., Williams, S.C., ... Sharma, T. (2002). Procedural learning in schizophrenia: A functional magnetic resonance imaging investigation. *Schizophrenia Research*, 57(1), 97-100. doi:10.1016/S0920-9964(01)00270-5
- Kurtz, M.M., Wexler, B.E., Fujimoto, M., Shagan, D.S., & Seltzer, J.C. (2008). Symptoms versus neurocognition as predictors of change in life skills in schizophrenia after outpatient rehabilitation. *Schizophrenia Research*, 102, 303-311. doi:10.1016/j.schres.2008.03.023
- Lezak, M.D., Howieson, D.B., & Loring, D.W. (2004). *Neuropsychological assessment* (4th ed.). New York, NY: Oxford University Press.
- Liemburg, E., Castelein, S., Stewart, R., van der Gaag, M., Aleman, A., Knegtering, H., & Genetic Risk and Outcome of Psychosis (GROUP) Investigators. (2013). Two subdomains of negative symptoms in psychotic disorders: Established and confirmed in two large cohorts. *Journal of Psychiatric Research*, 47, 718-725. Retrieved from <http://www.journalofpsychiatricresearch.com/>
- Lindenmayer, J.P., Ozog, V.A., Khan, A., Ljuri, I., Fregenti, S., & McGurk, S.R. (2017). Predictors of response to cognitive remediation in service recipients with severe mental illness. *Psychiatric Rehabilitation Journal*, 40(1), 61-69. doi:10.1037/prj0000252

- Lukoff, D., Nuechterlein, K. H., & Ventura, J. (1986). Manual for expanded Brief Psychiatric Rating Scale (BPRS). *Schizophrenia Bulletin*, *12*, 594-602. Retrieved from <https://academic.oup.com/schizophreniabulletin>
- Mack, W.J., Freed, D.M., Williams, B.W., & Henderson, V.W. (1992). Boston Naming Test: Shortened versions for use in Alzheimer's disease. *Journal of Gerontology*, *47*, 154-158. doi:10.1093/geronj/47.3.P154
- MacWhinney, B. (1997). Implicit and explicit processes. *Studies in Second Language Acquisition*, *19*, 277-281. doi:10.1017/S0272263197002076
- Manns, J.R., & Squire, L.R. (2001). Perceptual learning, awareness, and the hippocampus. *Hippocampus*, *11*, 776-782. doi:10.1002/hipo.1093
- Marcopulos, B.A., & Kurtz, M.M. (Eds.). (2012). *Clinical neuropsychological foundations of schizophrenia*. New York, NY: Psychology Press.
- Mayoral, M., Zabala, A., Robles, O., Bombin, I. Andres, P., Parellada, M., ... Arango, C. (2008). Neuropsychological functioning in adolescents with first episode psychosis: A two-year follow-up study. *European Psychiatry*, *23*, 375-383. doi:10.1016/j.eurpsy.2008.01.1420
- McCarley, R.W., Shenton, M.E., O'Donnell, B.F., & Nestor, P. (1993). Uniting Kraepelin and Bleuler: The psychology of schizophrenia and biology of temporal lobe abnormalities. *Harvard Review of Psychiatry*, *1*(1), 36-56. doi:10.3109/10673229309017055
- McClellan, J.M., Susser, E., & King, M.C. (2007). Schizophrenia: A common disease caused by multiple rare alleles. *British Journal of Psychiatry*, *190*, 194-199. doi:10.1192/bjp.bp.106.025585
- McGrath, J.J., & Richards, L.J. (2009). Why schizophrenia epidemiology needs neurobiology-and vice versa. *Schizophrenia Bulletin*, *35*, 577-581. Retrieved from <http://schizophreniabulletin.oxfordjournals.org/>
- McGrew, K.S. (2009). CHC theory and human cognitive abilities project: Standing on the shoulders of the giants of psychometric intelligence research. *Intelligence*, *37*(1), 1-10. doi:10.1016/j.intell.2008.08.004
- McGuire, P.K., Silbersweig, D.A., Wright, I., Murray, R.M., David, A.S., Frackowiak, R.S., & Frith, C.D. (1995). Abnormal monitoring of inner speech: A physiological basis for auditory hallucinations. *Lancet*, *346*, 596-600. Retrieved from <http://www.thelancet.com>
- Medalia, A., & Choi, J. (2009). Cognitive remediation in schizophrenia. *Neuropsychology Review*, *19*, 353-364. doi:10.1007/s11065-009-9097-y

- Mednick, S.A., Machon, R.A., Huttunen, M.O., & Bonett, D. (1988). Adult schizophrenia following prenatal exposure to an influenza epidemic. *Archives of General Psychiatry*, *45*(2) 189-192. Retrieved from <http://archpsyc.jamanetwork.com/>
- Mertler, C.A., & Vannatta, R.A. (2010). *Advanced and multivariate statistical methods*. Los Angeles, CA: Pyrczak.
- Mesholam-Gately, R.I., Guiliano, A.J., Goff, K.P., Faraone, S.V., & Seidman, L.J. (2009). Neurocognition in first-episode schizophrenia: A meta-analytic review. *Neuropsychology*, *23*, 315-336. doi:10.1037/a0014708
- Minzenberger, M.J., Ober, B.A., & Vinogradov, S. (2002). Semantic priming in schizophrenia: A review and synthesis. *Journal of International Neuropsychological Society*, *8*, 699-720. doi:10.1017.S1355617702801357
- Moghaddam, B., & Javitt, D. (2012). From revolution to evolution: The glutamate hypothesis of schizophrenia and its implication for treatment. *Neuropsychology*, *37*, 4-15. doi:10.1038/npp.2011.181
- Mueser, K.T., & Jeste, D.V. (2008). *Clinical handbook of schizophrenia*. New York, NY: Guilford Press.
- Nuechterlein, K.H., Barch, D.M., Gold, J.M., Goldberg, T.E., Green, M.F., & Heaton, R.K. (2004). Identification of separable cognitive factors in schizophrenia. *Schizophrenia Research*, *72*, 29-39. Retrieved from <http://www.schres-journal.com/>
- Nuechterlein, K.H., Green, M.F., Kern, R.S., Baade, L.E., Barch, D.M., Cohen, J.D., ... Marder, S.R. (2008). The MATRICS Consensus Cognitive Battery, Part 1: Test Selection, Reliability, and Validity. *American Journal of Psychiatry*, *165*(2), 203-213. doi:1176/appi.ajp.2007.07010042
- O'Donovan, M. (2015). Novel genetic advances in schizophrenia: An interview with Michael O'Donovan. *BMC Medicine*, *13*(181). doi:10.1186/s12916-015-0417-1
- Overall, J.F., & Gorham, D.R. (1962). The Brief Psychiatric Rating Scale. *Psychological Report*, *10*, 799-812. doi:10.2466/pr0.1962.10.3.799
- Palmer, B.W., Dawes, S.E., & Heaton, R.K. (2009). What do we know about neuropsychological aspects of schizophrenia. *Neuropsychology Review*, *19*, 365-384. doi:10.1007/s11065-009-9109-y
- Palmer, B.W., Heaton, R.K., Paulsen, J.S., Kuck, J., & Braff D. (1997). Is it possible to be schizophrenic yet neuropsychologically normal? *Neuropsychology*, *11*, 437-446. doi:10.1037/0894-4105.11.3.437

- Palmer, B.W., Savla, G.N., Fellows, I.E., Twamley, E.W., Jeste, D.V., & Lacro, J.P. (2010). Do people with schizophrenia have differential impairment in episodic memory and/or working memory relative to other cognitive abilities? *Schizophrenia Research, 116*, 259-265. doi:10.1016/j.schres.2009.11.002
- Palmer, C. D., Heiby, E., Fujii, D., & Kameoka, V. (2008). Executive functioning in schizophrenia: The contributions of attention, working memory, processing speed and general intelligence. *Graduate Student Journal of Psychology, 10*, 38-44. doi:10.1093/schbul/sbr081
- Pankow, A., Knobel, A., Voss, M., & Heinz, A. (2011). Neurobiological correlates of delusion: Beyond the salience attribution hypothesis. *Neuropsychobiology, 66*, 33-43. doi:10.1159/000337132
- Penades, R., Catalan, R., Puig, O., Masana, G., Pujol, N., Navarro, V., ... Gasto, C. (2010). Executive function needs to be targeted to improve social functioning with cognitive remediation therapy. *Psychiatry Research, 177*, 41-45. Retrieved from <http://www.psy-journal.com/>
- Purdon, S.E., Waldie, B., Wiman, A.H., Woodward, N.D., & Tibbo, P.G. (2011). Procedural learning in first episode schizophrenia investigated with functional magnetic resonance imaging. *Neuropsychology, 25*, 147-158. doi:10.1037/a0021222
- Rabe-Jablonska, J., Kotlicka-Antczak, M., & Gmitrowicz, A. (2000). Clinical picture and duration of prodromal period of schizophrenia in adolescents. *Archives of Psychiatry and Psychotherapy, 2*, 31-36. Retrieved from <http://www.archivespp.pl/>
- Rajji, T.K., Voineskos, A.N., Butters, M.A., Miranda, D., Arenovich, T., Menon, M., ... Mulsant, B.H. (2013). Cognitive performance of individuals with schizophrenia across seven decades: A study using the MATRICS consensus cognitive battery. *American Journal of Geriatric Psychiatry, 21*, 108-118. doi:10.1016/j.jagp.2012.10.011
- Ravheim, N., Butler, P.D., Schechter, I., Jalbrzikowski, M., Silipo, G., & Javitt, D.C. (2006). Reading impairment and visual processing deficit in schizophrenia. *Schizophrenia Research, 87*(1-3), 238-245. doi:10.1016/j.schres.2006.06.022
- Reitan, R. M. (1969). *Manual for administration of neuropsychological test batteries for adults and children*. Tucson, AZ: Neuropsychology Laboratory.
- Reitan, R.M., & Wolfson, D. (1985). *The Halstead-Reitan Neuropsychological Test Battery: Theory and clinical interpretation*. Tucson, AZ: Neuropsychology Laboratory.
- Reitan, B. (1996, May) *Entrepreneurial intentions: A combined models approach*. Paper presented at the 9th Nordic Small Business Research Conference, Lillehammer, Norway.

- Riedel, M., Schennach-Wolff, R., Musil, R., Dehning, S., Cerovecki, A., Opgen-Rhein, M., Matz, J., ... Spellman, I. (2010). Neurocognition and its influencing factors in the treatment of schizophrenia-effects of aripiprazole, olanzapine, quetiapine and risperidone. *Human Psychopharmacology*, *25*, 116-125. doi:10.1002/hup.1101
- Russeler, J., Kuhlicke, D., & Munte, T.F. (2003). Human error monitoring during implicit and explicit learning of a sensorimotor sequence. *Neuroscience Research*, *47*(2), 233-240. Retrieved from <http://www.journals.elsevier.com/neuroscience-research/>
- Sans-Sansa, B., McKenna, P.J., Canales-Rodriguez, E.J., Cortiz-Gil, J., Lopéz-Araquistain, L., Sarró, S., ... Pormarol-Cortet, E. (2013). Association of formal thought disorder in schizophrenia with structural brain abnormalities in language related cortical regions. *Schizophrenia Research*, *146*(1-3), 308-313. doi:10.1016/j.schres.2013.02.032
- Sayers, S.L., Curran, P.J., & Mueser, K.T. (1996). Factor structure and construct validity of the scale for the assessment of negative symptoms. *Psychological Assessment*, *8*(3), 269-280. Retrieved from <http://www.apa.org/pubs/journals/pas/>
- Seidman, L.J. (1983). Schizophrenia and brain dysfunction: An integration of recent neurodiagnostic findings. *Psychological Bulletin*, *94*, 195-238. doi:10.1037/0033-2909.94.2.195
- Seidman, L.J. (1990). The neuropsychology of schizophrenia: A neurodevelopmental and case approach. *Journal of Neuropsychiatry*, *2*, 301-312. doi:10.1176/jnp.2.3.301
- Seidman, L.J., Cassens, G.P., Kremen, W.S., & Pepple, J.R. (1992). Neuropsychology of schizophrenia. In R.F. White (Ed.), *Clinical syndromes in adult neuropsychology: The practitioner's handbook* (pp. 381-449). New York, NY: Elsevier.
- Selva-Vera, G., Balanzá-Martínez, V., Salazar-Fraile, J., Sánchez-Moreno, J., Martínez-Aran, A., Correa, P., ... Tabarés-Seisdedos, R. (2010). The switch from conventional to atypical antipsychotic treatment should not be based exclusively on the presence of cognitive deficits. A pilot study. *BMC Psychiatry*, *10*, 47. Retrieved from <http://www.biomedcentral.com/bmcp psychiatry>
- Schwartz, B.L., Rosse, R.B., & Deutsch, S.I. (1993). Limits of the processing view in accounting for dissociations among memory measures in a clinical population, *Memory and Cognition*, *21*, 63-72. Retrieved from <http://www.springer.com/psychology/cognitive+psychology/journal/13421>
- Shanks, D.R., Silverstein, S.M., Schenkel, L.S. Valone, C., & Nuernberger, S.W. (1998). Cognitive deficits and psychiatric rehabilitation outcomes in schizophrenia. *Psychiatric Quarterly*, *69*, 169-191. doi:10.1023/A:1022197109569

- Stone, W.S., & Hsi, X. (2011). Declarative memory deficits in schizophrenia: Problems and prospects. *Neurobiology of Learning and Memory*, 96(4), 544-552. doi:10.1016/j.nlm.2011.04.006
- Strauss, E., Sherman, E.M., & Spreen, O. (2006). *A compendium of neuropsychological tests: Administration, norms, and commentary* (3rd ed.). New York, NY: Oxford University Press.
- Sun, R., Slusarz, P., & Terry, C. (2005). The interaction of the explicit and implicit in skill learning: A dual-process approach. *Psychological Review*, 112, 159-192. doi:10.1037/0033-295X.112.1.159
- Tandon, R., Shipley, J.E., Greden, J.F., Mann, N.A., Eisner, W.H., & Goodson, J.A. (1991). Muscarinic cholinergic hyperactivity in schizophrenia. Relationship to positive and negative symptoms. *Schizophrenia Research*, 4(1), 23-30. Retrieved from <http://www.schres-journal.com/>
- Thompson, P.M., Vidal, C., Giedd, J.N., Gochman, P., Blumenthal, J., Nicolson, R., Toga, A.W., & Rapoport, J. (2001). Mapping adolescent brain change reveals dynamic wave of accelerated gray matter loss in very early-onset schizophrenia. *PNAS*, 98(20), 11650-11655. doi:10.1073/pnas.201243998
- Thorsen, C., Gustafsson, J.E., & Cliffordson, C. (2014). The influence of fluid knowledge and crystallized intelligence on the development of knowledge and skills. *British Journal of Educational Psychology*, 84(4), 556-570. doi:10.1111/bjep.12041
- Tilborg, I.A., Kessels, R.P., & Hulstijn, W. (2011). Learning by observation and guidance in patients with Alzheimer's dementia. *NeuroRehabilitation*, 29, 295-304. doi:10.3233/NRE-2011-0705
- Tramley, E.W., Doshi, R.R., Nayak, G.V., Palmer, B.W., Golshan, S., Heaton, R.K., ... Jeste, D.V. (2002). Generalized cognitive impairments, ability to perform everyday tasks, and level of independence in community living situations of older patients with psychosis. *American Journal of Psychiatry*, 159(12), 2013-2020. doi:10.1176/appi.ajp.159.12.2013
- Ucok, A., Polat, A., Cakir, S., & Genc, A. (2006). One year outcome in first episode schizophrenia: Predictors of relapse. *European Archives of Psychiatry and Clinical Neuroscience*, 256, 37-43. doi:10.1007/s00406-005-0598-2
- Van den Bos, G.R., & American Psychological Association. (2013). *APA Dictionary of Clinical Psychology*. Washington, DC: American Psychological Association.
- Venkatasubramanian, G., Chittiprol, S., Neelakantachar, N., Naveen, M.N., Thirthall, J., Gangadhar, B.N., & Shetty, K.T. (2007). Insulin and insulin-like growth factor-1 abnormalities in antipsychotic-naïve schizophrenia. *American Journal of Psychiatry*, 164(10), 1557-1560. Retrieved from <http://ajp.psychiatryonline.org>

- Ventura, J., Ered, A., Gretchen-Doorly, D., Subotnik, K.L., Horan, W.P., Helleman, G.S., & Nuechterlein, K.H. (2015). Theory of mind in the early course of schizophrenia: stability, symptom and neurocognitive correlates, and relationship with functioning. *Psychological Medicine*, *45*(10), 2031-43.
doi:10.1017/S0033291714003171
- Ventura, J., Green, M., Shaner, A., & Liberman, R.P. (1993). Training and quality assurance with the brief psychiatric rating scale: The drift busters. *International Journal of Methods in Psychiatric Research*, *3*, 221-244. Retrieved from <http://www.wiley.com/WileyCDA/WileyTitle/productCd-MPR2.html>
- Volkow, N.D., Wolf, A.P., Brodie, J.D., Cancro, R., Overall, J.E., Rhoades, H., & Van Gelder, P. (1988). Brain interactions in chronic schizophrenics under resting and activation conditions. *Schizophrenia Research*, *1*, 47-53. Retrieved from <http://www.schres-journal.com/>
- Walsh, T., McClellan, J.M., McCarthy, S.E., Addington, A.M., Pierce, S.B., Cooper, G.M., ... Sebat, J. (2008). Rare structural variants disrupt multiple genes in neurodevelopmental pathways in schizophrenia. *Science*, *320*, 539-543.
doi:10.1126/science.1155174
- Waters, F., Allen, P., Aleman, A., Fenryhough, C., Woodward, T.S., Badcock, J.C., ... Laro, F. (2012). Auditory hallucinations in schizophrenia and nonschizophrenia populations: A review and integrated model of cognitive mechanisms. *Schizophrenia Bulletin*, *38*, 683-692. doi:10.1093/schbul/sbs045
- Wechsler, D. (1997). *Wechsler Adult Intelligence Scale* (3rd ed.). San Antonio, TX: Harcourt Assessment.
- Wechsler, D. (2001). *The Wechsler Test of Adult Reading manual*. San Antonio, TX: Pearson.
- Wechsler, D. (2009). *The Wechsler Memory Scale-IV technical and interpretive manual*. San Antonio, TX: Pearson.
- Weickert, T.W., Goldberg, T.E., Gold, J.M., Bigelow, L.B., Egan, M.F., & Weinberg, D.R. (2000). Cognitive impairments in patients with schizophrenia displaying preserved and compromised intellect. *Archives of General Psychiatry*, *57*(9), 907-913. Retrieved from <http://archpsyc.jamanetwork.com/>
- Weinert, S. (2009). Implicit and explicit modes of learning: Similarities and differences from a developmental perspective. *Linguistics*, *47*, 241-271.
doi:10.1515/LING.2009.010
- Wessel, J.R., Haider, H., & Rose, M. (2012). The transition from implicit to explicit representations in incidental learning situations: More evidence from high frequency EEG coupling. *Experimental Brain Research*, *217*, 153-162.
doi:10.1007/s00221-011

- Wetmore, D.Z., Mukamel, E.A., & Schnitzer, M.J. (2007). Lock-and-key mechanisms of cerebellar memory recall based on rebound currents. *Journal of Neurophysiology*, *100*, 2328-2347. doi:10.1152/jn.00344.2007
- Wiersma, D., Nienhuis, F.J., Slooff, C.J., & Giel, R. (1998). Natural course of schizophrenic disorders: A 15-year follow-up of a Dutch incidence cohort. *Schizophrenia Bulletin*, *24*(1), 75-85. Retrieved from <http://schizophreniabulletin.oxfordjournals.org>
- Wilk, C.M., Gold, J.M., McMahon, R.P., Humber, K., Iannone, V.N., & Buchanan, R.W. (2005). No, it is not possible to be schizophrenic yet neuropsychologically normal. *Neuropsychology*, *19*(6), 778-786. doi:10.1037/0894-4105.19.6.778
- Wilkinson, G.S., & Robertson, G.J. (2006). *Wide Range Achievement Test* (4th ed.). Lutz, FL: Psychological Assessment Resources.
- Woodward, N.D., Tibbo, P., & Purdon, S.E. (2007). An fMRI investigation of procedural learning in unaffected siblings of individuals with schizophrenia. *Schizophrenia Research*, *94*(1-3), 306-216. doi:10.1016/j.schres.2007.04.026
- Wozniak, J. R., Block, E. E., White, T., Jensen, J. B., & Schulz, C. (2008). Clinical and neurocognitive course in early-onset psychosis: A longitudinal study of adolescents with schizophrenia-spectrum disorder. *Early Intervention in Psychiatry*, *2*, 169-177. doi:10.1111/j.1751-7893.2008.00075.x
- Xie, Q., Gao, X., & King, R.B. (2013). Thinking styles in implicit and explicit learning. *Learning and Individual Differences*, *23*, 267-271. Retrieved from <http://www.scimagojr.com/journalsearch.php?q=13453&tip=sid>
- Zabala, A., Rapado, M., Arango, C., Robles, O., de la Serna, E., Gonzalez, C., ... Bombin, I. (2010). Neuropsychological functioning in early-onset first-episode psychosis: Comparison of diagnostic subgroups. *European Archives of Psychiatry and Clinical Neuroscience*, *260*, 225-233. doi:10.1007/s00406-009-0046-9.
- Zakzanis, K.K. (1998). Neuropsychological correlates of positive vs. negative schizophrenia symptomatology. *Schizophrenia Research*, *29*(23), 227-233. Retrieved from <http://www.schres-journal.com>

Appendix A: Informed Consent (Participant)



Informed Consent (Form A)

Project Title: Learning and Schizophrenia: Neurocognition and symptom severity

Primary Investigator: Camilla S. Seippel, M.A.

Dissertation Chair: Henry V. Soper, PhD

You volunteered to be a participant in this research investigation, to understand more about the neuropsychology of schizophrenia. The researcher of this study is Ms. Camilla S Seippel, MA. Ms. Seippel is completing this study to fulfill her requirement to complete a formal research project as a dissertation at Antioch University Santa Barbara.

Background Information

The purpose this investigation to contribute to the current literature and knowledge of schizophrenia, so to develop further insight into the relationship between symptoms of thought disorders and different thinking processes. It is proposed that this investigation will improve treatment efficacy by contributing to the literature on treatment approaches in thought disorders.

Procedures

In your agreement to participate in this study, you agree to answer questions on the questionnaire and the paper-and-pencil assessment battery. Responding to the sociodemographic questionnaire and test battery should take you approximately 2-2.5 hours. Your participation is anonymous, and your responses will be kept confidential.

Voluntary Nature of the Study

Participation in this study is voluntary. You may refuse to enter, or withdraw from the investigation at any time without any consequence. Your decision to participate or withdraw will not affect your current patient status with the County of Orange, or affect the current, or future services you receive from the County of Orange. You are free to refuse to answer any of the questions in the investigation without any consequences. However, the more detailed and honest information you provide, the more complete the results of the study will be. You may quit at any time up until the end of the session. After that time, no information can be deleted because we won't be able to tell which responses are yours.

Risks and Benefits of Participation

There are effectively no risks to taking part in this research study. You may experience fatigue during the meeting with the investigator, or following the meeting. In the event that that you experience any distress or discomfort as a result of your participation in this investigation, you are referred to your county care coordinator for support.

This investigation is of a research nature and will not offer direct benefits to you. However, on the day of testing, there will be drinking water available for the participant, and they will be rewarded a \$20 "Health & Wellness" Walmart gift card for their participation. The indirect benefit to the participant from their participation in this study is that it will contribute to the knowledge that currently exists in the literature on the schizophrenia disorder, and it's etiology, neuropsychological profiles, and treatment efficacy.

Information about the study was discussed with me by the researcher, Camilla Seippel, M.A. If I have further questions I can contact her at xxx@xxxxxxl.xxx.

Confidentiality

The records of this study will be kept private, and safely stored where only the researcher, Camilla Seippel, will have access to the records. Data collected during this study will be shared and discussed with dissertation committee members. The researcher also intends to include the data and results of the study in future scholarly publications and presentations. However, no participant identification information will be made public. Our confidentiality agreement, as articulated above, will be effective in all cases of data sharing.

Limits to Confidentiality

Your participation in this study will remain confidential within the limits of the law.

Participant Name

Participant Signature

Date

Researcher Name

Researcher Signature

Date

Appendix B: Informed Consent (Conservator)



Informed Consent (Form B)

Project Title: Learning and Schizophrenia: Neurocognition and symptom severity

Primary Investigator: Camilla S. Seippel, M.A.

Dissertation Chair: Henry V. Soper, PhD

The participant, (Name of Participant) volunteered to be a participant in this research investigation, to understand more about the neuropsychology of schizophrenia. The researcher of this study is Ms. Camilla S Seippel, MA. Ms. Seippel is completing this study to fulfill her requirement to complete a formal research project as a dissertation at Antioch University Santa Barbara.

Background Information

The purpose this investigation to contribute to the current literature and knowledge of schizophrenia, so to develop further insight into the relationship between symptoms of thought disorders and different thinking processes. It is proposed that this investigation will improve treatment efficacy by contributing to the literature on treatment approaches in thought disorders.

Procedures

In the participant's agreement to participate in this study, you agree to answer questions on the questionnaire and the paper-and-pencil assessment battery. Responding to the sociodemographic questionnaire and test battery should take the participant

approximately 2-2.5 hours. Participation is anonymous, and the participant's responses will be kept confidential.

Voluntary Nature of the Study

Participation in this study is voluntary. The participant may refuse to enter, or withdraw from the investigation at any time without any consequence. The participant's decision to participate or withdraw will not affect his/her current patient status with the County of Orange, or affect the current, or future services they receive from the County of Orange. The participant is free to refuse to answer any of the questions in the investigation without any consequences. However, the more detailed and honest information provided by the participant, the more complete the results of the study will be. The participant may quit at any time up until the end of the session. After that time, no information can be deleted because we won't be able to tell which responses belong to the participant.

Risks and Benefits of Participation

There are effectively no risks to taking part in this research study. The participant may experience fatigue during the meeting with the investigator, or following the meeting. In the event that that the participant experiences any distress or discomfort as a result of their participation in this investigation, they are referred to their county care coordinator for support.

This investigation is of a research nature and will not offer direct benefits to the participant. However, on the day of testing, there will be drinking water available for the participant, and they will be rewarded a \$20 "Health & Wellness" Walmart gift card for their participation. The indirect benefit to the participant from their participation in this

study is that it will contribute to the knowledge that currently exists in the literature on the schizophrenia disorder, and it's etiology, neuropsychological profiles, and treatment efficacy.

Information about the study was discussed with me by the researcher, Camilla Seippel, M.A. If I have further questions I can contact her at xxxx@xxxxx.xxx.

Confidentiality

The records of this study will be kept private, and safely stored where only the researcher, Camilla Seippel, will have access to the records. Data collected during this study will be shared and discussed with dissertation committee members. The researcher also intends to include the data and results of the study in future scholarly publications and presentations. However, no participant identification information will be made public. Our confidentiality agreement, as articulated above, will be effective in all cases of data sharing.

Limits to Confidentiality

The participant's participation in this study will remain confidential within the limits of the law.

Conservator's Name

Conservator's Signature

Date

Researcher Name

Researcher Signature

Date

Appendix C: Informed Assent (Participant)**Informed Assent**

Project Title: Learning and Schizophrenia: Neurocognition and symptom severity

Primary Investigator: Camilla S. Seippel, M.A.

Dissertation Chair: Henry V. Soper, PhD

You volunteered to be a participant in this research investigation, to understand more about the neuropsychology of schizophrenia. The researcher of this study is Ms. Camilla S Seippel, MA. Ms. Seippel is completing this study to fulfill her requirement to complete a formal research project as a dissertation at Antioch University Santa Barbara.

Background Information

The purpose this investigation to contribute to the current literature and knowledge of schizophrenia, so to develop further insight into the relationship between symptoms of thought disorders and different thinking processes. It is proposed that this investigation will improve treatment efficacy by contributing to the literature on treatment approaches in thought disorders.

Procedures

In your agreement to participate in this study, you agree to answer questions on the questionnaire and the paper-and-pencil assessment battery. Responding to the

sociodemographic questionnaire and test battery should take you approximately 2-2.5 hours. Your participation is anonymous, and your responses will be kept confidential.

Voluntary Nature of the Study

Participation in this study is voluntary. You may refuse to enter, or withdraw from the investigation at any time without any consequence. Your decision to participate or withdraw will not affect your current patient status with the County of Orange, or affect the current, or future services you receive from the County of Orange. You are free to refuse to answer any of the questions in the investigation without any consequences. However, the more detailed and honest information you provide, the more complete the results of the study will be. You may quit at any time up until the end of the session. After that time, no information can be deleted because we won't be able to tell which responses are yours.

Risks and Benefits of Participation

There are effectively no risks to taking part in this research study. You may experience fatigue during the meeting with the investigator, or following the meeting. In the event that that you experience any distress or discomfort as a result of your participation in this investigation, you are referred to your care coordinator for support.

This investigation is of a research nature and will not offer direct benefits to you. However, on the day of testing, there will be drinking water available for the participant, and they will be rewarded a 10 "Health & Wellness" Walmart gift card for their participation. The indirect benefit to the participant from their participation in this study is that it will contribute to the knowledge that currently exists in the literature on the

schizophrenia disorder, and it's etiology, neuropsychological profiles, and treatment efficacy.

Information about the study was discussed with me by the researcher Camilla Seippel, M.A. If I have further questions I can contact her primary investigator at xxxx@xxxxx.xxx.

Confidentiality

The records of this study will be kept private, and safely stored where only the researcher, Camilla Seippel, will have access to the records. Data collected during this study will be shared and discussed with dissertation committee members. The researcher also intends to include the data and results of the study in future scholarly publications and presentations. However, no participant identification information will be made public. Our confidentiality agreement, as articulated above, will be effective in all cases of data sharing.

Limits to Confidentiality

Your participation in this study will remain confidential within the limits of the law.

Participant Name

Participant Signature

Date

Researcher Name

Researcher Signature

Date

II. Finger Tapping Test

Group # ____

Participant ID #: _____

Dominant Hand (R/L)

Trial #	Raw Score	T-Score

Non-Dominant Hand (R/L)

Trial #	Raw Score	T-Score

Copyright 2017 Camilla Seippel

All rights reserved

III. Boston-Naming Test (15 Item-I)

Item	Response
1. Bed (Item 1)	
2. Flower (Item 8)	
3. Helicopter (Item 11)	
4. Mushroom (Item 14)	
5. Camel (Item 17)	
6. Seahorse (Item 24)	
7. Globe (Item 27)	
8. Harmonica (Item 30)	
9. Igloo (Item 33)	
10. Knocker (Item 40)	
11. Pyramid (Item 43)	
12. Funnel (Item 46)	
13. Asparagus (Item 49)	
14. Yoke (Item 56)	
15. Trellis (Item 57)	
Total Correct	

Copyright 2017 Camilla Seippel

All rights reserved

IV. Digit-Symbol-Search Incidental

Trial	Raw Score	T-Score
Immediate		
P.		
F.R.		

Copyright 2017 Camilla Seippel

All rights reserved

V. Verbal Fluency Paragraphs

Fluency Paragraphs

Animals

I am going to name some places where you can see animals: the zoo, the jungle, a shelter, the safari, the farm, the ocean, and in a river. You might also find different types of animals in different climates or countries such as Australia, the North Pole, Alaska. Some animals live in the wild and some are domesticated, and some are farmed. Animals come in all different sizes from very small to very large. When I say “Go” I want you to tell me as many animals as you can think of. You will have 60 seconds, and try not to repeat any animals, as repetitions will not count. Are you ready? “Go.”

Clothes

I am going to name some occasions and places where you might wear different types of clothes: at work, on a summer vacation, in the water, in the snow, in a thunderstorm, at a party, or for playing different sports. Other times your might wear specific clothes is during specific seasons such as fall, winter, spring or summer. Clothes come in different styles and colors, and some clothes are made for boys, some for girls, some for men, and some for women. When I say “Go” I want you to tell me as many different items of clothing you can think of. You will have 60 seconds, and try not to repeat any items, as they will not count. Are you ready? “Go.”

Neutral Paragraphs

One can find lots of pieces of furniture to decorate a house. Things like couches, dinning tables, and sofas will often come in handy. Other times where one might find themselves looking for furniture include shopping for office furniture, office chairs, desks, and desk lamps are essentials for comfortable office settings. Sometimes people choose furniture based on it’s color, and other times based on its style. Furniture comes in many shapes and sizes, and is very useful for many reasons.

Appendix E: Participant Recruitment Material/Script

A STUDY OF THOUGHT DISORDER

I am a doctorate student collecting data on individuals who have received a thought disorder diagnosis, specifically as schizophrenia diagnosis at some time in their life. My study has been approved by, the Antioch University, and the County of Orange IRB committee's. You are invited to participate in the research project if you meet this criteria, which is being conducted by me, Camilla S. Seippel, PsyD, under the direction of Henry Soper, PhD,. The purpose of this study is to further understanding of thought and cognition in thought disorder.

Background of the study: The purpose of this research is to contribute to the current literature and knowledge of schizophrenia, so to develop further insight into the relationship between symptoms of thought disorders and different thinking processes. It is proposed that this investigation will improve treatment efficacy by contributing to the literature on treatment approaches in thought disorders.

Participation is Voluntary: Their participation in this study is completely voluntary in nature, and there are no consequences to choosing not to participate in this study. Your participation will contribute to learning about thought disorders. If participants have had a brain injury, or have another medical disorder affecting cognitive abilities, or who's primary language is not English may result in their being excluded from participating in the study. This will be further screened on the demographic questionnaire on the day of the trial. In this case participants will be thanked, but will not receive compensation.

What is expected of the participant: The participant and researcher will arrange a time, day, and location to meet. During the meeting, participants will be expected to provide approximately 2-2.5 hours of their time. Participants will be able to request a 10-15 minute break during the testing, as needed. At the conclusion of the meeting, participants will either be rewarded a 10 "Health & Wellness" Walmart gift card, or will have the card mailed to their conservator, depending on their conservatorship status.

The trial: Participants will be asked to answer some demographic questions, and provide responses on some paper-and-pencil exercises.

Benefits to the participant: There will be water available for the participant throughout the testing. Participants will also be awarded a 10 “Health & Wellness” Walmart gift card that will either be given directly to the participant at the conclusion of the study, or sent to their conservator (if participant is on conservatorship) at the conclusion of the study. Participants will receive a bus pass if needed for any transportation required to and from the trial on the scheduled day of testing.

Thank you note to Participant

I, Camilla Seippel, M.A. and doctoral candidate want to thank you for your willingness to participate in this study. It is my goal that this study will contribute to the current literature on symptom severity and thinking in schizophrenia. My primary objective in pursuing this study is to increase awareness of cognitive impairment as it relates to symptoms in schizophrenia. Increasing quality of life for, as well as protecting, individuals with thought disorder is my motivating force behind pursuing this study. The members of my dissertation committee are respected experts in the field of neuropsychology, and neuropsychology research with individuals with Schizophrenia. Individuals who choose to participate in this study will be protected and will be exposed to effectively no risks as a result of their participation.

Also, if you should have any questions, or would like more information, please contact the researcher directly at email: xxxx@xxxxx.xxx, or phone number [phone number redacted].

The dissertation chair may also be contacted by e-mail: [email address redacted].

Sincerely,
Camilla

Appendix F: Sociodemographic Questionnaire

Participant Identification #: _____

(Begin Here; Please mark "X" for all that apply to you).

1. Age: _____

2. Gender: Male _____ Female _____

3. Racial/ethnic background. Please choose one category that best captures how you see yourself.

African-American _____. Asian _____. Biracial _____. Black-Hispanic/Latino _____.

Middle Eastern _____. Native American _____. Pacific Islander _____. White/Caucasian _____.

Other- (please specify)

4. Is English your primary language? Yes _____ No _____

5. What is the highest grade in school you completed: _____

5b. If college, how many years of college have you completed: _____

5c. If post-secondary education, how many years completed: _____

How many years of other post-12th grade education have you completed: (Please

Describe)

6. What medications are you currently taking? (please indicate the name of the medication, the dosage amount and how many times a day you take your medications.)

Medication	Dosage (mg)	Frequency

7. Have you ever had a brain injury? ___ Yes ___ No. If Yes, did you lose consciousness for more than 10 minutes? ___ Yes ___ No.

8. Which is your dominant/preferred hand: Right: _____. Left: _____.

Appendix G: Ethical assurances

THIS FORM IS TO BE COMPLETED BEFORE RESEARCH BEGINS

Insuring Informed Consent of Participants in Research: Questions to be answered by AUSB Researchers

Form B-Ethical Assurances

The following questions are included in the research proposal.

1. Are your proposed participants capable of giving informed consent? Are the persons in your research population in a free-choice situation?...or are they constrained by age or other factors that limit their capacity to choose? For example, are they adults, or students who might be beholden to the institution in which they are enrolled, or prisoners, or children, or mentally or emotionally disabled? How will they be recruited? Does the inducement to participate significantly reduce their ability to choose freely or not to participate?

Participants that will be recruited for this study are considered member of a vulnerable population. These individuals are considered to have a mentally disabling condition. It will be made clear to potential participants that their choice to participate in the study is completely voluntary in nature. Participants will voluntarily contact the experimenter to participate in the study. There will be no consequences to the participant if he/she chooses not to participate in this study. Some of the participants may not be able to give informed consent and may be on conservatorship. In such cases, the participant's conservator will be provided an informed consent form to sign, and participants will be asked to sign the informed assent. Both of these documents are included in the proposal, and attached to the IRB application as Appendix C: Form B – Informed Consent for Conservator, and Appendix C: Form C - Informed Assent. Participants who are able to give informed consent will be asked to sign the participant informed consent prior on the day of testing, this form is included in the proposal in Appendix C: Form A - Informed Consent.

2. How are your participants to be involved in the study?

Following contact by participant, the researcher will provide the participant with an individual testing date, time, and location. Forty-six participants will be recruited for this study. At the beginning of the meeting participants will be briefed on the nature of the study, expectations of their participation, and then be asked to give informed consent. Once the participant provides informed consent or informed assent, the researcher will ask the participant will be administered a paper-and-pencil neurobehavioral test battery. Data will be collected on the data collection forms included in Appendix D: Form B – Data collection forms. The expected time to complete the sociodemographic question and the test battery is 2-2.5 hours. The tests included in this battery include the COWAT, FTT, Digit-Symbol Coding Incidental test, BNT-15-I, WRAT-4-Word Reading, and the BPRS. Once the battery has been administered, this examiner will collect participant

responses on the sociodemographic questionnaire, Appendix D: Form A - Sociodemographic Questionnaire. The exact amount of time required to complete the test battery will depend on the speed at which the participant is able to complete the questionnaire and answer the test questions.

What are the potential risks – physical, psychological, social, legal, or other? If you feel your participants will experience “no known risks” of any kind, indicate why you believe this to be so. If your methods do create potential risks, say why other methods you have considered were rejected in favor of the method chosen.

There are minimal risks of participating in this study because the procedure involves the participant providing paper-and-pencil responses. However the participant may experience fatigue or emotional upset during the meeting. If this should be the case, this researcher will make sure to clarify to the participant at the beginning of the session that that she/she is able to request a break during the testing protocol. Also, participants will be referred back to their care coordinator, or the referring party, for assistance in processing any negative feelings that may come up as a result of their participation. Initially, a more comprehensive battery was considered for this research study, however due to the nature of the population and potential for test fatigue, the final battery included in this study was selected.

3. What procedures, including procedures to safeguard confidentiality, are you using to protect against or minimize potential risks, and how will you assess the effectiveness of those procedures?

The records of this study will be kept private, and safely stored where only the primary investigator will have access to the records. Data collected during this investigation will be shared, and discussed with dissertation committee members. I also intend to include the data and results of the study, but not participant identifiable information, in future scholarly publications and presentations. The participant will be informed of the limits to confidentiality at the beginning of the session, and will be listed in the both forms of the Informed Consent, conservator and participant, as well as in the Informed Assent. However, no participant identification information will be made public, and no copyright material will be published or included in the dissertation paper.

In order to safeguard confidentiality of the participant’s identifiable information, and their anonymity, participants will be assigned a code number at the beginning of the testing meeting. This code number will be used collectively on the sociodemographic questionnaire, and for identifying all test forms and results of that participant. The code number will be placed in the upper right corner of each response form. At the end of each session the primary investigator will transport the test response packet containing the Informed Consent, Sociodemographic Questionnaire and the Test Battery Response Forms, in a locked box to a second locked box for permanent storage. These lock boxes will remain in the custody of the primary investigator, who will be the only individual with access to the lock boxes. The Informed Consent Forms will be removed, and stored in a lock box separate from the Sociodemographic and Test Battery Response Forms. At

the conclusion of each testing session data will be transferred into electronic form. This information, and responses to the sociodemographic questionnaire will be stored on a flashdrive. The uploaded demographic materials and testing data will be contained in separate folders on the flashdrive. Each individual data set and information will be identifiable only through assigned coded numbers. The flashdrive will be stored in the previously indicated lock box, that will also contain the Sociodemographic and Test Battery Response Forms. The electronic data developed during the process of data integration will be stored in encrypted documents. These documents will be password protected, and stored on the flashdrive, protected with a password. Only the primary researcher will have access to the passwords required to access the flashdrive and the confidential data files. All material contained in these lock boxes will be kept locked, and stored by this examiner throughout the duration of the study, and for an additional seven years following the conclusion of this study. At the conclusion of seven years, the paper records will be shredded, and the flashdrive will be deleted.

In the event that that they experience any distress or discomfort as a result of their participation in this investigation, participants will be referred to their county care coordinator for psychological support.

4. Have you obtained (or will you obtain) consent from your participants in writing?

Participants who are able to give informed consent will be have the informed consent explained to them, including the limitations to confidentiality, and will be asked to sign Informed Consent A form. This will be done at the beginning of the testing session. Participants who are unable to give informed consent and who are on conservatorship at the time of testing will have the informed consent explained to them informed consent, and will be asked to sign the Informed Assent C form at the beginning of the session. Additionally, for these participants informed consent will have the informed consent explained to the conservator, and they will be asked to sign the Informed consent form B (Appendix C) prior to the session day.

5. What are the benefits to society, and to your participants that will accrue from your investigation?

This investigation is of a research nature and will not offer direct benefits to the participant. However, there will be water available for the participant on the day of testing, and participants will be rewarded with 10 “Health & Wellness” Walmart gift card. The gift card will be mailed directly to the conservator in cases in which the participant is under conservatorship. The indirect benefit resulting from subject participation in this study is that their partaking will contribute to the literature on the relationship of symptom severity and thought to cognition in schizophrenia. Ultimately, potentially contributing to treatment approach and efficacy in this population.

6. Do you judge that the benefits justify the risks in your proposed research?
Indicate why. Both the student and his/her Dissertation Chair must sign this form
and submit it

As indicated previously, there are effectively no risks to participating in this study. Better understanding of this relationship may contribute to the current knowledge of neurocognition in schizophrenia. Thus, this researcher judges that the methods selected for this study are justified.

Date: _____ Signed: _____ Student

Date: _____ Signed: _____ Dissertation Chair