

INFORMATION TO USERS

This manuscript has been reproduced from the microfilm master. UMI films the text directly from the original or copy submitted. Thus, some thesis and dissertation copies are in typewriter face, while others may be from any type of computer printer.

The quality of this reproduction is dependent upon the quality of the copy submitted. Broken or indistinct print, colored or poor quality illustrations and photographs, print bleedthrough, substandard margins, and improper alignment can adversely affect reproduction.

In the unlikely event that the author did not send UMI a complete manuscript and there are missing pages, these will be noted. Also, if unauthorized copyright material had to be removed, a note will indicate the deletion.

Oversize materials (e.g., maps, drawings, charts) are reproduced by sectioning the original, beginning at the upper left-hand corner and continuing from left to right in equal sections with small overlaps. Each original is also photographed in one exposure and is included in reduced form at the back of the book.

Photographs included in the original manuscript have been reproduced xerographically in this copy. Higher quality 6" x 9" black and white photographic prints are available for any photographs or illustrations appearing in this copy for an additional charge. Contact UMI directly to order.

UMI

A Bell & Howell Information Company
300 North Zeeb Road, Ann Arbor MI 48106-1346 USA
313/761-4700 800/521-0600

**Initiation and Inhibition Deficits, Syndromes,
and Cognitive Competency in Schizophrenia**

by

Karin Maria Christensen
B.A., McGill University, 1989
M.Sc., University of Victoria, 1992

**A Dissertation Submitted in Partial Fulfillment of the
Requirements for the Degree of**

DOCTOR OF PHILOSOPHY

in the Department of Psychology

**We accept this dissertation as conforming
to the required standard**

Dr. Catherine A. Mateer, Supervisor (Department of Psychology)

Dr. Michael A. Hunter, Departmental Member (Department of Psychology)

Dr. Holly Tuokko, Departmental Member (Department of Psychology)

Dr. Roger E. Graves, Departmental Member (Department of Psychology)

Dr. Rennie Warburton, Outside Member (Department of Sociology)

Dr. Richard Williams, Additional Member (Royal Jubilee Hospital, Victoria Mental Health Centre)

Dr. Peter F. Liddle, External Examiner (Department of Psychiatry, University of British Columbia)

© Karin Maria Christensen, 1998
University of Victoria

All rights reserved. This dissertation may not be reproduced in whole or in part, by photocopying or other means, without the permission of the author.

Supervisor: Dr. Catherine A. Mateer

Abstract

Deficits in attention, memory, and executive functioning have been associated with schizophrenia. Neuropsychological functioning is considered to bridge neuropathology and clinical symptoms in this illness. However, relatively few studies have investigated the connections between specific cognitive deficits and other variables. "Psychomotor poverty" and "disorganization" are two of the three schizophrenia syndromes. Previous work suggests that neuropsychological deficits in the initiation and inhibition of thoughts and actions underlie psychomotor poverty and disorganization, respectively. Part of this study aimed to replicate and extend these findings. Unstructured tasks were used, because they elicit impairments in executive functioning.

The present study also examined the effects of neuropsychological functioning and syndromes on overall cognitive competency. Cognitive competency refers to the integrity of cognitive skills important for independent functioning in everyday life. Though often neglected, cognitive competency is an important functional outcome. It was hypothesized that both psychomotor poverty and disorganization would predict reduced cognitive competency.

Participants were 40 in- and outpatients, aged 17 to 51 years, with a current diagnosis of schizophrenia according to DSM-IV criteria. A psychiatrist rated symptomatology using the Scale for the Assessment of Negative Symptoms and the Scale for the Assessment of Positive Symptoms. Two unstructured neuropsychological tasks were administered: a verbal description of a picture and the Tinkertoy test. Using these tasks, measures of initiation and inhibition capacity were developed; these revealed good interrater reliability. The Cognitive Competency Test used simulated situations to evaluate cognition in areas that affect everyday functioning. Finally, a brief insight measure was included for exploratory analyses.

Data were analyzed within the framework of a causal model. Initiation capacity failed to predict the psychomotor poverty syndrome. One indicator of disinhibition - intermingling - predicted the disorganization syndrome. Disorganization mediated the impact of intermingling on cognitive competency. In contrast, initiation capacity affected cognitive competency

directly. Insight correlated with the disorganization syndrome, and contributed to the prediction of cognitive competency. Initiation, disorganization syndrome, and insight, combined, accounted for 58% of the variance in cognitive competency; each variable contributed uniquely. Implications include suggestions as to which deficits and syndromes should be targeted for remediation, to improve patients' independent functioning.

Examiners:

Dr. Catherine A. Mateer, Supervisor (Department of Psychology)

Dr. Michael A. Hunter, Departmental Member (Department of Psychology)

Dr. Holly Tuokko, Departmental Member (Department of Psychology)

Dr. Roger E. Graves, Departmental Member (Department of Psychology)

Dr. Rennie Warburton, Outside Member (Department of Sociology)

Dr. Richard Williams, Additional Member (Royal Jubilee Hospital, Victoria Mental Health Centre)

Dr. Peter F. Liddle, External Examiner (Department of Psychiatry, University of British Columbia)

Table of Contents

Abstract	ii
Table of Contents	iv
List of Tables	viii
List of Figures	ix
Acknowledgements	x
Dedication	xi
Introduction	1
Brain Abnormalities in Schizophrenia	1
Classification Schemes	2
Levels of Understanding in Schizophrenia	3
Neuropsychology of Schizophrenic Syndromes	6
Studies of Schizophrenic Syndromes and General Neuro- psychological Performance	6
Speculations about Localization of Syndromes	7
Relationship of Schizophrenic Syndromes to Indices of Frontal Lobe Malfunction	10
Patterns of Cerebral Malfunction Associated with Schizophrenic Syndromes	12
Clinical Significance of Psychomotor Poverty and Disorganization Syndromes	13
Cognitive Competency in Schizophrenia	14
Significance	14
Definitions	15
Review of Relevant Research	15
Unawareness of Illness in Schizophrenia	18
Etiology	19

Consequences	20
Overview of Present Research.....	21
First Aim	21
Proposed Measure of Initiation	23
Proposed Measure of Inhibition.....	24
Hypothesis 1	24
Hypothesis 2	24
Second Aim	25
Hypothesis 3	25
Method	27
Participants	27
Procedures and Materials	28
Ratings of Psychomotor Poverty and Disorganization	
Syndromes	28
Neuropsychological Measures	29
Interrater Reliability	35
Development of Measures.....	35
Test of Reliability	36
Preliminary Considerations Regarding Data.....	40
Plan of Analysis	40
Hypotheses	40
Influence of Awareness of Illness	42
Results	43
Summary of Main Findings	43
Confirmation of Three-Factor Symptom Structure	43
Longer Item Set	43
Shorter Item Set	44
Hypotheses	50
Hypothesis 1	50

Hypothesis 2	50
Hypothesis 3	52
Awareness of Illness	54
Predicting Cognitive Competency	55
Influence of Sex	57
Influence of Anticholinergic Medications	57
Discussion	58
Three-Syndrome Model of Schizophrenia	58
Initiation Capacity Fails to Predict Psychomotor Poverty Syndrome	58
Initiation, But Not Psychomotor Poverty Syndrome, Predicts Cognitive Competency	61
Intermingling Predicts the Disorganization Syndrome	62
The Disorganization Syndrome Mediates the Effect of Intermingling on Cognitive Competency	64
Unawareness of Illness is Associated with the Disorganization Syndrome.....	65
Unawareness of Illness Predicts Reduced Cognitive Competency	66
Implications	67
Representativeness of the Sample	68
Directions for Future Research	69
References	71
Appendix A: Research Study Information Form	81
Appendix B: Informed Consent Form	82
Appendix C: Test Reliability and Validity Data	83
Appendix D: Scales for the Assessment of Negative and Positive Symptoms (SANS & SAPS)	85
Appendix E: Guidelines for Scoring MHP Transcripts	89
Appendix F: Guidelines for Tinkertoy Test Scoring	92
Appendix G: Cognitive Competency Test Record Sheet	93

Appendix H: Awareness of Illness Questionnaire	97
Appendix I: Descriptive Information for Psychometric and Demographic Measures.....	99
Appendix J: Intercorrelations of Psychometric Measures	100

List of Tables

Table 1.	Syndromes Resulting from Brain Injury Which Resemble Chronic Schizophrenic Syndromes, and Neural Systems Implicated	9
Table 2.	Development Phase Intercorrelations Between Raters for Picture Description and Tinkertoy Test	38
Table 3.	Test Phase Intercorrelations Between Raters for Picture Description and Tinkertoy Test	39
Table 4.	Component Loadings Resulting From Principal Component Analysis of Longer SANS & SAPS Item Set	46
Table 5.	Component Loadings Resulting From Principal Component Analysis of SANS & SAPS Items Used for Scoring.....	49

List of Figures

Figure 1. Levels of understanding in schizophrenia.....4

Figure 2. Proposed causal model.....26

Figure 3. Models used to test mediation41

Figure 4. Models used to test mediation, with standardized regression coefficients53

Figure 5. Revised causal model, with standardized regression coefficients56

Acknowledgements

This dissertation was a collaborative effort involving the support of many people. Each and every one of them helped to make the dissertation process a pleasant one. I would like to thank the members of my (large) supervisory committee for their contributions: Katy Mateer, for her consistent ideas, support, and encouragement as my supervisor; Richard Williams, for his tireless efforts toward subject recruiting and psychiatric ratings, and for consultation as an expert in schizophrenia; Mike Hunter, Holly Tuokko, Roger Graves, and Rennie Warburton, for their helpful comments and suggestions throughout this project.

I am very appreciative of the work of two wonderful research assistants. Thanks to Christine Schwartz for her meticulous scoring of transcripts and videos; for help with library research and copying; and for her ready smile. Thanks to Todd Woodward for his generous statistical consulting and data analysis, and his endless patience with all my questions.

I am also indebted to the nursing and office staff at the Eric Martin Pavilion and the Victoria Mental Health Centre, for their congenial help arranging meetings with participants and gathering information from files. I especially thank Lizanne Walsh and Jasmine Sharp for their roles in this research.

I am grateful to Todd for being such a complete and loving companion throughout this and every "project" I undertake in life; and to Gail Woodward, for her love and support via angels, dinners, and tarot readings. I have also appreciated the closeness and encouragement of all my friends scattered around the world. Finally and most importantly, I thank my family for instilling in me self-confidence, and the understanding that good things can take time. Without these qualities, I could not have undertaken to become a psychologist. To Mom, Far, Michael, Lisa, Evie, and the memory of my grandparents, thank you for your constant love and support.

Financial support for this work from the following sources is gratefully acknowledged: a research grant from the Dr. Norma Calder Schizophrenia Foundation; the Sara Spencer Research Award in Applied Social Sciences; the Robert and Douglas Vickery Graduate Award; and a research grant from Janssen-Ortho Inc.

Dedication

This dissertation is dedicated to all people
living with schizophrenia.

INTRODUCTION

Schizophrenia will affect approximately one person of every 100 at some point in their life (American Psychiatric Association [APA], 1994). Prevalence rates are similar throughout the world, and the disorder is equally common in both sexes. A diagnosis of schizophrenia implies significant deterioration in social, occupational, and intellectual functioning. Furthermore, the disease most commonly manifests itself around the transitional time when a young adult is in the process of establishing a career and developing meaningful relationships. Although the course is variable, complete remission seems uncommon and many affected individuals suffer significant lifelong impairments.

Brain Abnormalities in Schizophrenia

Early this century, Kraepelin speculated that the brain regions primarily affected in schizophrenia might be the frontal and temporal lobes (1913/1919; cited in Randolph, Goldberg, & Weinberger, 1993). Although numerous parts of the human brain have been considered for a prominent role in schizophrenia, it is now evident that the frontal lobes are indeed especially relevant. Evidence of structural and functional abnormalities in the prefrontal lobes has accumulated. These prefrontal pathologies include maldevelopment of neuronal architecture, lateral ventricular enlargement, cortical atrophy, decreased CT density, reduced neuronal activity as measured by cerebral blood flow and positron emission tomography, specific failure to activate the prefrontal lobes during task performance requiring prefrontal contribution, and neuropsychological test performance indicative of prefrontal dysfunction. Overall, *structural* abnormalities are inconsistently reported and are not specific to schizophrenia; the evidence of disordered frontal lobe *function* is more convincing (Williamson, 1987). For reviews, see Weinberger, Aloia, Goldberg, & Berman, 1994, and Williamson, 1987.

Although referred to less frequently than the frontal lobes, the temporal lobes have also been implicated in the neuropathology of schizophrenia (Bogerts, Meertz, & Schonfeldt-Bausch, 1985; Suddath, Christison, Torrey, Casanova, & Weinberger, 1990). It has been

suggested that the available evidence supports Kraepelin's early formulations, i.e., that both medial temporal and (dorsolateral) prefrontal cortical systems are implicated in the pathophysiology of schizophrenia (Randolph et al., 1993).

Classification Schemes

Schizophrenia encompasses a wide variety of clinical presentations. Since Kraepelin, who was the first to recognize schizophrenia as a distinct illness, clinicians and researchers have sought ways in which to divide schizophrenia into subtypes. The classical subdivision into paranoid, hebephrenic, catatonic, and simple schizophrenia (e.g., International Classification of Diseases, tenth edition; WHO, 1993) has met with limited success, suggesting that a different approach is required. Crow (1980) proposed two pathological processes, called Type 1 and Type 2, which he felt reflected a single illness and could co-exist within individuals. Crow suggested that Type 1 is manifest as positive symptoms (delusions, hallucinations, formal thought disorder), which reflect a biochemical imbalance. In contrast, Type 2 is considered to consist of negative symptoms (poverty of speech, flat affect) and to involve structural brain abnormalities.

Crow's formulation has been successful in guiding research into the heterogeneous nature of schizophrenia. However, empirical evidence has provided only partial support for this model. For example, the fact that Type I tends to be acute and Type II more chronic raises the issue of whether it is the symptoms or the degree of chronicity that is the distinguishing factor. To address such issues, Liddle (1987b) performed a factor analysis on the symptoms of a group of chronic schizophrenic patients with stable symptoms. He found that schizophrenic symptoms segregated into three syndromes, which he designated "psychomotor poverty" (the classic negative symptoms: poverty of speech, blunted affect, decreased spontaneous movement); "disorganization" (inappropriate affect and disorders of thought form); and "reality distortion" (delusions and hallucinations). Similar factor structures have since been reported in many other studies, including studies of non-chronic patients (Arndt, Alliger, & Andreasen, 1991; Peralta, de Leon, & Cuesta, 1992; Thompson

& Meltzer, 1993). Thus, there is a substantial body of evidence suggesting that the symptoms of schizophrenia segregate into three major syndromes. The available evidence suggests that these syndromes are independent, and that patients can show evidence of more than one syndrome at a given time (Allen, Liddle, & Frith, 1993; Liddle, 1987b; Liddle & Morris, 1991). Therefore, the syndromes do not represent separate illnesses. Rather, they are distinguishable but overlapping pathological processes within schizophrenia.

Levels of Understanding in Schizophrenia

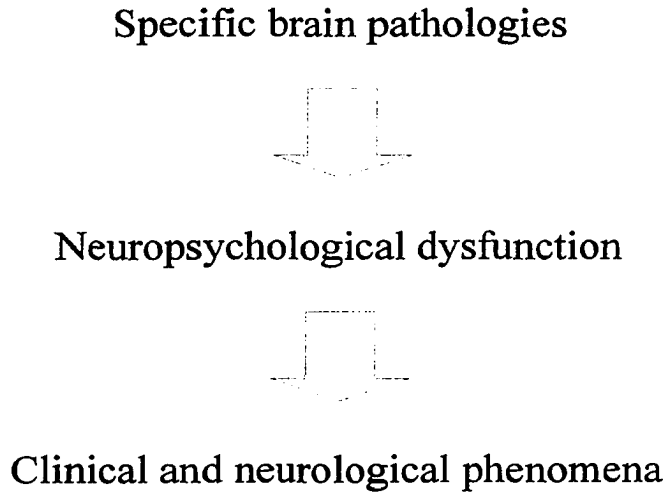
Despite intensive research, little has been learned that helps to elucidate the etiology and pathophysiology of schizophrenia. It is widely believed that the pathology observed in schizophrenia can only be understood through clarification of how its abnormalities at different levels interact with each other. This concept is referred to as the "levels of understanding" model (see Frith, 1992, p.13; Mortimer, 1992; Mortimer & McKenna, 1994).

Mortimer (1992) reviewed two years of schizophrenia research in a psychiatric journal, and reported that most studies investigated either the structure and basic functioning of the brain, or the phenomenology of schizophrenia (i.e., the precise description of psychopathology). Few studies focused on neuropsychology, and few studies used any combination of methods (e.g., investigating links between basic brain sciences and neuropsychological functioning). Thus, the body of research appeared fragmented into disparate, narrow, unconnected approaches.

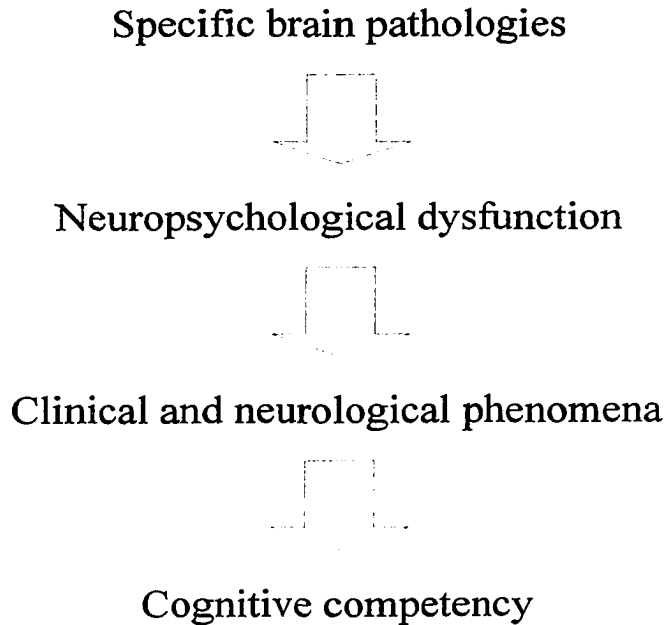
Despite the difficulties inherent in attempting to link approaches, it appears more profitable to use research strategies in which at least two of these levels are combined, in a manner designed to shed light on their interconnections. Figure 1a illustrates a three-level model of schizophrenia, in which the levels are levels of understanding, and the interconnections between levels need to be elucidated. Note that the level of explanation intermediate between neuropathology and symptoms is the neuropsychological (cognitive) level. Indeed, specific cognitive deficits have, in recent years, been identified in people with schizophrenia. These are mainly in the domains of executive functions, memory, and

Figure 1. Levels of understanding in schizophrenia.

a. Three levels of understanding in schizophrenia (Mortimer, 1992).



b. Four levels of understanding in schizophrenia.



attention (Goldsamt, Barros, Schwartz, Weinstein, & Naveed (1993); Mortimer & McKenna, 1994; Randolph et al., 1993). The next appropriate research task is the identification of those aspects of neuropsychology, or cognitive functioning, that have the capability to link neuroscience (brain variables) with phenomenology (symptoms; Mortimer & McKenna, 1994).

The levels of understanding approach rests upon two key propositions. The first is that studying "schizophrenia" as one entity is not fruitful. The diagnostic criteria are subject to variation over time (e.g., changes from the Diagnostic and Statistical Manual of Mental Disorders [DSM]-III-R to DSM-IV [APA, 1987, 1994]), and depending on the diagnostic system being used (e.g., Research Diagnostic Criteria vs. DSM). Also, the reliability and validity of some diagnostic systems for schizophrenia are not favourable (Costello, 1993). In addition, because the disorder is so heterogeneous (regardless of the diagnostic system), studies that investigate "schizophrenia" as a single entity inevitably yield very mixed groups in terms of symptoms, as well as associated structural and functional brain variables. Therefore, whatever the dependent variables of interest, potentially significant trends are likely to be overshadowed. A focus on symptoms has been advocated (Costello, 1993; Frith, 1992) as an aid in determining what causal mechanisms may be involved in various aspects of the illness.

The second proposition is that attempting to link clinical presentation with brain dysfunction is futile. The gap between these two levels is too vast, and the neuropsychological level should be investigated since it forms the bridge between them (Mortimer & McKenna, 1994). Thus, research should focus on elucidating the neuropsychological features underlying specific schizophrenic symptoms, or groups of symptoms (i.e., syndromes) within the disorder. These findings can then be related to brain variables. As stated by David (1992), an attempt to construct a neuropsychology of psychiatric disorders "...must go beyond crude localisationism and instead define psychiatric phenomena in terms of a breakdown or malfunction of psychological processes." (p. 246)

The levels of understanding model, as presented by Mortimer, ends at the level of symptoms. However, if one wanted to address the overall impact of schizophrenia on basic

cognitive functioning, another level could be added: cognitive competency (Figure 1b). Cognitive competency will be defined below, and evidence will be presented in favour of investigating this level of functioning in schizophrenia. Furthermore, such an investigation will be incorporated into this study.

Neuropsychology of Schizophrenic Syndromes

Studies of Schizophrenic Syndromes and General Neuropsychological Performance

Each of the three syndromes has been associated with a specific pattern of neuropsychological deficits. Given a battery of neuropsychological tests, Liddle (1987a) found that patients with the psychomotor poverty syndrome evidenced difficulties in abstract reasoning, object naming, and long-term memory. The disorganization syndrome was associated with poor concentration and difficulty learning new information. Reality distortion, in contrast, showed very little association with neuropsychological impairment, being only weakly correlated with poor figure-ground perception. Frith, Leary, Cahill, & Johnstone (1991) found that symptoms associated with the psychomotor poverty and disorganization syndromes correlated with cognitive impairments such as a decline in IQ and poor source memory for words. However, consistent with Liddle's (1987a) results, positive symptomatology was not related to cognitive impairments.

Other studies have investigated the relationships of "negative" and "positive" symptoms to neuropsychological measures. These designations closely correspond to the psychomotor poverty and reality distortion syndromes, as defined by Liddle. One such study reported that participants with more severe negative symptoms tended to generate fewer words on a fluency task, to make more perseverative errors on the Wisconsin Card Sorting Test (WCST), to have reduced right-hand tapping speed, to require more time for Trails A of the Trail Making Test, and to recall fewer words on the Rey Auditory Verbal Learning Test. The only significant association for subjects with more positive symptoms was that they made fewer perseverative errors on the WCST (Hammer, Katsanis, & Iacono, 1995). Basso, Nasrallah, Olson, and Bornstein (1998) reported that negative and disorganized

symptoms (the latter being defined mainly as thought disorder and bizarre behaviour) predicted verbal, performance, and full-scale IQ, as well as concentration and delayed memory indices, and sensory and motor functions. Negative symptoms also predicted executive functioning, including measures of abstraction, verbal fluency, and alternating attention. Psychotic symptoms were associated with increased speed of visual search and more correct responses on a card-sorting task.

The investigations reviewed above are uniform in reporting many more neuropsychological impairments in patients with psychomotor poverty/negative and disorganization symptoms than in patients with reality distortion/positive symptoms.

Speculations about Localization of Syndromes

The association of each syndrome with a characteristic pattern of neuropsychological deficits suggests that the three syndromes are associated with three different types of brain dysfunction. This hypothesis was supported when Liddle (1987a) compared the syndromes' clinical features to the sequelae of focal brain injury. Indeed, psychomotor poverty and disorganization bear some resemblance to two distinct syndromes produced by frontal lobe injury. Blumer and Benson (1975) designated these syndromes "pseudodepression" and "pseudopsychopathy":

Patients who suffer the "pseudodepressed" type of frontal lobe personality alteration appear to have lost all initiative. They respond in an automaton-like fashion, but the responses are usually proper and intelligent...Slowness, indifference, and apathy are the personality traits...The "pseudopsychopathic" type of frontal lobe personality is best characterized by the lack of adult tact and restraints. Such patients may be coarse, irritable, facetious...they often lack social graces and may, on impulse, commit anti-social acts. (pp. 157-158)

Clinically, psychomotor poverty has been described as resembling the pseudodepression syndrome, and disorganization as similar to pseudopsychopathy (Liddle, 1987a; Liddle, Barnes, Morris, & Haque, 1989). Blumer and Benson proposed that pseudodepression arises from lesions to the dorsolateral prefrontal regions, certain related subcortical structures, and

their connections, while the pseudopsychopathic syndrome stems from injury to the orbital frontal lobes or related pathways. The resemblance between the schizophrenic syndromes and Blumer and Benson's frontal syndromes suggest that psychomotor poverty and disorganization might reflect dysfunction of dorsolateral and mediobasal prefrontal cortex, respectively. Liddle (1987a) notes that the intellectual impairments documented in patients with psychomotor poverty and disorganization are consistent with these localizations. The following lines of evidence provide additional support for this hypothesis.

There is abundant evidence (reviewed by Liddle, 1988; cited in Liddle et al., 1989) indicating the presence of two separate but overlapping prefrontal-subcortical-hippocampal neural systems in mammals. One system involves dorsal structures, and the other involves ventral structures. The dorsal system is important for temporal structuring of behaviour in monkeys, and also appears to play a role in the initiation of activity in humans. The ventral system is concerned with the inhibition of interference from extraneous influences (Liddle et al., 1989). Disruption of these two systems is reflected in the clinical syndromes of pseudodepression and pseudopsychopathy, because these syndromes are known to be associated with dorsal prefrontal and orbitofrontal injuries, respectively.

Quite recently, a series of five parallel frontal-subcortical anatomical circuits has been described, to which a wide variety of behavioural alterations can be linked (see Cummings, 1993). Lesions to component structures of two of these circuits, the dorsolateral prefrontal circuit and the orbitofrontal circuit, result in behavioural symptoms that correspond, at least to a degree, to the two syndromes described by Blumer and Benson as a result of damage to specific prefrontal cortical regions. Specifically, lesions in the dorsolateral prefrontal circuit result in "executive dysfunction" (difficulty in generating hypotheses and in maintaining and shifting sets; reduced fluency; and poor organization) and motor programming deficits. Lesions in the orbitofrontal circuit lead to disinhibition, irritability, and inappropriate behaviour. Table 1 illustrates all of these associations. Thus, theoretically it appears likely that the schizophrenic syndromes of psychomotor poverty and disorganization may reflect dysfunction within the dorsolateral prefrontal and the orbitofrontal circuits, respectively.

Table 1

Syndromes Resulting From Brain Injury Which Resemble Chronic Schizophrenic Syndromes, and Neural Systems Implicated

Syndromes due to brain injury	Schizophrenic syndrome	Neural system
<p>Pseudodepression (Dorsal prefrontal)</p> <ul style="list-style-type: none"> loss of initiative flat affect slowness apathy 	<p>Psychomotor poverty</p> <ul style="list-style-type: none"> poverty of speech flat affect decreased spontaneous movement 	<p>Dorsolateral prefrontal circuit</p> <ul style="list-style-type: none"> poor hypothesis generation poor set maintenance/shifting reduced fluency poor organization
<p>Pseudopsychopathy (Orbital prefrontal)</p> <ul style="list-style-type: none"> lack of tact and restraint irritability, impulsivity garrulous speech facetiousness 	<p>Disorganization</p> <ul style="list-style-type: none"> inappropriate affect disorders of the form of thought poverty of content of speech 	<p>Orbitofrontal circuit</p> <ul style="list-style-type: none"> disinhibition irritability inappropriate behaviour

Not all investigations have supported the views above. For example, Norman et al. (1997) failed to find the expected associations between (a) psychomotor poverty and impaired performance on tests reflecting dorsolateral prefrontal functioning, and (b) disorganization and deficits on tests reflecting mediobasal prefrontal functioning. However, results were influenced by less-than-optimal choice of measures; did provide some indirect support for the hypotheses; and were consistent with there being different cortical-subcortical circuits associated with the two syndromes.

In contrast to these two syndromes that seem to involve prefrontal disturbance, the reality distortion syndrome has been described as resembling the interictal psychosis of temporal lobe epilepsy, which is characterized by hallucinations and delusions but little affective flattening or formal thought disorder (Parnas & Korsgaard, 1982; cited in Liddle et al., 1989). The reported correlation between the reality distortion syndrome and poor figure-ground perception (Liddle, 1987a) is consistent with the finding that temporal lobe lesions cause deficits in selective attention (Kolb & Wishaw, 1980, p. 407). Norman et al. (1997) confirmed the association of reality distortion with tests sensitive to temporal lobe functioning (i.e., verbal memory). Despite these findings, there is also evidence suggesting that the cognitive defect underlying hallucinations and delusions is in a more frontally-mediated ability, namely self-monitoring of thoughts and actions (Frith & Done, 1988; Frith & Done, 1989; Frith, 1992; Mlakar, Jensterle, & Frith, 1994). This issue remains to be resolved by further research.

Relationship of Schizophrenic Syndromes to Indices of Frontal Lobe Malfunction

Several studies have investigated the association of the three syndromes with purported measures of frontal lobe functioning, and these results have further illuminated the neuropsychology of schizophrenic syndromes. Both psychomotor poverty and disorganization are consistently related to deficits on neuropsychological tests sensitive to frontal lobe damage. Psychomotor poverty is associated with slowed performance on tasks requiring the generation of a plan to act. For example, these patients show reduced output on word generation tasks (Allen, Liddle, & Frith, 1993; Frith et al., 1991; Hammer, Katsanis,

& Iacono, 1995; Liddle & Morris, 1991). Allen et al. (1993) provided evidence that schizophrenic adults with the psychomotor poverty syndrome have intact lexicons (i.e., they have as many words available in semantic memory as normal controls), and that the deficit is specifically in the generation of a plan to retrieve the stored words. It has further been demonstrated that the verbal fluency impairment in people with the psychomotor poverty syndrome cannot be accounted for entirely by slowness in articulating words (Liddle & Morris, 1991). This was taken to support the hypothesis that the cognitive mechanism underlying the psychomotor poverty syndrome involves a slowing of mental activity, which affects not only articulation, but also the generation of words.

The disorganization syndrome is associated with deficient suppression of inappropriate responses. Thus, disorganization is correlated with impaired performance on the Stroop test interference task (Liddle & Morris, 1991), the production of inappropriate words on verbal fluency tasks (Allen et al., 1993), and poor inhibition of inappropriate responses during a continuous performance test (Frith et al., 1991). Liddle and Morris (1991) summarized the reason for deficits on the Stroop test, Trail Making Test B, and a card sorting test as difficulty inhibiting an established response and employing a different strategy.

In contrast to psychomotor poverty and disorganization, reality distortion is not associated with impairment on tests of frontal lobe functioning, at least not in a way discerned by available measures (Frith et al., 1991; Liddle & Morris, 1991; Royall et al., 1993). Nonetheless, there is some evidence suggesting that this syndrome reflects a defect in the internal monitoring of self-generated mental or physical activity (Frith, 1992; Frith & Done, 1988, 1989; Mlakar, Jensterle, & Frith, 1994). For example, patients with Schneiderian symptoms (such as delusions of alien control and of thought insertion) were found to have difficulty keeping track of their performance and remembering what actions they had made, while drawing without immediate visual feedback. This difficulty increased in proportion to the reliance on central monitoring (Mlakar et al., 1994). There are also data implicating a sensory gating deficit (Judd et al., 1992).

Overall, then, neuropsychological findings indicate that the three characteristic schizophrenic syndromes - psychomotor poverty, disorganization, and reality distortion -

reflect impairments of the supervisory mental processes responsible for the initiation, selection, and monitoring of self-generated mental activity, respectively (Liddle, 1995). The importance of defective *supervisory* mental functions is consistent with Kraepelin's clinical observations that schizophrenic patients exhibited difficulties specifically when performing tasks that involved novel experiences, independent mental activity, and the overcoming of difficulties (Kraepelin, 1913/1919; cited in Liddle, 1995). The capacity to adaptively perform goal-oriented behaviour in novel situations, as opposed to the production of responses that are largely elicited by circumstances, is a hallmark of the "executive functions," considered to be localized in the frontal, especially prefrontal, lobes (Lezak, 1983, pp. 507-508). Zec (1995, p. 221) provided evidence that Kraepelin documented numerous executive functioning deficits in schizophrenia, which, because they were observed during the preneuroleptic era, could not be a side effect of neuroleptic treatment. The "weakening and disjointing of volition," which Kraepelin regarded as the most characteristic features of schizophrenia, seem to underlie the psychomotor poverty and disorganization syndromes. Furthermore, these proposed major deficits are consistent with neuropsychological evidence that two of the primary impairments in schizophrenia are deficient initiation of a plan to act and deficient suppression of inappropriate mental activity (Liddle, 1995).

Patterns of Cerebral Malfunction Associated with Schizophrenic Syndromes

Individuals with schizophrenia show a specific failure to activate the prefrontal lobes during performance of the Wisconsin Card Sorting Task (Weinberger, Berman, & Zec, 1986). This task is a measure of hypothesis generation and set shifting, which is consistently found to be impaired in schizophrenia (Weinberger & Berman, 1996). More recently, a trend toward impaired temporal lobe activation during memory tasks was also shown (Liddle, 1996).

Data from neuropsychological testing of schizophrenic patients suggested to Liddle (1987a) that the two core syndromes reflect dysfunction at different frontal-lobe sites. Specifically, dorsolateral prefrontal cortex (PFC) dysfunction was proposed to underlie the psychomotor poverty syndrome, and mediobasal PFC dysfunction was proposed to be

associated with the disorganization syndrome. The reality distortion syndrome was felt to reflect temporal lobe dysfunction. It was further noted that psychomotor poverty appeared to be associated with left-hemisphere dysfunction, whereas disorganization seemed related to right-hemisphere dysfunction. Using positron emission tomography to study the relationship between regional cerebral blood flow and symptom profiles, Liddle et al. (1992) confirmed the predictions above: psychomotor poverty correlated negatively with activity in the left dorsolateral PFC; disorganization correlated with hypoperfusion in the right ventral PFC; and reality distortion correlated positively with blood flow in the left parahippocampal region. Furthermore, this study demonstrated that abnormalities of brain function are not confined to single loci, but involve the neural networks discussed previously (i.e., frontal-subcortical circuits). Work to date also suggests that it is imbalances between neural activity in various interconnected brain areas, rather than aberrant function at individual locations, that are responsible for the observed psychopathology. For example, the disorganization syndrome involves not only decreased blood flow in the right ventral PFC, but also increases in the right medial PFC and the contiguous anterior cingulate and thalamus. (For a review of functional imaging in schizophrenia, see Liddle, 1996.)

Clinical Significance of Psychomotor Poverty and Disorganization Syndromes

It has been noted that the negative symptoms of schizophrenia account for a substantial amount of the morbidity associated with the disorder (APA, 1994). Relative to hallucinations and delusions, negative symptoms are resistant to the effects of antipsychotic medications, tending to persist between acute illness episodes (APA, 1994; Pogue-Geile, 1989). There is some evidence suggesting that negative symptoms might become increasingly prominent in some individuals over the course of the illness (APA, 1994). Furthermore, review of the research indicates that negative symptoms form part of a cluster of variables which co-occur quite consistently, and include generalized cognitive impairment (Frith et al., 1991; Hammer, Katsanis, & Iacono, 1995; Randolph et al., 1993), deficits on tests of frontal lobe functioning (Hammer et al., 1995), hypofrontality (Weinberger et al., 1994), poor premorbid functioning (Randolph et al., 1993; Walker et al., 1993), inpatient

status (Frith et al., 1991), lower educational achievement (Randolph et al., 1993), lower premorbid IQ (Frith et al., 1991), ventricular enlargement, and structural brain changes in general (for a review of structural and functional brain changes in relation to symptomatology, see Rubin, 1994).

Despite the obvious clinical and prognostic importance of negative symptoms, schizophrenia research has tended to focus on studying the mechanisms underlying the classical positive symptoms (e.g., hallucinations, delusions, incoherence of speech). There have been few studies investigating processes underlying negative symptoms. This imbalance resulted from a diagnostic focus on Schneider's "first-rank" (positive) symptoms. However, in the last two decades there has been a return to Kraepelinian thinking, which ascribes primary importance to negative symptoms. Interest in researching negative symptoms has increased correspondingly. In addition, the recent research reviewed above suggests that the features of the disorganization syndrome, a newer concept, represent the second most important characteristic of schizophrenia.

Investigations of the cognitive processes underlying negative symptoms (or psychomotor poverty) have proven fruitful, as have studies of the neuropsychological mechanisms associated with the disorganization syndrome. Part of this study will focus on further elucidating the cognitive deficits underlying these two syndromes.

Cognitive Competency in Schizophrenia

Significance

Green (1998) provides the following introduction to a section on neurocognition and functional outcome in schizophrenia:

Once one becomes aware of the scope of neurocognitive deficits in schizophrenia, it is natural to wonder how these deficits affect the way patients function in their daily life. Do the deficits make it difficult for patients to catch the right bus, prepare their dinner, or keep their job? Do they make it hard for patients to hold a conversation with a family member or to remember to take their medication? Despite

a long tradition of research on the nature of neurocognitive deficits in schizophrenia, these fundamental questions have been largely ignored. (p.149)

To address this neglected and critical issue, this study incorporates a measure of cognitive competency as a specific type of functional outcome measure. As shown in Figure 1b, for the purposes of this research, cognitive competency is considered the end point of the cognitive and syndromal variables described above.

Definitions

Mental competency, in a medico-legal context, generally can be defined as the ability to understand and to knowingly act upon information provided in certain situations (Wang & Ennis, 1986). Two kinds of competency can be distinguished: 1) cognitive competency (possessing the necessary intellectual skills to, for example, manage finances or personal affairs - "cognitive skills for living" [Wang & Ennis, 1986; p.120]); and 2) mental competency in the psychiatric sense (possessing adequate reality testing abilities, independent of cognitive competency). While the two types of competency are dissociable, it is clear that in certain disorders, such as schizophrenia, each type may be impaired. During an acute episode with delusions and hallucinations, contact with reality is clearly lost. During the remission or residual phases of schizophrenia, when negative symptoms often persist, reality testing may be intact but intellectual deficits remain, as evidenced by neuropsychological test results from such patients. The focus of the present study is on cognitive competency, a multidimensional construct that is subject to variation (Wang & Ennis, 1986).

Review of Relevant Research

It is apparent from the literature that there has been a dearth of research specific to cognitive competency in schizophrenia. One difficulty in reviewing the relevant literature is that there is no clear differentiation between "cognitive competency" and functional competency in terms of capacity to live independently, or to make rational decisions, etc. These constructs must be related; however, there are differences. Although some types of

competency could be affected by a broad range of impairments, including physical handicaps, "cognitive competency" suggests a focus on the cognitive abilities that support such life skills as rational decision-making and independent living. Clearly, several cognitive domains would be implicated. It appears that most relevant research has focused on a level slightly more removed from the basic cognitive skills: either the capacity to make rational decisions or the capacity to live safely and independently.

Despite the lack of research on cognitive competency in schizophrenia, there are countless studies focusing on social competency or skills. These represent other (conceivably related) types of functional outcome measures. Some authors have reported associations between symptoms and specific competencies. For example, several studies have reported that impaired social functioning is associated with negative symptoms (Bellack, Morrison, Mueser, & Wade, 1989; Jackson et al., 1989). Assuming that normal social functioning requires certain underlying cognitive abilities, one might predict that patients with negative symptoms would also evidence weaknesses in the types of cognitive skills that might subservise competency in interpersonal situations. As elaborated below, there is also reason to expect that patients with the disorganization syndrome would show impaired cognitive competency.

Given the data from studies of Liddle's three syndromes, showing that both psychomotor poverty and disorganization are associated with specific neuropsychological deficits in executive functions, one would expect both of these syndromes to predict reduced cognitive competency. The important link might be between the executive deficits, not the syndromes supposedly caused by them, and impaired cognitive competency. The paragraphs below support this prediction.

First, it is widely recognized that individuals may show quite intact performance on many neuropsychological tests, and yet suffer significant functional handicaps. In such cases, the disruptions in their social/occupational functioning or decisional capacity often appear to be caused by executive dysfunction, confirmed by careful testing (Bayless, Varney, & Roberts, 1989; Schindler, Ramchandani, Matthews, & Podell, 1995). Supporting this view of the importance of frontal lobe integrity to adaptive functioning, it has been noted that

although global dementing processes are generally responsible for impaired decisional capacity, focal frontal lobe dementia is increasingly being recognized as a cause of severely impaired functional abilities (Schindler et al., 1995). Furthermore, executive or frontal-lobe functions are generally considered to give rise to our most advanced, uniquely human capacities (Benson, 1993), and to be responsible for the most important cognitive declines seen with aging and frontal-lobe damage (Dempster, 1992).

Second, some competency research has been carried out in the dementia population, and results from these studies help to inform hypotheses about cognitive competency in schizophrenia. For example, Marson, Cody, Ingram, and Harrell (1995) administered to normal elderly and Alzheimer's patients a battery of neuropsychological tests representing cognitive domains linked theoretically to the competency to consent to treatment. Results showed that word fluency measures (phonemic and semantic) predicted both the intact competency performance of controls and the impaired performance of patients. A word fluency measure also emerged as the best predictor of competency status, correctly classifying 82% of all subjects. Word fluency was a superior predictor compared to dementia severity, verbal reasoning, and memory. Because word fluency has been shown to be frontally mediated, the findings were taken to suggest that frontal lobe functions underlie the ability to formulate rational reasons for a choice. Results were consistent with current views of the role of the prefrontal cortex in guiding decision-making in complex social milieus (Damasio, 1991). The authors stress that neuropsychological studies of competence have significant theoretical and clinical value (e.g., to specify the cognitive changes that threaten competency). They also indicate the need for examination of cognitive correlates of competency in other diagnostic groups, including psychiatric illnesses.

A recent article emphasized the fact that executive deficits affect the functional status of elderly patients as well as other clinical groups (Royall et al., 1993). This study compared young schizophrenic inpatients with elderly residents at three levels of care, and showed that the level of dependence was correlated with performance on the Executive Interview (EXIT), a measure of the behavioural sequelae of executive dyscontrol, and the Mini-Mental State Examination (MMSE). Furthermore, the EXIT discriminated subjects at each level of care,

was more sensitive than the more general MMSE, and correlated the strongest with level of care. Executive impairment had the same impact on functioning regardless of significant differences in diagnosis, age, sex, and neuroleptic use. These findings suggest that the degree of executive dysfunction within a disease may vary with functional status and vice versa. In other words, it may be the executive deficits, and not the symptoms specific to a disease, that are essential to the associated functional burdens. Further research was seen as important in clarifying the relationship between executive functions and symptoms.

The studies reviewed above suggest that executive dysfunction predicts impaired cognitive competency, in terms of constructs such as decisional capacity and need for care. As yet, the relationships between overall cognitive competency and aspects of schizophrenia have not been investigated. It would be interesting to determine whether the executive deficits in schizophrenia (decreased initiation and inhibition) affect cognitive competency indirectly, through the clinical syndromes which they underlie.

Unawareness of Illness in Schizophrenia

Unawareness of mental illness, or poor insight, is a prevalent feature of schizophrenia (Amador, Strauss, Yale, & Gorman, 1991; Amador et al., 1994). It is more severe and pervasive in schizophrenic patients than in patients with schizoaffective or major depressive disorders with or without psychosis (Amador et al., 1994). It appears that poor insight is not simply a consequence of greater overall severity of psychotic symptoms. Rather, poor insight seems to be uniquely characteristic of schizophrenia as compared to other psychotic disorders, and may help to distinguish schizophrenia from these other disorders. (For a review, see Amador & Kronengold, 1998.) Unawareness of illness in schizophrenia has been conceptualized in numerous ways. At one end of the spectrum, poor insight is understood as a psychological defense mechanism, whereas at the other extreme, it implies a cognitive deficit. Furthermore, poor insight is not a unitary construct. Subtypes of insight can be identified (e.g., retrospective vs. current insight) and some argue that insight comprises a variety of phenomena (for a review of these concepts, see Amador et al., 1991). Unawareness

of illness can be described as the situation in which an individual's perception of him/herself is grossly discrepant from that of the ambient community and culture (Amador et al., 1991). In the broadest sense, what has been described in schizophrenia is an apparent lack of awareness of the associated deficits, the consequences of the disorder, and the need for treatment (Amador et al., 1991).

Etiology

The etiology of awareness deficits in schizophrenia is poorly understood, as there have been no specific studies of the neurological substrates of unawareness in schizophrenia (in contrast, such research has been done for a variety of neurological disorders). Investigations of anosognosia have generally pointed to the right parietal area and the prefrontal lobes as being important for self-awareness (McGlynn & Schacter, 1989; Stuss & Benson, 1986). These models may be relevant to schizophrenia, since neurological signs and frontally mediated neuropsychological deficits have been documented (Amador et al., 1991). The available literature suggests that at least some forms of unawareness in schizophrenia are a direct result of the pathophysiology of the disease.

Unawareness is generally independent of symptom severity (Amador et al., 1993; McEvoy et al., 1989) and of medication effects, although weak associations have been noted between the severity of certain symptoms and decreased awareness of mental disorder (Amador et al., 1991; Amador et al., 1994). It has been suggested that unawareness may be a core expression of schizophrenia and thus could be useful in the neuropsychological understanding, diagnosis, and treatment of schizophrenia (Amador et al., 1991).

A few studies have examined the relationship between insight and performance on neuropsychological tests in schizophrenia. Young, Davila, and Scher (1993) reported a correlation between a measure of impairment of insight and performance on the WCST, a neuropsychological test sensitive to frontal lobe dysfunction. The authors concluded that these results support the hypothesis that in some cases, poor insight has an organic etiology, possibly related to frontal lobe impairment. Lysaker and Bell (1994) replicated this finding, and additionally reported that, when the effects of IQ were partialled out, subjects with

impaired insight demonstrated consistently poorer WCST performance over a period of one year than subjects with unimpaired insight. This finding was interpreted as suggesting that enduring cognitive deficits may underlie poor insight in schizophrenia. A subsequent study supported this conclusion by demonstrating that performance on certain cognitive measures, some of which are considered executive in nature, predicted the amount of improvement in insight, whereas psychosocial functioning and symptomatology did not (Lysaker & Bell, 1995).

Consequences

Despite difficulties in comparing across studies of awareness due to varying measures and definitions, the bulk of the evidence supports the idea that patients displaying awareness of their illness show better treatment compliance and clinical outcome (Amador et al., 1991). Furthermore, poor insight is associated with poorer psychosocial functioning (Amador et al., 1994) and also with functional impairments in work settings - specifically, deficient social skills and inappropriate personal presentation (Lysaker & Bell, 1995).

In summary, the available literature suggests that unawareness of illness in schizophrenia may have a complex relationship to other aspects of the illness. Some researchers have proposed that poor insight may be caused by enduring cognitive (specifically executive) deficits. However, it is conceivable that the reverse is also true, i.e., that being unaware of mental illness leads to deficits in certain cognitive domains. Research has additionally suggested that poor insight is associated with certain difficulties in living, such as problems in psychosocial and work-related skills, and poor treatment compliance. No studies to date have examined the relationship between awareness of illness and cognitive competency in schizophrenia. This relationship will be investigated in an exploratory manner in the present study.

Overview of Present Research

First Aim

The first aim was to extend previously reported findings relating performance on executive functioning tests to the clinical syndromes of psychomotor poverty and disorganization. As reviewed above, it has been quite well established using conventional tests that there are distinct executive deficits associated with the psychomotor poverty and disorganization syndromes. The pattern of test results suggests that poor initiation underlies the former syndrome, while poor inhibition underlies the latter. No study of schizophrenic syndromes has tested these hypotheses using non-conventional tests. However, there is reason to believe that it would be informative to test these hypotheses using *unstructured* neuropsychological tasks.

First, as pointed out by Lezak (1982), traditional neuropsychological examinations rarely allow the patient to demonstrate abilities other than what he/she is asked to do. Yet, to accurately assess the executive functions (including the initiation of behaviour and the inhibition of inappropriate responses), it is important to observe the patient's actions when required to act on his/her own behalf. In advocating for an unstructured task for these reasons, Lezak (1982) suggested the following:

Generally, the more open-ended and unstructured the task, the more likely will impairments in programming become evident. Thus, tests of verbal fluency (Lezak, 1976), free writing or drawing can be used to assess the patient's capacity to produce, maintain, and stop an intended series of responses at will. (p.290)

Second, at least two studies have demonstrated the usefulness of this approach in different patient populations. Lezak (1982) first used Tinkertoys as a neuropsychological measure. She asked patients to "make whatever you want" using 50 Tinkertoy pieces. Tinkertoys are children's play materials consisting of knobs, wheels, dowels, etc., of various sizes. The pieces can easily be combined to make virtually limitless constructions. Upon completion of their construction, patients can be asked to tell what they have made. The number of pieces used (NP) and the complexity of the construction (COMP) can be scored.

Lezak (1982) divided patients into dependent and non-dependent groups, based on their need for support and supervision. The groups did not differ in age, education, or WAIS Information subtest scores. A normal control group (younger and better educated) was also included. Results showed that both the NP and the COMP score differentiated the three groups from each other. Furthermore, nearly all the dependent patients used fewer than 23 pieces; non-dependent patients used 23 or more; and 50% of the controls used all 50 pieces. The study provided evidence that Tinkertoy test performance was not simply dependent upon cognitive abilities or constructional skills. Results suggested that patients who have difficulty initiating or carrying out purposive activities tend to use fewer pieces although their constructions may be recognizable and appropriately named. In contrast, those with deficient goal formulation or planning may use more pieces, but their constructions are more likely to be unnamed or inappropriate for their names. It was noted that some patients with extensive executive dysfunction evidenced both difficulties (i.e., used very few pieces to make unnamed or unplanned constructions). Pathologically inert patients were considered most likely to do nothing at all given the open-ended task of Tinkertoy construction.

Bayless, Varney, and Roberts (1989) administered the Tinkertoy test to a group of patients who had sustained closed-head injuries, half of whom had returned to work and half of whom had been unable to sustain employment as a result of their head injuries. They reported that whereas all but one of the patients able to return to work scored normally on the TT, nearly half of the non-returnees performed below the level of the lowest non-injured control subject. A subsequent study (Martzke, Swan, & Varney, 1991) administered a battery of tests considered especially sensitive to frontal lobe damage, which included the Tinkertoy Test, to a group of head-injured patients with indicators of orbitofrontal damage. Results revealed that the Tinkertoy Test was the only "frontal-lobe" test that was failed by a majority of the subjects.

Mendez and Ashla-Mendez (1991) tested the hypothesis that two unstructured tasks would distinguish between multi-infarct dementia (MID) and Alzheimer-type dementia (DAT) better than conventional structured tests. The groups were matched for the severity of dementia. Subjects were administered several conventional, more structured tests aimed

at assessing initiation, sustained attention, constructions, language, and memory. The unstructured tasks were a verbal description of the Cookie Theft picture from the Boston Diagnostic Aphasia Examination (Goodglass & Kaplan, 1972) - a verbal task, and the Tinkertoy test - a non-verbal task. The researchers reasoned that these tasks assess the initiation and execution of independent activity, free of the constraints implicit in more traditional tests. The number of words per minute, the number of pieces used, and the complexity of constructions were measured. As predicted, the unstructured tasks differentiated the dementia groups better than the conventional tests. The only conventional measures which discriminated the groups were certain memory measures (worse performance by the DAT group). On the unstructured tasks, the MID group scored worse than the DAT group, and both did worse than normal controls. These results make sense in light of the fact that MID involves frontal-subcortical pathology, which would impair the initiation and maintenance of behaviour. Although this study was interested mainly in poor production, there is also evidence that certain groups (i.e., schizophrenic patients with incoherence) produce relatively more inappropriate words than those with negative features (Allen, Liddle, & Frith, 1993).

The results of the above investigations argue in favour of using tasks such as verbal picture description and the Tinkertoy test as more sensitive measures of difficulties in behavioural initiation and maintenance, as well as inhibition capacities. Indeed, it appears that these unstructured tasks might highlight the types of executive difficulties described in the above studies, relative to many conventional tests. The present study applied these two tasks to the study of the cognitive processes presumed to underlie the psychomotor poverty syndrome (i.e., impaired initiation) and the disorganization syndrome (i.e., deficient inhibition of inappropriate responses). The tasks were used to provide information relevant to the hypotheses, in the following manner.

Proposed measure of initiation. Initiation capacity was defined as a latent factor, measured by four scores derived from unstructured tasks. Two of these were expected to have significant positive loadings on the initiation factor: the number of words produced on an unstructured verbal description task (the Mammoth Hunt Picture; described in Method

section) and the number of pieces used in an unstructured non-verbal construction task (the Tinkertoy test). Two other variables, the latency to respond on the picture description and the latency to respond on the Tinkertoy test, were expected to have significant negative loadings on initiation.

Proposed measure of inhibition. A latent factor reflecting inhibition capacity was proposed, measured by three scores derived from unstructured tasks. The number of inappropriate communications (defined in Method section) on the picture description, the number of inappropriate constructions (defined in Method section) on the Tinkertoy test, and the sum of three error types which appear to reflect disinhibition on the Tinkertoy test (defined in Method section) were each expected to have significant negative loadings on the inhibition factor.

Hypothesis 1. Poor ability to initiate self-generated thoughts and actions causes the psychomotor poverty syndrome. Thus, a negative relationship was expected between initiation capacity and the psychomotor poverty syndrome.

Although this hypothesis may at first appear to be a tautology, the question is considered valid because two different levels of understanding are being related. Specifically, a cognitive deficit is being proposed as a precursor to a group of clinical symptoms within schizophrenia. Furthermore, the psychomotor poverty syndrome is defined by features in addition to those clearly related to initiation difficulties (for example, decreased emotional responsivity would not necessarily be predicted as a result of poor behavioural initiation). That is, the syndrome appears to encompass a broader range of features than could be predicted based solely on the cognitive deficit involved. Similar to Mortimer's (1992) and Mortimer and McKenna's (1994) suggestions, Green (1998) also recommends viewing clinical presentation as a separate construct that might be related to, but not identical to, underlying neurocognition.

Hypothesis 2. Poor inhibition of inappropriate responses causes the disorganization syndrome. A negative relationship was expected between inhibition and the disorganization syndrome.

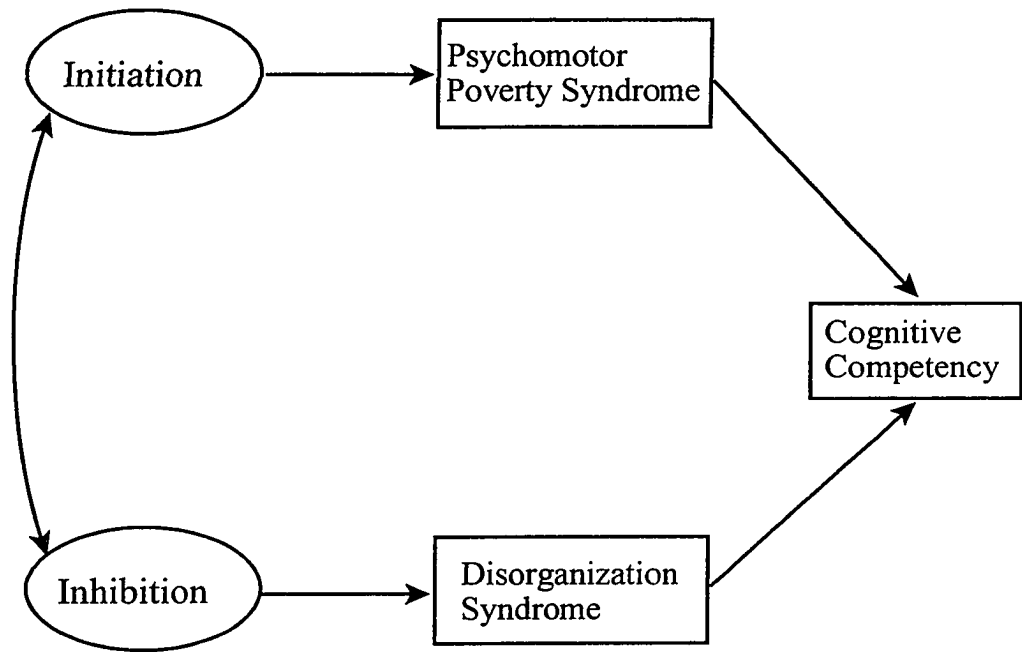
Second Aim

The second aim was to investigate the relationships between the two syndromes and cognitive competency.

Hypothesis 3. Both the psychomotor poverty syndrome and the disorganization syndrome are related to deficits in cognitive competency. Initiation and inhibition were expected to relate indirectly to cognitive competency, through the psychomotor poverty and disorganization syndromes, respectively. Such a model is called *mediational*: the clinical syndromes were expected to mediate the effects of initiation and inhibition on cognitive competency.

The model depicted in Figure 2 illustrates all of the hypotheses that were tested. The data were analyzed using multiple regression, within the framework of a causal model. Exploratory analyses were carried out to determine the effect of awareness of mental illness on the model.

Figure 2. Proposed causal model. Straight arrows represent hypotheses.



METHOD

Participants

Participants were 40 patients with schizophrenia (27 males and 13 females). Participants were currently being treated at either the Eric Martin Pavilion of the Royal Jubilee Hospital (in Victoria, B.C.) or the Victoria Mental Health Centre. All had a current diagnosis of schizophrenia based on DSM-IV criteria. Potential participants were excluded if they had another Axis I disorder concurrently, or if review of their medical file revealed a history of neurological disease or head injury. With one exception (who was being treated by another psychiatrist), all participants were patients of Dr. Richard Williams, a psychiatrist specializing in the treatment of schizophrenia.

Participants ranged in age from 17 to 51 (the mean age was 30.75 years). An upper age limit of 55 years was chosen so that performance on cognitive measures would not be confounded by the effects of aging. The mean years of education completed was 11.65. On an estimate of premorbid verbal intellectual ability (the Quick Test; described below), the mean score was 98.28. The sample included 11 inpatients and 29 outpatients. The mean duration of illness was 6.95 years. All participants were taking antipsychotic medications at the time of assessment, and 10 were taking anticholinergic medications in addition. Because the performance of inpatients on neuropsychological measures can be inconsistent due to acute psychotic symptoms, only patients whose condition was clinically stable, as judged by the treating psychiatrist, were included.

Participants were recruited through a written description of the study, specifying what their involvement would entail; risks and benefits; freedom to withdraw; confidentiality; etc. (Appendix A). This description was provided to patients by one of three people: Dr. Williams, following a routine treatment interview; the patient's case manager; or the author. Potential participants were asked whether the author might contact them at a later date, at which time they were free to accept or decline participation. People granting such permission were contacted. If, at that time, the patient was still interested in participating, a time for data collection was arranged. Prior to beginning testing, subjects read and signed an informed

consent form (Appendix B).

Procedures and Materials

Medication data, symptom ratings, and psychometric data were gathered on the same day or one day apart, so that they corresponded closely in time.

Ratings of psychomotor poverty and disorganization syndromes. These ratings were completed by Dr. Williams during each participant's regular meeting with him, at either the Eric Martin Pavilion or the Victoria Mental Health Centre. To measure current symptoms, the **Scale for the Assessment of Negative Symptoms (SANS;** Andreasen, 1983, 1989a, 1989b) and the **Scale for the Assessment of Positive Symptoms (SAPS;** Andreasen, 1984) were administered by Dr. Williams. Evidence of the scales' reliability and validity is provided in Appendix C. The SANS consists of five scales that evaluate five different aspects of negative symptoms: alogia, affective blunting, avolition-apathy, anhedonia-asociality, and attentional impairment. The SAPS consists of four scales considered to measure aspects of positive symptoms: hallucinations, delusions, bizarre behaviour, and positive formal thought disorder. Together, the two scales (shown in Appendix D) provide a comprehensive measure of schizophrenic symptoms. The SANS and SAPS were chosen for use in this study because previous findings indicate that the three schizophrenic syndromes - psychomotor poverty, disorganization, and reality distortion - can be derived from these scales (Liddle, 1987b; Liddle & Barnes, 1990; Liddle et al., 1992; Malla, Norman, Williamson, Cortese, & Diaz, 1993).

Scores for the psychomotor poverty and disorganization syndromes were calculated by summing the scores for the component symptoms. The item scores to be summed have been identified by factor analytic studies of schizophrenic symptomatology (Liddle, 1987b; Liddle & Barnes, 1990; Liddle et al., 1992; Malla et al., 1993) and have been used previously in this manner (Liddle, 1987a; Liddle & Morris, 1991). Although various methods of calculating syndrome scores tend to correlate highly with one another, the scoring method of Liddle was chosen because it is the most conservative and thus the most appropriate for initial analyses (R. M. G. Norman, personal communication, January 28, 1997). The score

for psychomotor poverty included four items which are aspects of blunting of affect, namely affective non-responsivity, unchanging facial expression, paucity of expressive gestures, and lack of vocal inflections. The mean of the scores for these four symptoms was calculated, and the psychomotor poverty syndrome score was obtained by adding this score for blunting of affect to the scores for poverty of speech and decreased spontaneous movement. The score for the disorganization syndrome was the sum of the scores for inappropriate affect, poverty of content of speech, and positive formal thought disorder. The reality distortion syndrome score, which was used only for confirmatory factor analyses and not as a variable, was the sum of the scores for voices commenting, persecutory delusions, and delusions of reference. Before proceeding with analyses using two of these syndrome scores as variables, we tested the hypothesis that the symptoms would reveal a three-factor solution, similar to previously reported factor solutions (see Results).

Neuropsychological measures. The author administered the neuropsychological assessment instruments in a quiet room within the treatment facility, with the participant sitting across from her at a table. Short breaks were provided as required for smoking, use of the bathroom, and avoidance of excessive fatigue (no participant required more than one break). The unstructured tasks were randomly assigned to first or last position relative to the other tests: thus, for half the participants, they were administered first, and for half, last. This manipulation was introduced to control for the potential effects of initial test anxiety early in the session vs. decreased inhibition once rapport has been established. Testing required approximately 1.5 hours.

1. An estimate of IQ was obtained using the **Quick Test (QT)**; Ammons & Ammons, 1962). The QT was designed to briefly screen "verbal-perceptual intelligence in practical situations." There are three forms, each comprised of 50 words. The patient is presented with a card depicting four different scenes, and is asked to choose the picture that best illustrates the meaning of a specific word. The vocabulary in the QT ranges from very simple and concrete to very difficult and requiring verbal abstraction. No verbal response is required, as the subject may point to the picture. Theoretically, this form of intelligence is considered resistant to acquired brain injury (Wang, 1990). Thus, the QT provides an indication of

premorbid verbal conceptual ability. At the very least, it is considered to supply information regarding the upper limits of verbal intellectual potential at the time of testing (Wang, 1990). Norms are provided so that one can determine both an IQ estimate and a mental age for the subject. The QT is viewed as resistant to the performance reductions on intelligence tests that can be associated with schizophrenic psychopathology (Dizzone and Davis, 1973). The QT has also been identified as a useful IQ estimate for patients with reduced ability to respond verbally (Dalton & Pederson, 1987), or to respond as required by other tests (Lambirth & Panek, 1982). Appendix C provides validity and reliability data.

2. The first unstructured task was a verbal description of the picture in form A of the Test of Written Language - 2 (TOWL-2; Hammill & Larsen, 1988). Although the TOWL-2 is designed to assess children's written language skills, this picture was chosen over others that have been used in adult populations and oral format, such as the Cookie Theft picture from the Boston Diagnostic Aphasia Examination (Goodglass & Kaplan, 1972), because (a) it presents less stereotyped gender roles, and (b) it was considered to be more interesting and complex, and hence more likely to elicit speech. The picture depicts a prehistoric mammoth hunt scene, and will be referred to as the **Mammoth Hunt Picture (MHP)**. Although of questionable relevance here (due to the written administration format), reliability data for the TOWL-2 subtest scores that include the MHP have been reported by Hammill and Larsen (1988; see Appendix C).

Participants were instructed as follows: "I am going to show you a picture. I want you to look carefully at the whole picture, and then tell me a story about it. Do not point to the picture. Keep talking about the picture until I ask you to stop. Do you understand what to do?" A prompt was given if a participant produced no speech for 30 seconds at any time during the task; this was done only once. If a participant declared that he/she had finished before the time had elapsed, he/she was told "You have a little more time." Responses were recorded on audiotape for three minutes. The latency to respond (i.e., seconds elapsed before first word uttered) was recorded. Tapes were transcribed, and the total number of words, as well as the total number of inappropriate communications, were tabulated for each subject.

Three types of inappropriate communications were defined, based on some of the

relevant literature on schizophrenic communication disturbances. Each of the three appears theoretically to be related to disinhibition, and/or has demonstrated correlations with features of the disorganization syndrome.

(a) "Intermingling" refers to the inappropriate blending of personal material from one's experiences into one's thinking and speech. This occurs in a disruptive manner (i.e., the relevance of these utterances to the current context is not made clear to the listener), leading to bizarre speech that is difficult to understand. This type of speech is associated with positive thought disorder (Harrow, Lanin-Kettering, Prosen, & Miller, 1983). Furthermore, it has been submitted that intermingling results from the failure of a higher-order cognitive control function (Harrow et al., 1983), or from deficient executive functioning (Harrow & Prosen, 1979). This executive function has been described as (a) the ability to take the perspective of the listener, or of what would be considered appropriate to the situation (Harrow et al., 1983); and (b) self-monitoring to prevent personal concerns from intermingling with speech (Harrow & Prosen, 1979). Thus, there is reason to consider intermingling an indication of an executive failure to inhibit the inclusion of personal concerns in one's speech. In this study, intermingling was scored only when there was clear evidence that the personal material which intruded into the response was not consistent with a typical response.

(b) An "unclear reference" (UR) is a word or phrase with a referent that is either ambiguous or completely unknown to the listener. The frequency of URs has been found to discriminate the speech of schizophrenic and manic patients (who perform similarly) from nonpsychiatric controls. URs also correlate with measures of thought disorder and disorganization, and can provide a measure of overall unclarity of speech (Docherty, Sledge, & Wexler, 1994; Docherty, DeRosa, & Andreasen, 1996). One particular type of UR has been identified as especially common in schizophrenia: references to information that is completely unknown to the listener, and that the listener should not be expected to have. These are referred to as missing information references (MIRs; Docherty et al., 1996). For the present study, the number of MIRs was counted.

(c) Perseverations are a commonly mentioned feature of schizophrenic speech (e.g.,

Freeman & Gathercole, 1966; Frith, 1992; Marengo, Harrow, & Edell, 1993). These were operationalized as inappropriate repetitions of words, phrases or ideas.

Examples of each of these speech abnormalities are provided in Appendix E. Combining these three types of inappropriate communications provided a numerical score for each subject. For 15 (37.5%) of the participants, a second rater independently scored the transcripts.

3. The second unstructured task was the **Tinkertoy test (TT)**. The task included 50 pieces as described by Lezak (1982) and others (Bayless, Varney, & Roberts, 1989; Mendez & Ashla-Mendez, 1991). No reliability data are available for this task. Due to some changes in the manufacturing of Tinkertoys since Lezak's study, a few minor changes were made in the specific test items, but essentially the same 50-item array was used.

So that an accurate assessment of latency could be made, the Tinkertoys were presented covered by a towel, and timing was begun after the towel was removed. Instructions were similar to those of Mendez and Ashla-Mendez (1991), but subjects were allowed 10 minutes instead of 15: "Under this cloth are some plastic pieces. When I uncover them, I'd like you to make whatever you want with them. You will have at least 5 minutes, and as much more time as you wish, up to 10 minutes, to make something. Do you understand what to do?" A prompt was given if a participant produced no action for 30 seconds at any time during the task; this was done only once. If a participant declared that he/she had finished before five minutes had elapsed, he/she was told "You have a little more time." Performance was videotaped (when possible, hands and constructions only). After 10 minutes, participants were stopped and asked what they had made; responses were recorded. Scoring included an evaluation of the end product, as described by Lezak (1982) and including the number of pieces used, the appropriateness of the name given to the construction, and a complexity score. The appropriateness of the name was scored either "yes" or "no". If a participant made more than one construction, the operational definition required each one to be appropriately named to receive a "yes" score. Lezak's (1982) complexity score includes the following: use of any combination of pieces, appropriateness of the name, movement (wheels or moving parts), symmetry, three-dimensionality, free-

standing status, and an error score. The complexity score was not relevant for the hypotheses, but was retained for potential exploratory analyses.

Scores derived from the approach to the task were obtained from the videotape. Latency to respond (i.e., seconds elapsed before the first piece was touched) was recorded. Also scored were several measures presumed to reflect disinhibition: the number of "false starts" (touching a piece but not picking it up, or picking up a piece only briefly); the number of "disconnections" (disconnecting pieces that had been connected); and the number of odd/unusual/inappropriate behaviours during the task. The guidelines used for these scores are presented in Appendix F. Combining these three error types yielded a numerical score for each subject. For 15 (37.5%) of the participants, a second rater independently scored the videotapes.

4. Assessment of cognitive competency was accomplished using the **Cognitive Competency Test (CCT; Wang & Ennis, 1986; Wang, Ennis, & Copland, 1987)**. The CCT was developed to evaluate cognitive competency from a practical and multidimensional perspective. It assesses a person's understanding of basic, practical life skills, using several simulated paper-and-pencil situations for different aspects of everyday life. The CCT items vary from overlearned concrete skills to basic abstract reasoning and judgment, and the CCT is believed to offer a more representative assessment of cognitive competency than other commonly-used methods (Wang & Ennis, 1986). Furthermore, the test is designed to minimize the inference that is often required to cross the gap between psychological assessment and everyday functioning (Wang, 1990). It is important to note that cognitive competency as rated by the CCT may not necessarily coincide with the psychiatric concept of *mental* competency. Examples (Wang & Ennis, 1986) include individuals who are declared mentally incompetent as a result of poor insight or psychotic symptoms, yet who demonstrate intact cognitive competency or only require partial assistance in living. The CCT was used for this study because cognitive functioning, in areas that impact everyday capacities, was of interest in terms of its relation to the neuropsychological deficits underlying the schizophrenic syndromes. Using the CCT allowed examination of cognitive correlates of the syndromes, even in individuals experiencing psychotic symptoms which

may render them "mentally incompetent."

The CCT consists of eight subtests: 1. Personal Information, in which the subject is required to complete a written application or registration form such as those found at banks or public agencies; 2. Card Arrangement, in which the subject is asked to rearrange a set of four picture cards in order to demonstrate the steps involved in performing common household tasks; 3. Picture Interpretation, in which the subject is asked to describe and interpret hand drawn pictures and to make inferences about interpersonal interaction; 4. Memory, in which the subject is required to recall a short grocery list, two prices, and information about a doctor's appointment (immediate and delay components included); 5. Practical Reading Skills, in which the subject must read labels or signs and answer simple questions about them; 6. Management of Finances, in which the subject is requested to perform simple calculations, make distinctions between cheques, bills and other monetary items, and write a simulated cheque; 7. Verbal Reasoning, in which the subject must respond to questions designed to test his or her knowledge and judgment regarding safety issues; and 8. Route Learning and Directional Orientation, in which the subject is required to use visual spatial memory and orientation skills. For a more complete description of the development and administration of the CCT, see Wang and Ennis (1986) or the CCT Manual (Wang, Ennis & Copland, 1987). The Average Total Score (ATS) provides a general index of cognitive functioning, and was therefore used in this study. The CCT record sheet is provided in Appendix G. Validity and reliability data are presented in Appendix C.

5. To assess **awareness of illness**, each participant was asked several questions related to his/her symptomatology (see Appendix H). This brief questionnaire was constructed based on literature addressing the issue of the assessment of insight in schizophrenia (Amador et al., 1991; Amador et al., 1993). The questionnaire was modelled after the general items from the Scale to assess Unawareness of Mental Disorder (SUMD; Amador & Strauss, 1990; Amador et al., 1993). These questions were not intended to provide a detailed assessment of insight, but rather, as the authors described them, to approximate the three most widely used definitions of awareness of illness (global awareness of mental disorder, awareness of the achieved effects of medication, and awareness of the social

consequences of having a mental disorder). Amador et al. (1993) have reported evidence of reliability and validity for the general items (see Appendix C). Current and retrospective insight were rated ("retrospective insight" refers to current awareness that one had a mental illness at some previous time). The scale was shortened to a three-point scale with each point clearly designated. A score of one indicated that the subject was clearly aware in that domain, a two signified "somewhat aware," and a three indicated unawareness. Correlations in the Results section are reported with the sign reversed, for ease of interpretation (i.e., higher scores reflect greater awareness). Interrater reliability for this questionnaire was determined by obtaining ratings by both Dr. Williams and the author (for all but one participant).

Interrater reliability

The author was blind to psychomotor poverty and disorganization scores at the time of testing. However, it is impossible not to become aware of certain symptoms when one is in the presence of a participant. This potential bias was minimized by ensuring acceptable interrater reliability with a second, truly blind, rater for the test scores hypothesized to measure initiation and inhibition. A second reason for establishing and reporting interrater reliability was that some of the scores used to reflect initiation and inhibition had not been used previously.

Development of measures. Interrater reliability was established in the following manner: Prior to data collection, pilot data were gathered using a sample of 12 healthy adults (graduate students, professors, and staff) and 4 hospitalized, clinically stable patients with schizophrenia. Because the two raters required further experience scoring unusual responses at the end of this practice, the first 8 study participants were also used for pilot scoring. (Thus, 32 subjects remained from which to select data to test reliability; see "Test of reliability", below.) Using these data, the second rater and the author practiced and refined scoring techniques in detail, aiming for the criterion of $r = .85$. A set of specific scoring criteria, with examples, was generated and used for reference while scoring (Appendices E and F). This valuable experience assured that the reliabilities would be maximized.

During this "development phase", interrater reliability was established and reported for the number of words on the MHP; the appropriateness of TT constructions; all three types of inappropriate communications on the MHP (intermingling, missing information references, and perseverations); and all three types of inhibition failures on the TT (false starts, disconnections, and odd actions). The remaining scores (latency to respond and number of TT pieces used) were simple and objectively measurable, and thus did not necessitate interrater reliability.

The development phase results presented in Table 2 reflect data scored following discussion and refinement of scoring criteria. Pearson correlations revealed that the goal of $r = .85$ was exceeded for number of words, perseverations, and total inappropriate communications for the MHP. Intermingling and MIRs failed to show interrater reliability at this time. (It was noted that, for these two measures, the majority of the inconsistency between raters occurred in scoring one particular transcript, which contained many bizarre communications which were exceptionally difficult to classify.) All three inhibition errors on the TT, as well as their total, also exceeded our criterion. The appropriateness score did not.

Test of reliability. A stratified random sample ($N = 15$) of the remaining 32 subjects (i.e., 37.5% of the total sample of 40) was selected and scored by both raters, in order to report interrater reliability of the scores purported to measure initiation and inhibition. During this "test phase", the raters did not discuss scoring in specific terms, nor were criteria altered. Test phase results are presented in Table 3. In view of the high interrater agreement for number of words during the development phase, the reliability of this score was not tested further. In addition, because the intermingling score (i.e., the number of intermingled *ideas*) failed to correlate, we added a score for the number of *words* considered to be intermingled. The rationale was that it may be easier to agree on the number of words representing personal material intruding upon a response than on the number of different ideas represented by those words. All five scores now revealed high correlations between raters (ranging from .92 to .97). For TT scores, two variables could not produce correlations because of lack of variation (i.e., odd actions were quite rare, as were appropriately named constructions); the

others showed correlations of .98 and .99. Thus, excellent interrater reliability was demonstrated for the majority of the experimental scores derived from the unstructured tasks. The only exceptions occurred when a variable, by its nature, lacked variation; that two variables would be so constant could not have been predicted.

The interrater reliability of the awareness questionnaire, as scored by Dr. Williams and the author, was .88 for awareness of current mental illness, and .75 for awareness of past mental illness.

Table 2

Development Phase Intercorrelations Between Raters for Picture Description and Tinkertoy Test

Score	<i>r</i>
	Picture description (<i>n</i> = 13)
Number of words ^a	1.00***
Intermingling	.28
Missing information references	-.12
Perseverations	.94***
Total inappropriate communications	.89***
	Tinkertoy test (<i>n</i> = 8)
False starts	.89**
Disconnections	.97***
Odd actions	1.00***
Total inhibition errors	.98***
Appropriateness of name	.66

^a*n* = 10.

p* < .01; *p* < .001.

Table 3

Test Phase Intercorrelations Between Raters for Picture Description and Tinkertoy Test

Score	<i>r</i>
Picture description (<i>n</i> = 15)	
Intermingling (no. of ideas)	.96***
Intermingling (no. of words)	.97***
Missing information references	.97***
Perseverations	.97***
Total inappropriate communications	.92***
Tinkertoy test (<i>n</i> = 15)	
False starts	.98***
Disconnections	.99***
Odd actions	a
Total inhibition errors	.99***
Appropriateness of name	a

^aCorrelation cannot be computed because at least one of the variables is constant.

****p* < .001.

Preliminary Considerations Regarding Data

1. The author's scoring was used even when two raters had scored a test.
2. Due to experimenter error, there were missing data for two participants on the measure of latency to respond on the MHP description. The median score for the other 38 participants was used to estimate these scores.
3. In the calculation of interrater reliability for the awareness of illness questionnaire, data from four participants were omitted. One of these cases was omitted because the awareness questionnaire had been completed by only one rater; two cases were omitted because one or more of the items received a rating of "Cannot be assessed or item not relevant;" and one was omitted because there were missing data (for both current and past awareness) for one of the questions.

Plan of Analysis

Hypotheses. As shown in Figure 2, deficient initiation is hypothesized to cause the psychomotor poverty syndrome (PPS), which in turn causes reduced cognitive competency (CC). Similarly, poor inhibition capacity is assumed to cause the disorganization syndrome (DS), which then also causes impairment in cognitive competency. These hypotheses were tested using regression.

Mediation effects form part of these hypotheses. Figures 3a and 3b depict the two models used to establish mediation (using the bottom part of Figure 2, involving inhibition and the DS). The nonmediated model (Figure 3a) indicates a direct pathway from inhibition to CC. In the mediational model (Figure 3b), the DS is hypothesized to mediate the relationship between inhibition and CC. Accordingly, inhibition has a direct effect on the DS, and DS has a direct effect on CC. Both pathways between inhibition and CC may be important: inhibition may have an indirect effect on CC via the DS, and inhibition may also exert a direct effect on CC that is not mediated by DS. However, the direct effect in 3b should be smaller than the direct effect in 3a.

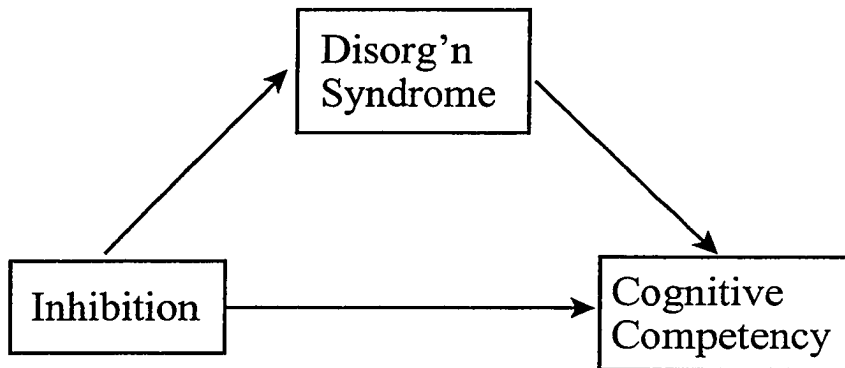
To determine whether the clinical syndromes mediate the impact of initiation and inhibition on CC, a series of regressions was performed. Evidence for a mediation effect

Figure 3. Models used to test mediation.

a. Nonmediated model



b. Mediator model



would be the following (Baron & Kenny, 1986):

1. The direct effect of inhibition on CC must be significant, indicating that inhibition predicts CC when no other variables are considered. This was tested by performing a regression with inhibition as the predictor of CC, and examining the standardized regression coefficient (i.e., the beta weight, β) for inhibition.

2. The direct effect of inhibition on DS must be significant, indicating that inhibition predicts DS. This was tested by performing a regression with inhibition as the predictor of DS, and examining the beta weight for inhibition.

3. The direct, unique effect of DS on CC must be significant, indicating that DS predicts CC. This was tested using a regression with inhibition and DS as predictors of CC, and examining the beta weight for DS. (The beta weight for inhibition was used for the comparison below.)

4. To test for mediation, the effect of inhibition on CC was compared for the first equation (in which the effect of DS was not considered) and the third equation (in which the effect of DS was partialled out). Mediation is indicated when the beta weight for inhibition is greater in the first equation than in the third. Evidence of complete mediation occurs when, after controlling for DS (i.e., equation 3), the beta weight for the effect of inhibition on CC is nonsignificant.

Influence of awareness of illness. As reviewed above, there is evidence to suggest that unawareness of illness in schizophrenia may be associated with frontally-mediated cognitive deficits (Lysaker & Bell, 1994; Young et al., 1993), and with indicators of clinical outcome (Amador et al., 1991). Therefore, the impact of unawareness of illness on cognitive competency will be explored in the following manner: (a) Determine whether there is a significant correlation between the two variables; (b) if there is a correlation, regress cognitive competency on unawareness and the other variables in the causal model, to determine whether unawareness is *uniquely (directly)* related to cognitive competency; and (c) determine the place of unawareness within the context of the model.

RESULTS

Appendix I presents the descriptive information for psychometric and demographic measures; Appendix J provides the intercorrelations of the psychometric measures.

Summary of Main Findings

The following results are detailed in the sections below. Results corroborated previous findings in revealing the existence of three independent syndromes within this sample. Contrary to hypothesis 1, initiation capacity did not predict the psychomotor poverty syndrome (PPS). In partial support of hypothesis 2, a measure of disinhibition predicted the disorganization syndrome (DS). One prediction within Hypothesis 3 was supported: DS mediated the impact of a disinhibition measure on cognitive competency (CC). However, such a mediational structure was not found for PPS; instead, initiation capacity had a direct effect on CC. Awareness of illness was correlated with DS, and contributed uniquely to the prediction of CC. Initiation, disorganization syndrome, and awareness of illness, combined, accounted for a sizeable proportion of the variance in CC. Each variable had a unique contribution. Sex and anticholinergic medication had no significant effect on the model.

Confirmation of Three-Factor Symptom Structure

Longer item set. We tested the hypothesis that data reduction of symptoms would reveal a three-factor solution, similar to previously reported factor solutions. Items contained in the SANS which measure avolition, anhedonia, and attentional problems were excluded, because there is some disagreement as to whether these items should be included. Liddle (1987b, p.147) did not include such items because he felt that they measured performance in daily life, rather than symptoms. The remaining SANS and SAPS data were subjected to principal component analysis with varimax rotation, using SPSS for Windows. Three orthogonal components were extracted, in accordance with prediction. The items with the highest correlations with (i.e., loadings on) the first component were those assessing various hallucinations and delusions. Measures of affective flattening and alogia loaded on the

second component. High loadings on the third component included inappropriate affect, positive formal thought disorder, and poverty of content of speech (although the latter symptom loaded higher on psychomotor poverty). This pattern of results corresponds closely to those reported by others (Liddle, 1987b; Liddle & Barnes, 1990; Malla et al., 1993). The three components can be designated reality distortion, psychomotor poverty, and disorganization syndromes, respectively, and accounted for 57% of the variance in symptoms. These results additionally provide evidence of the validity of the symptom ratings in this study. Table 4 reports the loadings.

Shorter item set. Due to concerns about the ratio of subjects to variables, a second, more limited, analysis was carried out. We selected only those symptoms previously found to be representative of the three syndromes (Liddle, 1987b) and used for scoring (see Method). Thus, nine items were selected: poverty of speech, decreased spontaneous movement, inappropriate affect, poverty of content of speech, global rating of positive formal thought disorder, voices commenting, persecutory delusions, delusions of reference, and a score for affective flattening derived by taking the mean of the scores for unchanging facial expression, affective nonresponsivity, paucity of expressive gestures, and lack of vocal inflections.

In initial analyses (above), poverty of content of speech loaded on psychomotor poverty syndrome in addition to disorganization syndrome, in contrast to previous findings. The correlation between poverty of speech and poverty of content of speech was .70 ($p < .001$), demonstrating that these two scores overlapped significantly. This situation alerted us to the possibility that these items may have been scored in a way that confounds them. In other words, although it is not surprising that poverty of *content of* speech would be observed in patients with poverty of speech (i.e., patients with psychomotor poverty syndrome), theoretically these two constructs should be separable. In order to statistically correct this confound, the effect of poverty of speech was partialled out of poverty of content of speech. This resulted in a measure of poverty of content of speech that is independent of poverty of speech. This correction was maintained for all analyses involving the disorganization syndrome score. Again, results of a principal component analysis with varimax rotation

confirmed our prediction of three orthogonal components using this subset of items. The pattern of loadings on each of the components (Table 5) supported the scoring method used in this study, and confirmed segregation into the three syndromes. The only unpredicted result was that the rating of positive formal thought disorder loaded on the reality distortion syndrome, in addition to disorganization. The components accounted for 72% of the variance in the nine items.

Table 4

Component Loadings Resulting From Principal Component Analysis of Longer SANS & SAPS Item Set

Symptom	Component 1	Component 2	Component 3
Auditory hallucinations	.72	.00	.00
Voices commenting	.45	.19	.00
Voices conversing	.73	.00	-.20
Somatic or tactile halluc.	.72	.00	.00
Olfactory hallucinations	.41	.00	.00
Visual hallucinations	.72	-.30	.38
Global rating - halluc.	.77	.00	.32
Persecutory delusions	.57	.00	.21
Delusions of guilt or sin	.47	-.11	.00
Grandiose delusions	.56	.00	.12
Religious delusions	.59	.00	.00
Somatic delusions	.50	.20	.19
Delusions of reference	.74	.00	.00
Delus. of being controlled	.66	-.24	.31
Delus. of mind reading	.73	.00	.14
Thought broadcasting	.63	.00	.38
Thought insertion	.78	-.20	.28
Thought withdrawal	.79	-.23	.00
Global rating - delusions	.73	.00	.30
Clothing and appearance	.41	-.12	.16
Social and sexual behav.	.49	-.27	.25

Table 4 (cont'd)

Component Loadings Resulting From Principal Component Analysis of Longer SANS & SAPS Item Set

Symptom	Component 1	Component 2	Component 3
Unchanging facial exp.	.00	.88	-.14
Decreased movement	-.11	.80	-.14
Paucity of gestures	-.17	.89	-.16
Poor eye contact	.00	.77	.00
Affective nonresponsivity	.00	.88	.00
Lack of vocal inflections	-.10	.90	.00
Global rating - flattening	.00	.80	.41
Poverty of speech	-.20	.84	.00
Pov. of speech content	-.24	.72	.47
Blocking	.00	.58	.39
Increased latency	.00	.85	.11
Global rating - alogia	-.21	.80	.42
Inappropriate affect	-.13	.15	.67
Global rating - PFTD	.39	.20	.80
Derailment	.49	.15	.55
Tangentiality	.39	.27	.75
Illogicality	.53	.12	.71
Incoherence	.28	.13	.77
Circumstantiality	.12	.00	.79
Pressure of speech	-.15	-.14	.76
Distractible speech	.00	.11	.75
Clanging	.00	-.18	.78

Table 4 (cont'd)

Component Loadings Resulting From Principal Component Analysis of Longer SANS & SAPS Item Set

Symptom	Component 1	Component 2	Component 3
Aggressive & agitated	.25	-.10	.54
Global rating - bizarre	.35	-.12	.59
% variance accounted for:	21	19	17

Note. Only those items with a loading of .4 or greater on at least one component are listed.
PFTD = positive formal thought disorder.

Table 5

Component Loadings Resulting From Principal Component Analysis of SANS & SAPS
Items Used for Scoring

Symptom	Component 1	Component 2	Component 3
Affective flattening	.94	.00	.00
Decreased movement	.89	.00	.00
Poverty of speech	.89	.00	.00
Voices commenting	.00	.69	.00
Persecutory delusions	-.10	.72	.11
Delusions of reference	-.17	.92	.00
Global rating - PFTD	.00	.50	.66
Inappropriate affect	.00	.00	.83
Pov. of speech content	.00	.00	.83
% variance accounted for:	28	23	21

Note. PFTD = positive formal thought disorder.

Hypotheses

Because multiple significance tests were performed to test the hypotheses, a conservative critical p value was used to reduce the chance of making Type I errors. Thus, serious consideration was given only to effects for which the significance level was $p < .01$. For hypothesized effects, p values are based on 1-tailed tests.

Hypothesis 1 tested the assumption that poor ability to initiate self-generated thoughts and actions causes the psychomotor poverty syndrome (a negative relationship was expected between initiation capacity and the psychomotor poverty syndrome). This hypothesis was not supported.

There were two problems with using a latent factor model (i.e., common factor analysis; CFA), as hypothesized, to reflect initiation capacity. First, the intercorrelations of the four scores presumed to reflect initiation capacity were surprisingly low; they ranged from .16 to .41. This suggests that there was no strong influence of a single underlying ability (i.e., low saturation). Combined with having only four variables to one factor (i.e., a poorly identified factor), this is a situation in which principal component analysis is preferable (PCA; Velicer & Jackson, 1990b, p. 101). Second, a theoretical problem inherent in CFA is that factor scores are mathematically indeterminate, whereas PCA components are determinate (Schönemann, 1990; Velicer & Jackson, 1990a). For these reasons, PCA was used to form a component (i.e., a weighted aggregate) using the four scores measuring initiation: MHP # words, MHP latency, TT # pieces, and TT latency. A regression of psychomotor poverty syndrome on the initiation component was nonsignificant ($\beta = .14, p > .01$).

Hypothesis 2 tested the assumption that poor inhibition of inappropriate responses causes the disorganization syndrome (a negative relationship was expected between inhibition capacity and the disorganization syndrome). This hypothesis was not supported when the proposed inhibition measure was used. However, when the scores comprising the inhibition measure were subsequently explored separately, it was evident that one measure of inhibition did support the hypothesis.

Inspection of the data prompted a change to the inhibition measure. Because the

scores for the appropriateness of TT constructions lacked variation (36/40 participants produced inappropriate constructions), this variable was excluded from analyses. A simple (unweighted) aggregate was formed using errors made on the MHP and on the TT. Each of the two scores in the aggregate was divided by a measure of initiation/output, to form a ratio: MHP errors was divided by the total number of words produced, and TT errors was divided by the amount of time spent on the task (i.e., between five and 10 minutes). This transformation removed the confound created by the fact that individuals differed in their ability to generate responses, and therefore presumably had differing opportunities to commit errors. Furthermore, a theoretical reason for this transformation is that the syndromes are considered independent. For example, an individual who has psychomotor poverty syndrome, and therefore produces little speech on the MHP, should be comparable in terms of disinhibition errors to someone who generates a long story. These ratios were used in all subsequent analyses. A regression of disorganization syndrome on this disinhibition aggregate was nonsignificant ($\beta = .19, p > .01$), contrary to hypothesis 2.

We then considered the variables representing MHP and TT errors individually, in order to learn whether some variables supported the hypothesis. The MHP error ratio was significantly correlated with the disorganization syndrome ($r = .34, p < .02$), whereas the TT error ratio was not ($r = -.09, p > .01$). In light of this result, we explored the individual MHP scores in relation to the disorganization syndrome. Of these, only the intermingling ratio (i.e., the number of intermingled words divided by total number of words) was significantly related to disorganization ($r = .58, p < .001$). Missing information references and perseverations (ratios) were not related to disorganization ($r = .17, p > .01$ and $r = .05, p > .01$, respectively).

In summary, the frequency of intermingling - a component of the verbal unstructured task - was the only measure of disinhibition that was related to the disorganization syndrome. Individuals who intermingled more personal material into their stories showed more features of the disorganization syndrome. Results partially supported hypothesis 2. Speculations as to why intermingling was the only score which supported the hypothesis are presented in the Discussion.

The first part of Hypothesis 3 tested the assumption that both the psychomotor poverty syndrome and the disorganization syndrome predict deficits in cognitive competency. A regression of CC on psychomotor poverty and disorganization revealed that disorganization was a significant predictor of CC, in the expected direction ($\beta = -.58, p < .001$). That is, participants with more severe disorganization syndrome obtained lower scores on the CCT. In contrast, psychomotor poverty failed to reach significance as a predictor of CC ($\beta = .05, p > .01$).

The second part of Hypothesis 3 tested the assumption that the psychomotor poverty and disorganization syndromes mediate the impact of initiation and inhibition impairments, respectively, on cognitive competency. Given that initiation failed to predict psychomotor poverty, and psychomotor poverty failed to predict CC, it was impossible for this syndrome to act as a mediator (as explained in Plan of Analysis, steps 2 and 3). However, the initiation component was directly related to CC ($r = .39, p < .01$). Furthermore, in light of the findings above regarding the relation of inhibition measures to disorganization, only the intermingling ratio was included in these analyses. Regression was used to test the hypothesis that the disorganization syndrome mediates the effect of intermingling on CC.

The relationships between variables were indicative of a mediation effect, as discussed above. Namely:

1. As shown in Figure 4a, intermingling significantly predicted CC when no other variables were considered ($\beta = -.38, p < .01$).

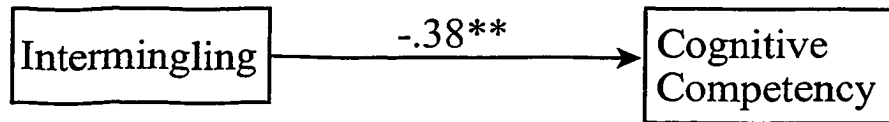
2. As shown in Figure 4b, intermingling significantly predicted the disorganization syndrome ($\beta = .58, p < .001$).

3. As shown in Figure 4b, the disorganization syndrome significantly predicted CC, independent of the effect of intermingling ($\beta = -.53, p < .005$).

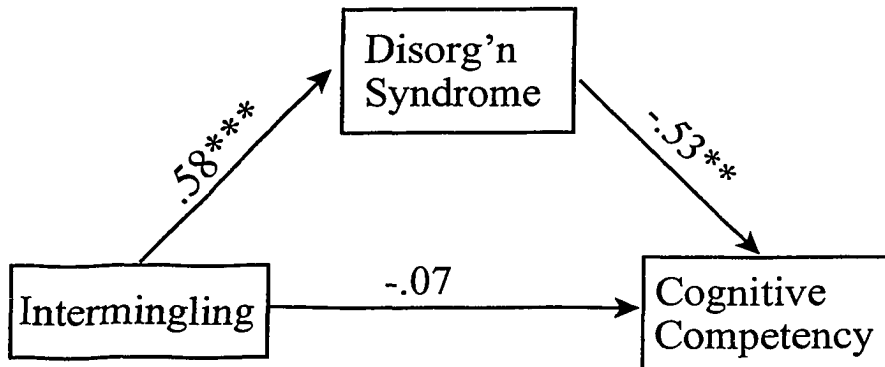
4. The intermingling ratio's prediction of CC decreased when the mediator, disorganization syndrome, was added to the model (from $\beta = -.38, p < .01$ to $\beta = -.07, p > .01$). In addition, after accounting for the disorganization syndrome, intermingling was no longer a significant predictor of CC. This outcome confirms a mediator model, as hypothesized for inhibition measures. In other words, individual differences in the

Figure 4. Models used to test mediation, with standardized regression coefficients.

a. Nonmediated model



b. Mediator model



p < .01; *p < .001

disorganization syndrome were caused by individuals' tendencies to intermingle, and effected the observed differences in CC. The proportion of the variance in CC that was accounted for uniquely by the disorganization syndrome was calculated as the squared beta weight for disorganization, in the regression using disorganization and intermingling as predictors of CC (Cohen & Cohen, 1983). Thus, disorganization accounted for $(-.53)^2$, or 28%, of the variance in CC, over and above the relationship between intermingling and CC.

As reported above, the initiation component was directly related to CC. That is, participants with better initiation capacity obtained higher CCT scores. In order to better understand the meaning of the initiation measure (especially because it failed to correlate with psychomotor poverty), the four scores were partitioned into a measure of latency (TT latency + MHP latency) and a measure of generation (TT # pieces + MHP # words). Examination of correlations revealed that it was generation measures that explained the association of the initiation component and CC: generation correlated with CC ($r = .42, p < .01$), whereas latency did not ($r = -.07, p > .01$). Patients who produced more speech during the story task and included more TT pieces in their constructions scored higher on the CCT. (Neither generation nor latency correlated with psychomotor poverty syndrome.)

In summary, hypothesis 3 was partially supported. Disorganization, but not psychomotor poverty, predicted CC. Disorganization mediated the impact of intermingling on CC. Initiation (specifically, the amount of speech generated and the number of Tinkertoys used during the tasks) exerted a direct influence on CC, rather than being mediated by psychomotor poverty.

Awareness of Illness

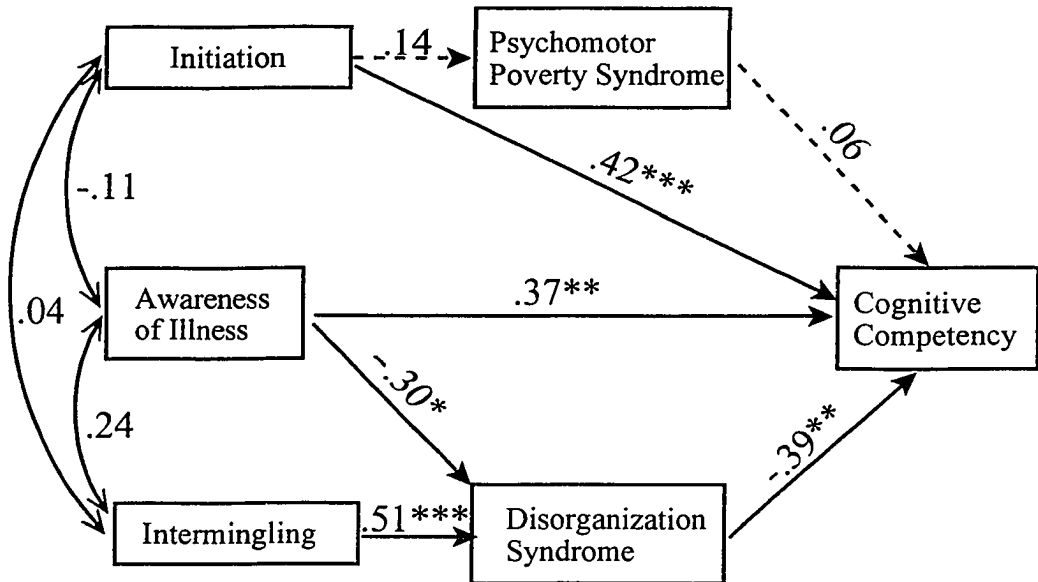
Awareness of current and of past mental illness were highly intercorrelated ($r = .82, p < .001$). Therefore, the sum of awareness of current and of past illness was used to provide an overall awareness measure. Overall awareness of illness correlated with CC in the expected direction ($r = .50, p = .001$). That is, individuals who were more aware of their illness had higher CC scores. Multiple regression analysis revealed that awareness *directly* predicted cognitive competency ($\beta = .38, p < .005$), over and above the effects of the

disorganization syndrome and the initiation component. Next, the place of unawareness within the context of the model was explored. Results indicated that, of the variables that predicted CC, awareness correlated only with disorganization syndrome ($r = -.41, p < .01$): individuals who were less aware of their illness exhibited more features of the disorganization syndrome.

Predicting Cognitive Competency

Multiple regression analyses were used to assess the contribution of each variable that was related to CC, independently of the other predictor variables. The variables that predicted CC, in decreasing order of magnitude of *unique* contribution, were: the initiation component ($\beta = .42, p < .001$); the disorganization syndrome ($\beta = -.41, p < .001$); and awareness of illness ($\beta = .38, p < .005$). Together, initiation, disorganization, and awareness accounted for 58% of the variance in CC ($R^2 = .58$; adjusted $R^2 = .54$). Based on these results, the model depicted in Figure 2 can be revised to conform with the data (see Figure 5). In this figure, the coefficients shown result from regressions including all possible predictors of each endogenous variable. (Therefore, results differ slightly from those reported above; however, the pattern of results is identical.) The exclusion of a path between two variables signifies that the path was nonsignificant. Dotted lines indicate predicted paths that were nonsignificant.

Figure 5. Revised causal model, with standardized regression coefficients.



* $p < .05$; ** $p < .01$; *** $p < .001$

Influence of Sex

Due to evidence of different correlation patterns between cognitive measures and symptoms for males and females (e.g., Perlick, 1992), it was important to determine the impact of sex on these correlations in the present study. Correlational analyses revealed that, of the variables in the model, sex correlated only with CC ($r = -.324, p < .05$). Since sex was coded as 1 = female and 2 = male, this result indicates that males had lower CCT scores than females.

To determine the impact of sex on the model as a whole, regression was employed. To test the interactions between sex and each of the three predictor variables in the model, three interaction terms were added to the equation that included initiation, disorganization, and awareness as predictors of CC. These interaction terms did not significantly improve the prediction of CC, $F \text{ Change}(3, 33) = .67, p = .58$. To test the main effect of sex, sex was added to the original equation. Sex did not significantly improve the prediction of CC, $F \text{ Change}(1, 35) = 1.17, p = .29$, indicating no independent contribution of sex to CC. Thus, because the main effect of sex was not significant, and sex did not interact with the independent variables in predicting CC, sex was omitted from the model.

Influence of Anticholinergic Medications

There is evidence that anticholinergic medications can have a negative impact on cognition, especially memory (Tune, Strauss, Lew, Brietlinger, & Coyle, 1982). Correlational analyses revealed only one significant relationship between the use of Kemadrin or Cogentin and a variable in the model: patients with more features of the disorganization syndrome were less likely to be taking anticholinergic medications ($r = -.41, p < .01$). Stated differently, patients taking anticholinergic medications exhibited fewer features of the disorganization syndrome. However, the use of anticholinergics was not associated with CC ($r = .09, p > .01$). Thus, the overall results are not affected by the use of anticholinergic medications.

DISCUSSION

The following paragraphs will examine each of the results related to the hypotheses, as well as the effect of unawareness of illness. Finally, implications of the study, generalizability of these results, and directions for future research will be discussed.

Three-Syndrome Model of Schizophrenia

This study confirmed, in a sample composed predominantly of young, recently-diagnosed outpatients, the existence of the three independent syndromes first delineated by Liddle (1987b). This replication was achieved using both a long data set and a subset including only those items from which the syndrome scores were obtained. The fact that these results were obtained with a sample of 40 underscores the general robustness of the pattern. Thus, these findings add to the expanding literature suggesting that there is more empirical support for using these *three* syndromes as dimensions along which to characterize individuals with schizophrenia than there is for the (still prevalent) use of "negative" and "positive" syndromes.

Initiation Capacity Fails to Predict Psychomotor Poverty Syndrome

Our initiation measure shows no relationship to the psychomotor poverty syndrome. This result is unexpected for two main reasons. First, it is inconsistent with previous suggestions that the cognitive impairment underlying the psychomotor poverty syndrome is slowness of mental activity or deficient generation of a plan to act (e.g., Liddle et al., 1993; Liddle & Morris, 1991). Our initiation component was composed of four scores that appear to be face-valid indicators of initiation, generation, and speed of mental activity: two scores measuring the latency to initiate a response, and two scores measuring the total production during a task. If the theory above is accurate, we would expect a correlation between such an initiation measure and psychomotor poverty (which is composed of poverty of speech, decreased movement, and affective flattening). Second, the lack of correlation in this study more directly contradicts the results of Mendez and Ashla-Mendez (1991). They reported

that patients with multi-infarct dementia produced fewer words on a picture description task, and used fewer pieces on the Tinkertoy test, than patients with Alzheimer-type dementia - presumably due to executive deficits affecting the initiation and maintenance of behaviour. Schizophrenic patients with high ratings on the psychomotor poverty syndrome presumably have a similar executive deficit in initiation and behavioural maintenance; therefore, they would be expected to show similar performance on these very same tasks. This was not obtained.

The psychomotor poverty syndrome has essentially the same composition and properties in this study as other studies have reported. Therefore, the lack of correlation does not appear to be due to an unusual pattern of symptom ratings. In addition, inspection of the data revealed that the lack of correlation cannot be attributed to a lack of variance in the psychomotor poverty or initiation measures. Given these conditions, the lack of correlation between initiation and psychomotor poverty appears to be related to either atypical features of the sample or unexpected properties of the initiation measure. There are thus several potential explanations for this result:

1. The studies upon which the hypothesis was based (Allen et al., 1993; Liddle, 1987a; Liddle & Morris, 1991) utilized patients with chronic schizophrenia. These samples differ from that in the present study, which, although the mean duration of illness was seven years, included a significant proportion of patients whose onset was very recent (14 patients had an illness duration of one year or less). Thus, the patients in this study may have had less stable (i.e., less reliable) symptom profiles, and associated cognitive deficits, than those in previous studies. Because reliable cognitive abilities are more likely to correlate with other abilities (McNemar, 1969, p. 172), such a difference in sample could potentially limit correlations, so that some of the associations found with more chronically ill patients were not obtained. This explanation would be consistent with the results of Baxter and Liddle (1998), who found an association between psychomotor poverty and slowed decision-making in patients with persistent, but not with remitting, schizophrenic illness.

A related possible explanation stemmed from suggestions that, at least in some people, negative symptoms become steadily more prominent over the course of the illness,

while positive symptoms, which are more responsive to treatment, become less important (APA, 1994). Thus, because many of the present subjects had a short illness duration, it appeared possible that they had less pronounced negative symptoms as compared to positive symptoms, resulting in an attenuation of the relationship between psychomotor poverty symptoms and initiation deficits. However, inspection of the data revealed that (a) the syndrome was equally severe for shorter vs. longer duration patients; and (b) neither of these groups demonstrated the expected prediction of the psychomotor poverty syndrome from initiation. Consequently, the lack of correlation cannot be attributed to reduced *variance* in psychomotor poverty due to sample characteristics. However, low *stability* of syndromes/cognitive deficits remains a possible explanation of this null result.

2. Another possible explanation for these findings is that, despite its face validity, our purported measure of initiation (derived from unstructured tasks) fails to measure the same construct that previous research has linked to the psychomotor poverty syndrome (e.g., Liddle & Morris, 1991). This explanation is supported by the fact that, as described in Results, the four initiation scores did not appear to reflect a single underlying construct. Previous research with schizophrenic samples reveals some rationale for using unstructured tasks to elicit difficulties in *inhibition*. Namely, Cohen (1978) reported that "challenging" tasks are more likely to elicit disordered speech from thought-disordered patients. Indeed, they found that such patients produced unusual verbal output on a (largely self-guided, i.e., unstructured) verbal fluency task. In contrast, there is no evidence, from studies using schizophrenic samples, suggesting that unstructured tasks elicit problems in *initiation*. Nonetheless, as discussed previously, there are excellent grounds for testing unstructured tasks for their ability to elicit executive impairments in this population.

From the above discussion, it can be concluded that the initiation measure fails to predict psychomotor poverty in this investigation because either (a) the subjects are not all chronic schizophrenia patients, limiting the stability, and thus the intercorrelations, of cognitive skills and symptoms; (b) our initiation measure represents a construct which differs from the constructs defined in previous studies; or (c) a combination of these reasons. Although (a) seems likely in light of Baxter and Liddle's (1998) conclusion, it should also

be noted that the power of the study may have been insufficient to detect some small effects. For example, given the sample size ($N = 40$), for a population effect size of .35 power was approximately .50. However, for effects in the range of .5 to .6, power was sufficient (about .90 to .99). Finally, despite accumulating evidence in its favour, the hypothesis that impaired initiation is associated with psychomotor poverty may be erroneous; the results of Norman et al. (1997), mentioned in the Introduction, lend provisional support to this idea.

Initiation, But Not Psychomotor Poverty Syndrome, Predicts Cognitive Competency

Results indicate that, contrary to prediction, having more severe psychomotor poverty syndrome does not predict lower cognitive competency. This finding suggests that symptoms of decreased speech and movement and affective flattening need not be a barrier to being competent to perform basic skills required for independent living. However, the present findings reveal that our measure of initiation capacity exerts a direct (nonmediated) positive effect on cognitive competency. The implication is that slowness of mental activity, or difficulty generating plans, detracts from cognitive skills for everyday living. The CCT is an untimed test; therefore, slowness *per se* would have no impact on performance. Yet, mental slowness, or impaired planning, clearly influences performance. The prediction is obtained because individuals with low initiation capacity are likely to produce deficient, not only slow, responses to daily-living tasks.

As demonstrated in Results, it was the generation (as opposed to latency) measures that explained the association of the initiation component and CC. That is, individuals who generated more speech and incorporated more Tinkertoys during the unstructured tasks obtained higher scores on the CCT. The interpretation suggested is that the ability to generate responses, verbally and non-verbally, increases the chances of obtaining maximal scores on the CCT. This is consistent with the general fact that, in neuropsychological testing, if an individual attempts several responses to an item, he/she is likely to achieve at least partial scores.

Our generation measure is very similar to that of Mendez and Ashla-Mendez (1991), described as a reflection of deficient initiation and maintenance of behaviour. In the present

sample, the cognitive ability to initiate and maintain behaviour is not redundant with the cognitive ability underlying the psychomotor poverty syndrome. In an earlier study, simple reaction time was found to predict clinical outcome, and to discriminate prospectively improved patients from those who did not improve (Zahn & Carpenter, 1978). The present relationship between our initiation (generation) measure and cognitive competency might be viewed as a similar finding, in the realm of functional outcome.

The present study thus indicates that (for initiation/generation and psychomotor poverty) the executive deficit causes the functional burden, whereas disease-specific syndromes are irrelevant in this regard. This outcome is consistent with previous research (Green, 1998, p. 178; Royall et al., 1993; Velligan et al., 1997). Although a mediated relationship was hypothesized in order to test the indirect pathway via psychomotor poverty syndrome (consistent with the four-level model in Figure 1b), the direct pathway obtained is consistent with research indicating that frontal lobe/executive functions underlie decision-making capacity and predict functional status in various groups (e.g., Green, 1995; Marson et al., 1995; Schindler et al., 1995).

Intermingling Predicts the Disorganization Syndrome

One of the purported indicators of disinhibition - the tendency to intermingle personally relevant material into one's thinking and speech in a disruptive manner - is significantly associated with the disorganization syndrome in this study. This finding can be taken to support previously articulated hypotheses that deficient inhibition underlies the disorganization syndrome. The evidence below supports this notion.

It might appear that intermingling does not measure cognitive disinhibition, but rather simply reflects the disorganization syndrome. This argument is suggested by the fact that the disorganization syndrome score includes a global rating of positive formal thought disorder (PFTD), two items of which (derailment and tangentiality) seem to describe the phenomenon of intermingling. However, there are several arguments supporting intermingling as a more basic measure of disinhibition. First (as mentioned in Method), intermingling is considered an indication of an executive, or cognitive control, failure to inhibit the inclusion of personal

concerns in one's speech (Harrow et al., 1983; Harrow & Prosen, 1979). Second, the instructions provided to subjects for the MHP task include a specification that they should talk *about the picture* throughout the time given, implying that they should not intermingle. This task parameter would suggest that when individuals did intermingle during the MHP task, the behaviour reflected an inability to inhibit this action. In contrast, the psychiatric ratings of derailment and tangentiality are made in the context of a more naturalistic observation, free of instructions to regulate their speech. Finally, the correlations of intermingling with the three components of the disorganization syndrome - poverty of speech content, inappropriate affect, and PFTD - suggest that intermingling measures a construct different from PFTD. Specifically, intermingling did not correlate significantly with the global rating of PFTD ($r = .29, p > .01$), and did correlate with inappropriate affect ($r = .63, p < .001$).

Neither of the other two disinhibition scores from the picture description task is useful in this study, for the following reasons. The speech error "missing information references" is noncontributory as its occurrence is rare in this sample (only six subjects made this type of error). The number of perseverations is unrelated to intermingling, suggesting that it does not measure disinhibition. Corroborating this statement, observations during testing indicated that the "perseverations" in the speech of this sample generally appeared no different from the speech repetitions made by healthy individuals. Hence, the suggestion that abnormal perseveration is common in schizophrenia (e.g., Frith, 1992) is not upheld in this sample.

The aggregate of the experimental scores derived from the Tinkertoy test (false starts, disconnections, and odd actions) is also not significantly related to the disorganization syndrome. (However, there is a hint that the number of odd/unusual actions may be related to the disorganization syndrome. This variable was not used for analyses, because only six subjects obtained scores greater than 0. However, these subjects tended to have high disorganization scores, resulting in a correlation [$r = .39, p < .05$] with the disorganization syndrome.) In summary, it appears that either (a) the Tinkertoy test is not helpful in eliciting inhibition impairments or (b) the scores derived from the test in this study, with the possible

exception of odd/unusual actions, are not valid indicators of disinhibition. Specific types of errors in speech appear more promising as measures of disinhibition.

The Disorganization Syndrome Mediates the Effect of Intermingling on Cognitive Competency

This mediation effect was predicted. This outcome suggests that the executive deficit (in this case, disinhibition as reflected by intermingling) explains the functional burdens in schizophrenia *indirectly*, through its impact on the clinical syndrome. Our results can be viewed in the context of previous findings. For example, Velligan et al. (1997) reported that cognitive functioning predicted the performance of everyday tasks, and that symptomatology had little direct impact on such functional measures. These authors suggested that cognition mediates the effect of symptomatology on the performance of everyday tasks, whereas we have proposed that symptoms mediate the effect of cognition on functioning (cognitive competency). Despite this theoretical difference and the differences in measures, our results (i.e., those related to the disorganization syndrome) and those of Velligan et al. (1997) are congruous, in finding that the variance shared between cognitive variables and clinical syndromes accounts for a significant proportion of the variance in functional capacity.

On the other hand, our results with initiation and psychomotor poverty syndrome (discussed above) suggest that the executive deficit affects cognitive competency *directly*. Whether the effect is direct or indirect, both of our results suggest a prominent role for executive deficits in determining the functional status of schizophrenic patients. This conclusion is consistent with past research implying that cognitive deficits are related to the level of social competence in chronic schizophrenic patients (Penn, Mueser, Spaulding, Hope, & Reed, 1995), and with the concept that cognitive deficits may act as a rate-limiting factor for acquiring skills in psychosocial rehabilitation programs (Lieberman & Corrigan, 1993).

To date, few studies have attempted to link levels of understanding in schizophrenia (Mortimer, 1992; see Figure 1). Therefore, it is difficult to integrate findings with those from the sparse literature that is comparable. How can we comprehend the results reported here?

In one case, a cognitive variable predicts cognitive competency directly, whereas in another instance the relationship is mediated by a clinical syndrome. We subscribe to the levels of understanding model proposed by Mortimer (1992; Figure 1a), which we have expanded (Figure 1b), because (a) it is consistent with a neuropsychological approach to understanding human behaviour; and (b) it provides a useful heuristic - a framework which helps in designing studies and analyzing data. However, in reality it is possible that there are no clear distinctions between the "levels." In other words, specific executive dysfunctions and associated clinical syndromes may overlap to form broader "neuropsychological/syndromal presentations", and these may be associated with functional outcome measures such as cognitive competency.

Unawareness of Illness is Associated With the Disorganization Syndrome

Results indicate that schizophrenic patients with the disorganization syndrome, compared to those with the psychomotor poverty syndrome, tend to be less aware that they have a mental illness. There is indirect evidence that self-awareness may involve the orbitofrontal circuit as part of its neural substrate. For example, a review by Cummings (1993) concluded that the orbitofrontal syndrome features marked personality changes (from which decreased self-awareness might be inferred), and Cicerone and Tanenbaum (1997) reported reduced self-awareness and functioning in social situations in a patient with an orbitofrontal injury. In this light, the present correlation is interesting, as the orbitofrontal circuit has also been implicated in the disorganization syndrome, as described in the introduction (Liddle et al., 1989, 1992). Although this suggestion of a specific (i.e., *orbitofrontal*) neural connection between insight and disorganization is speculative, there is indirect evidence that the prefrontal lobes more generally might subservise self-awareness (McGlynn & Schacter, 1989; Stuss & Benson, 1986) and insight in schizophrenia (Lysaker & Bell, 1994; Young et al., 1993). Our findings support the notion that impaired insight may result from fixed prefrontal-lobe neuropsychological deficits.

Our results are consistent with a study reporting that certain features of the disorganization and reality distortion syndromes (i.e., increased delusionality, thought

disorder, and disorganized behaviour) were correlated with decreased awareness of mental disorder (Amador et al., 1994). However, the significance of these modest correlations appears to reflect the large sample size used; in fact, the associations were quite weak.

Contrary to our findings, diminished insight has been described as characteristic of patients with the "deficit syndrome," or primary negative symptoms (which are similar to the symptoms used to score the psychomotor poverty syndrome; Amador & Kronengold, 1998). Based on the latter report, we would expect that the psychomotor poverty syndrome would also be related to unawareness of illness. However, this is not observed in the present data. An explanation for this finding might be that the present sample is less likely to have a reliable deficit syndrome, as discussed above. Finally, in a data reduction study involving the SANS, SAPS, and a measure of insight, Peralta and Cuesta (1994) found that insight produced a factor separate from negative symptoms, thought disorder, and delusions and hallucinations.

Unawareness of Illness Predicts Reduced Cognitive Competency

Although unawareness is correlated with the disorganization syndrome, the present results indicate that each of these variables contributes uniquely to the prediction of a functional outcome measure, cognitive competency. This suggests important differences between the two. These findings are thus similar to the conclusion of David's (1998) review of the clinical importance of insight; that is, insight represents a distinct aspect of psychosis, and is clinically important.

Unawareness of illness accounts for a unique, significant portion of the variance in cognitive competency. This result is not surprising, given previous research demonstrating that poor insight predicts functional impairments at work (Lysaker & Bell, 1995), and also that poor insight at initial assessment predicts the amount of time spent living independently and gainfully employed over a follow-up period (van Os et al., 1996). The present finding contributes to this literature in documenting that patients who lack awareness of their mental illness tend to experience more difficulty in performing the relatively simple mental tasks needed for daily living. Clearly, such basic cognitive difficulties as those measured by the

CCT would impact functioning in all settings, including work and independent home living.

Implications

The most important and unique contribution of this research is obtained by virtue of its focus on cognitive competency as the outcome measure. 58% of the variance in cognitive competency is explained by the combination of initiation capacity, disorganization syndrome, and level of insight. Therefore, if a patient's functioning in each of these domains could be maximized, this would presumably have a substantial impact on the patient's ability to perform basic cognitive skills necessary for living independently. Potentially, such improvements might reduce the number of future hospital admissions, the amount of assistance/supervision resources required to support the patient's functioning outside the hospital, and the disruption of family life.

The results of this study inform health professionals about which deficits and syndromes should be the focus of remediation/symptom control efforts. Such information has been called for previously; for example, Hogarty and Flesher (1992, p. 53) remarked that before embarking on the remediation of cognitive deficits, it would be helpful to know how a specific deficit or pattern of deficits relates to disability.

Given that they relate to cognitive competency, the following would appear to be useful interventions:

1. Improvement of the executive functions of generation of plans, as well as initiation and maintenance of responses. This idea is consistent with the notion that patients' prognosis might be improved by treatment designed to ameliorate executive functioning (Zec, 1995). As mentioned above, reaction time has been found to provide a simple yet informative index related to clinical outcome. Zahn and Carpenter's (1978) interpretation was that patients who can respond more appropriately to the environment are able to recover more quickly from acute psychotic episodes. The process-specific approach to cognitive rehabilitation (Sohlberg & Mateer, 1989, pp. 22-28) may be useful for improving initiation capacity in schizophrenic patients (Sohlberg & Mateer, 1989, ch. 10; p. 401).

2. Reducing the frequency of intermingling, which appears to reflect disinhibition at

a more basic cognitive level (Harrow & Prosen, 1979; Harrow et al., 1983). As with initiation, interventions designed to improve inhibition capacity might best be approached using the process-specific method. (For elaboration, refer to Sohlberg & Mateer, 1989, pp. 244-245 and 248-249.)

3. Achieving control of symptoms of the disorganization syndrome. The core symptoms consist of positive formal thought disorder, inappropriate affect, and poverty of speech content. This control will most likely be best realized through neuroleptic medications.

4. Increasing awareness of illness in those patients who have poor insight. Psychosocial rehabilitation appears to be one method of accomplishing this goal (for a review, see Lysaker & Bell, 1998). In addition, McFarlane and Lukens (1998) suggest that education, training, and social support involving not only the patient, but the patient's family and friends, foster insight. Awareness should be targeted early in the course of illness, since early poor insight seems to predict outcome regardless of subsequent improvements in awareness (David, 1998).

It should be noted that, given the relationship between cognitive deficits and functional outcome, other cognitive abilities have also been investigated in terms of their potential for improvement (for a review, see Green, 1998, ch. 9).

This research provides partial support for the proposals of Liddle and his colleagues, concerning the underlying cognitive causes of the syndromes of schizophrenia. The idea that psychomotor poverty manifests itself secondary to impaired initiation of mental activity was not supported in this study. However, we did substantiate suggestions that the disorganization syndrome is related to impaired inhibition of inappropriate mental activity.

Representativeness of the Sample

The participants in this study were typical of similar studies in several ways. They were diagnosed by widely accepted standards (DSM-IV), and were free of other Axis I diagnoses, neurological disorders, or head injuries. Their level of education and their estimated intellectual ability were average. All patients were taking neuroleptic medications.

There were, however, some aspects of the sample that appear less typical. This sample contained more outpatients than inpatients (29/40). In addition, a significant portion of the sample had a very recent onset of schizophrenia, and there were relatively few "chronic" patients who had been diagnosed for many years (the mean duration of illness was approximately 7 years). Therefore, the group may be less severely and chronically ill than the samples of some other studies. Pending replication in groups that differ from this one, the present results should not be taken to generalize to all people with schizophrenia.

Directions for Future Research

The fact that CCT performance was significantly predicted by measures in this study has implications for future work. The CCT may prove to be a useful outcome variable to use for cognitive remediation studies. However, before this could be implemented, it would be important to determine the relationship of CCT performance to real-life functioning. Although the test certainly has overall face validity and is believed to offer a representative assessment of everyday functioning (Wang, 1990), some of the subtests are identical to the real-life situation (e.g., providing personal information on a form), whereas other subtests are not so clearly analogous to real settings (e.g., tracing routes on a map with a fingertip). Furthermore, like traditional neuropsychological tests (as discussed in the Introduction), the CCT does not require patients to perform with minimal guidance. Prompts and cues are provided if a participant does not respond with an appropriate attempt for a given item (and, for some subtests, if a participant does not provide a full-credit response). For these reasons, the hypothesis that the CCT assesses real-life capacity should be carefully examined. If future research revealed that the CCT was highly correlated with functioning in daily life, then the development and validation of alternate forms would be of further help, so that the test could be used to monitor the efficacy of the remediation efforts discussed above, without the confounding influence of practice effects.

Investigations of the effectiveness of the four strategies mentioned above would be beneficial. The most useful approach may be to use single-subject design, which is more appropriate than group design for examining change in individuals over time; this design also

is suited to heterogeneous populations (for details about this methodology, see Sohlberg & Mateer, pp. 47-56). The outcome measures for such studies could be the CCT (given that it satisfies the conditions above), as well as various types of functional outcomes which appear to be affected by cognitive competency (e.g., work performance, ability to independently accomplish tasks at home and in the community). Such research would determine whether improvements in initiation, inhibition, insight, and disorganization syndrome features would generalize to the real-life setting.

Continued research focusing on the cognitive, or neuropsychological, underpinnings of the three syndromes of schizophrenia offers the potential for much greater understanding of the phenomenology of the disease. The results of this study corroborate others in reinforcing the notion of the heterogeneity of schizophrenia. Specifically, evidence is accumulating in favour of psychomotor poverty, disorganization, and reality distortion as separate dimensions, with distinct underlying brain and neuropsychological features. Therefore, the utility of future research related to the levels of understanding (and, in fact, the utility of all schizophrenia research) would be maximized by investigating the pathological, cognitive, and functional correlates of these three syndromes, rather than "positive" and "negative" dimensions or the category of "schizophrenia" as a whole.

This study has shown that certain executive functioning measures derived from unstructured tasks can predict a portion of cognitive competency. Furthermore, limited potential was demonstrated for unstructured tasks to contribute to the assessment of the executive deficits associated with schizophrenia syndromes. Further research in this area might continue to explore these possibilities, using different scores to assess initiation and inhibition, as well as employing different unstructured tasks.

References

- Allen, H. A., Liddle, P. F., & Frith, C. D. (1993). Negative features, retrieval processes and verbal fluency in schizophrenia. British Journal of Psychiatry, *163*, 769-775.
- Amador, X. F., Flaum, M., Andreasen, N. C., Strauss, D. H., Yale, S. A., Clark, S. C., & Gorman, J. M. (1994). Awareness of illness in schizophrenia and schizoaffective and mood disorders. Archives of General Psychiatry, *51*, 826-836.
- Amador, X. F., & Kronengold, H. (1998). The description and meaning of insight in psychosis. In X.F. Amador & A. S. David (Eds.), Insight and psychosis (pp. 15-32). New York: Oxford University Press.
- Amador, X. F., & Strauss, D. H. (1990). The Scale to assess Unawareness of Mental Disorder (SUMD). Columbia University and New York State Psychiatric Institute.
- Amador, X. F., Strauss, D. H., Yale, S. A., Flaum, M. M., Endicott, J., & Gorman, J. M. (1993). Assessment of insight in psychosis. American Journal of Psychiatry, *150*, 873-879.
- Amador, X. F., Strauss, D. H., Yale, S. A., & Gorman, J. M. (1991). Awareness of illness in schizophrenia. Schizophrenia Bulletin, *17*, 113-132.
- American Psychiatric Association. (1987). Diagnostic and statistical manual of mental disorders (3rd ed., revised). Washington, DC: Author.
- American Psychiatric Association. (1994). Diagnostic and statistical manual of mental disorders (4th ed.). Washington, DC: Author.
- Ammons, R. B., & Ammons, C. H. (1962). The Quick Test (QT): Provisional manual. Missoula, Montana: Psychological Test Specialists.
- Andreasen, N. C. (1983). The Scale for the Assessment of Negative Symptoms (SANS). Iowa City: The University of Iowa Press.
- Andreasen, N. C. (1984). The Scale for the Assessment of Positive Symptoms (SAPS). Iowa City: The University of Iowa Press.
- Andreasen, N. C. (1989a). The Scale for the Assessment of Negative Symptoms (SANS): Conceptual and theoretical foundations. Symposium: Negative symptoms in schizophrenia. British Journal of Psychiatry, *155*(Suppl. 7), 49-52.

- Andreasen, N. C. (1989b). Scale for the Assessment of Negative Symptoms (SANS). Symposium: Negative symptoms in schizophrenia. British Journal of Psychiatry, 155(Suppl. 7), 53-58.
- Andreasen, N. C., & Grove, W. M. (1986). Evaluation of positive and negative symptoms in schizophrenia. Psychiatrie et Psychobiologie, 1, 108-121.
- Arndt, S., Alliger, R. A., & Andreasen, N. C. (1991). The distinction of positive and negative symptoms: the failure of a two-dimensional model. British Journal of Psychiatry, 158, 317-322.
- Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, & statistical considerations. Journal of Personality and Social Psychology, 51, 1173-1182.
- Barr, W. B. (1998). Neurobehavioral disorders of awareness and their relevance to schizophrenia. In X. F. Amador & A. S. David (Eds.), Insight and psychosis (pp. 107-141). New York: Oxford University Press.
- Basso, M. R., Nasrallah, H. A., Olson, S. C., & Bornstein, R. A. (1998, February). Negative and disorganized symptoms predict neuropsychological deficit in schizophrenia. Poster presented at the 26th Annual Meeting of the International Neuropsychological Society, Honolulu, HI.
- Baxter, R. D., & Liddle, P. F. (1998). Neuropsychological deficits associated with schizophrenic syndromes. Schizophrenia Research, 30, 239-249.
- Bayless, J. D., Varney, N. R., & Roberts, R. J. (1989). Tinker Toy Test performance and vocational outcome in patients with closed-head injuries. Journal of Clinical and Experimental Neuropsychology, 11, 913-917.
- Bellack, A. S., Morrison, R. L., Mueser, K. T., & Wade, J. (1989). Social competence in schizoaffective disorder, bipolar disorder, and negative and non-negative schizophrenia. Schizophrenia Research, 2, 391-401.
- Benson, D. F. (1993). Prefrontal abilities. Special Issue: The origins of cognitive activity. Behavioural Neurology, 6, 75-81.
- Blumer, D., & Benson, D. F. (1975). Personality changes with frontal and temporal lobe lesions. In D. F. Benson & D. Blumer (Eds.), Psychiatric aspects of neurologic disease (pp. 151-170). New York: Grune & Stratton.

- Bogerts, B., Meertz, E., & Schonfeldt-Bausch, R. (1985). Basal ganglia and limbic system pathology in schizophrenia. Archives of General Psychiatry, 42, 784-791.
- Cicerone, K. D., & Tanenbaum, L. N. (1997). Disturbance of social cognition after traumatic orbitofrontal brain injury. Archives of Clinical Neuropsychology, 12, 173-188.
- Cohen, B. D. (1978). Referent communication disturbances in schizophrenia. In S. Schwartz (Ed.), Language and cognition in schizophrenia (pp. 1-34). Hillsdale, NJ: Lawrence Erlbaum.
- Cohen, J., & Cohen, P. (1983). Applied multiple regression/correlation analysis for the behavioral sciences. Hillsdale, NJ: Lawrence Erlbaum.
- Costello, C. G. (1993). From symptoms of schizophrenia to syndromes of schizophrenia. In C. G. Costello (Ed.), Symptoms of schizophrenia (pp. 291-303) New York: John Wiley & Sons.
- Craig, R. J., & Olson, R. E. (1988). Relationships between Wechsler scales and Quick Test IQs among disability applicants. Professional Psychology Research and Practice, 19, 26-30.
- Crow, T. J. (1980). The molecular pathology of schizophrenia: More than one disease process. British Medical Journal, 280, 66-68.
- Cummings, J. L. (1993). Frontal-subcortical circuits and human behavior. Archives of Neurology, 50, 873-880.
- Dalton, J. E., & Pederson, S. L. (1987). Estimating WAIS-R IQ from the Quick Test. International Journal of Clinical Neuropsychology, 2, 135-136.
- Damasio, A. (1991). Concluding comments. In H. S. Levin, H. M. Eisenberg, & A. L. Benton (Eds.), Frontal lobe function and dysfunction (pp. 401-407). New York: Oxford University Press.
- David, A. S. (1992). Frontal lobology - psychiatry's new pseudoscience. British Journal of Psychiatry, 161, 244-248.
- David, A. S. (1998). The clinical importance of insight. In X. F. Amador & A. S. David (Eds.), Insight and psychosis (pp. 332-351). New York: Oxford University Press.
- DeCato, C. M., & Husband, S. D. (1984). Quick Test and Wechsler Adult Intelligence Scale-Revised in a prison's clinical setting. Psychological Reports, 54, 939-942.

- Dempster, F. N. (1992). The rise and fall of the inhibitory mechanism: Toward a unified theory of cognitive development and aging. Developmental Review, 12, 45-75.
- Dizzonne, M. F., & Davis, W. E. (1973). Relationship between Quick Test and WAIS IQs for brain-injured and schizophrenic subjects. Psychological Reports, 32, 337-338.
- Docherty, N. M., DeRosa, M., & Andreasen, N. C. (1996). Communication disturbances in schizophrenia and mania. Archives of General Psychiatry, 53, 358-364.
- Docherty, N. M., Sledge, W. H., & Wexler, B. E. (1994). Affective reactivity of language in stable schizophrenic outpatients and their parents. The Journal of Nervous and Mental Disease, 182, 313-318.
- Freeman, T., & Gathercole, C. E. (1966). Perseveration - The clinical symptoms in chronic schizophrenia and organic dementia. British Journal of Psychiatry, 112, 27-32.
- Frith, C. D. (1992). The cognitive neuropsychology of schizophrenia. Hillsdale, N.J.: Lawrence Erlbaum.
- Frith, C. D., & Done, D. J. (1988). Towards a neuropsychology of schizophrenia. British Journal of Psychiatry, 153, 437-443.
- Frith, C. D., & Done, D. J. (1989). Experiences of alien control in schizophrenia reflect a disorder in the central monitoring of action. Psychological Medicine, 19, 359-363.
- Frith, C. D., Leary, J., Cahill, C., Johnstone, E. C. (1991). Disabilities and circumstances of schizophrenic patients - a follow-up study. IV. Performance on psychological tests. British Journal of Psychiatry, 159(Suppl. 13), 26-29.
- Goldsamt, L. A., Barros, J., Schwartz, B. J., Weinstein, C., & Naveed, I. (1993). Neuropsychological correlates of schizophrenia. Psychiatric Annals, 22, 151-157.
- Goodglass, H., & Kaplan, E. (1972). Assessment of aphasia and related disorders. Philadelphia: Lea and Febiger.
- Green, M. F. (1996). What are the functional consequences of neurocognitive deficits in schizophrenia? American Journal of Psychiatry, 153, 321-330.
- Green, M. F. (1998). Schizophrenia from a neurocognitive perspective: Probing the impenetrable darkness. Needham Heights, MA: Allyn & Bacon.
- Hammer, M. A., Katsanis, J., & Iacono, W. G. (1995). The relationship between negative symptoms and neuropsychological performance. Biological Psychiatry, 37, 828-830.

- Hammill, D. D., & Larsen, S. C. (1988). Test of Written Language (2nd ed.). Austin: Pro-Ed.
- Harrow, M., Lanin-Kettering, I., Prosen, M., & Miller, J. G. (1983). Disordered thinking in schizophrenia: Intermingling and loss of set. Schizophrenia Bulletin, 9, 354-367.
- Harrow, M., & Prosen, M. (1979). Schizophrenic thought disorder: Bizarre associations and intermingling. American Journal of Psychiatry, 136, 293-296.
- Hogarty, G. E., & Flesher, S. (1992). Cognitive remediation in schizophrenia: Proceed...with caution! Schizophrenia Bulletin, 18, 51-57.
- Jackson, H. J., Minas, I. H., Burgess, P. M., Joshua, S. D., Charisiou, J., & Campbell, I. M. (1989). Negative symptoms and social skills performance in schizophrenia. Schizophrenia Research, 2, 457-463.
- Judd, L. L., McAdams, L., Budnick, B., & Braff, D. L. (1992). Sensory gating deficits in schizophrenia: New results. American Journal of Psychiatry, 149, 488-493.
- Kolb, B., & Whishaw, I. Q. (1980). Fundamentals of human neuropsychology. San Francisco: Freeman.
- Kraepelin, E. (1919). Dementia praecox and paraphrenia (R. M. Barclay, Trans.). Edinburgh: E. & S. Livingstone. (Original work published 1913).
- Lambirth, T. T., & Panek, P. E. (1982). Comparison of the Quick Test and the Stanford-Binet, Form L-M for institutionalized mentally retarded adults. Psychological Reports, 51, 1065-1066.
- Lezak, M. D. (1982). The problem of assessing executive functions. International Journal of Psychology, 17, 281-297.
- Lezak, M. D. (1983). Neuropsychological assessment (2nd ed.). New York: Oxford University Press.
- Lieberman, R. P., & Corrigan, P. W. (1993). Designing new psychosocial treatments for schizophrenia. Psychiatry, 56, 238-249.
- Liddle, P. F. (1995). Inner connections within domain of dementia praecox: Role of supervisory mental processes in schizophrenia. European Archives of Psychiatry and Clinical Neurosciences, 245, 210-215.
- Liddle, P. F. (1996). Functional imaging - Schizophrenia. British Medical Bulletin, 52, 486-494.

- Liddle, P. F. (1987a). Schizophrenic syndromes, cognitive performance and neurological dysfunction. Psychological Medicine, 17, 49-57.
- Liddle, P. F. (1987b). The symptoms of chronic schizophrenia: A re-examination of the positive-negative dichotomy. British Journal of Psychiatry, 151, 145-151.
- Liddle, P. F. (1988, January). Two frontal-subcortical neuronal systems: Implications for schizophrenia. Paper presented at the Fourth Biennial Winter Workshop on Schizophrenia, Badgastein, Austria.
- Liddle, P. F., & Barnes, T. R. E. (1990). Syndromes of chronic schizophrenia. British Journal of Psychiatry, 157, 558-561.
- Liddle, P. F., Barnes, T. R. E., Morris, D., & Haque, S. (1989). Three syndromes in chronic schizophrenia. British Journal of Psychiatry, 155(Suppl. 7), 119-122.
- Liddle, P. F., Friston, K. J., Frith, C. D., Hirsch, S. R., Jones, T., & Frackowiak, R. S. J. (1992). Patterns of cerebral blood flow in schizophrenia. British Journal of Psychiatry, 160, 179-186.
- Liddle, P. F. & Morris, D. L. (1991). Schizophrenic syndromes and frontal lobe performance. British Journal of Psychiatry, 158, 340-345.
- Lysaker, P., & Bell, M. (1994). Insight and cognitive impairment in schizophrenia: Performance on repeated administrations of the Wisconsin Card Sorting Test. Journal of Nervous and Mental Disease, 182, 656-660.
- Lysaker, P., & Bell, M. (1995). Work rehabilitation and improvements in insight in schizophrenia. Journal of Nervous and Mental Disease, 183, 103-106.
- Lysaker, P. H., & Bell, M. D. (1998). Impaired insight in schizophrenia: Advances from psychosocial treatment research. In X. F. Amador & A. S. David (Eds.), Insight and psychosis (pp. 307-316). New York: Oxford University Press.
- Malla, A. K., Norman, R. M. G., Williamson, P., Cortese, L., & Diaz, F. (1993). Three syndrome concept of schizophrenia: A factor analytic study. Schizophrenia Research, 10, 143-150.
- Marengo, J. T., Harrow, M., & Edell, W. S. (1993). Thought disorder. In C. G. Costello (Ed.), Symptoms of schizophrenia (pp. 27-55). New York: John Wiley & Sons.

- Marson, D. C., Cody, H. A., Ingram, K. K., & Harrell, L. E. (1995). Neuropsychological predictors of competency in Alzheimer's disease using a rational reasons legal standard. Archives of Neurology, 52, 955-959.
- Martzke, J. S., Swan, C. S., & Varney, N. R. (1991). Posttraumatic anosmia and orbital frontal damage: Neuropsychological and neuropsychiatric correlates. Neuropsychology, 5, 213-225.
- McEvoy, J. P., Apperson, L. J., Appelbaum, P. S., Ortlip, P., Brecosky, J., Hammill, K., Geller, J. L., & Roth, L. (1989). Insight in schizophrenia. Its relationship to acute psychopathology. Journal of Nervous and Mental Disease, 177, 43-47.
- McFarlane, W. R., & Lukens, E. P. (1998). Insight, families, and education: An exploration of the role of attribution in clinical outcome. In X. F. Amador & A. S. David (Eds.), Insight and psychosis (pp. 317-331). New York: Oxford University Press.
- McGlynn, S. M., & Schacter, D. L. (1989). Unawareness of deficits in neuropsychological syndromes. Journal of Clinical and Experimental Neuropsychology, 11, 143-205.
- McNemar, Q. (1969). Psychological statistics (4th ed.). New York: John Wiley and Sons.
- Mendez, M. F., & Ashla-Mendez, M. (1991). Differences between multi-infarct dementia and Alzheimer's disease on unstructured neuropsychological tasks. Journal of Clinical and Experimental Neuropsychology, 13, 923-932.
- Mlakar, J., Jensterle, J., & Frith, C. D. (1994). Central monitoring deficiency and schizophrenic symptoms. Psychological Medicine, 24, 557-564.
- Mortimer, A. M. (1992). Phenomenology: Its place in schizophrenia research. British Journal of Psychiatry, 161, 293-297.
- Mortimer, A. (1994). Levels of explanation - symptoms, neuropsychological deficit and morphological abnormalities in schizophrenia. Psychological Medicine, 24, 541-545.
- Norman, R. M. G., Malla, A. K., Morrison-Stewart, S. L., Helmes, E., Williamson, P. C., Thomas, J., & Cortese, L. (1997). Neuropsychological correlates of syndromes in schizophrenia. British Journal of Psychiatry, 170, 134-139.
- Parnas, J., & Korsgaard, S. (1982). Epilepsy and psychosis. Acta Psychiatrica Scandinavica, 66, 89-99.

- Penn, D. L., Mueser, K. T., Spaulding, W., Hope, D. A., & Reed, D. (1995). Information processing and social competence in chronic schizophrenia. Schizophrenia Bulletin, 21, 269-281.
- Peralta, V., & Cuesta, M. J. (1994). Lack of insight: Its status within schizophrenic psychopathology. Biological Psychiatry, 36, 559-561
- Peralta, V., de Leon, J., & Cuesta, M. J. (1992). Are there more than two syndromes in schizophrenia? A critique of the positive-negative dichotomy. British Journal of Psychiatry, 161, 335-343.
- Perlick, D., Mattis, S., Stastny, P., & Silverstein, B. (1992). Negative symptoms are related to both frontal and nonfrontal neuropsychological measures in schizophrenia. [Letter to the editor]. Archives of General Psychiatry, 49, 245.
- Pogue-Geile, M. F. (1989). The prognostic significance of negative symptoms in schizophrenia. British Journal of Psychiatry, 155(Suppl. 7), 123-127.
- Randolph, C., Goldberg, T. E., & Weinberger, D. R. (1993). The neuropsychology of schizophrenia. In K. M. Heilman & E. Valenstein (Eds.), Clinical neuropsychology (3rd ed., pp. 499-522). New York: Oxford University Press.
- Rotatori, A. F. (1978). Test-retest reliability of the Quick Test for mentally retarded children. Perceptual and Motor Skills, 46, 162.
- Royall, D. R., Mahurin, R. K., True, J. E., Anderson, B., Brock, I. P., et al. (1993). Executive impairment among the functionally dependent: Comparisons between schizophrenic and elderly subjects. American Journal of Psychiatry, 150, 1813-1819.
- Rubin, P. (1994). Positive and negative symptoms' relation to structural and functional brain changes in schizophrenic patients. Nordic Journal of Psychiatry, 48 (Suppl. 31), 23-27.
- Schindler, B. A., Ramchandani, D., Matthews, M. K., Podell, K. (1995). Competency and the frontal lobe: The impact of executive dysfunction on decisional capacity. Psychosomatics, 36, 400-404.
- Schönemann, P. H. (1990). Facts, fictions, and common sense about factors and components. Multivariate Behavioral Research, 25, 47-51.
- Sohlberg, M. M., & Mateer, C. A. (1989). Introduction to cognitive rehabilitation: Theory & practice. New York: Guilford.

- Stuss, D. T., & Benson, D. F. (1986). The frontal lobes. New York: Raven.
- Suddath, R. L., Christison, G. W., Torrey, E. F., Casanova, M. F., & Weinberger, D. R. (1990). Anatomical abnormalities in the brains of monozygotic twins discordant for schizophrenia. New England Journal of Medicine, 322, 789-794.
- Thompson, P. A. & Meltzer, H. Y. (1993). Positive, negative and disorganization factors from the Schedule for Affective Disorders and Schizophrenia and the Present State Examination. British Journal of Psychiatry, 163, 344-351.
- Traub, G. S., & Spruill, J. (1982). Correlations between the Quick Test and Wechsler Adult Intelligence Scale - Revised. Psychological Reports, 51, 309-310.
- Tune, L. E., Strauss, M. E., Lew, M. F., Brietlinger, E., & Coyle, J. T. (1982). Serum levels of anticholinergic drugs and impaired recent memory in chronic schizophrenic patients. American Journal of Psychiatry, 139, 1460-1462.
- Vance, H., Prichard, K. K., & Jehle, W. O. (1980). Quick Test alternate-form reliability for rural mountain children and youth with learning problems. Psychological Reports, 47, 1109-1110.
- van Os, J., Fahy, T., Jones, P., Harvey, I., Sham, P., Lewis, S., Toone, B., Williams, M., & Murray, R. (1996). Psychopathological syndromes in the functional psychoses: Associations with course and outcome. Psychological Medicine, 26, 161-176.
- Velicer, W. F., & Jackson, D. N. (1990a). Component analysis versus common factor analysis: Some issues in selecting an appropriate procedure. Multivariate Behavioral Research, 25, 1-28.
- Velicer, W. F., & Jackson, D. N. (1990b). Component analysis versus common factor analysis: Some further observations. Multivariate Behavioral Research, 25, 97-114.
- Velligan, D. I., Mahurin, R. K., Diamond, P. L., Hazelton, B. C., Eckert, S. L., & Miller, A. L. (1997). The functional significance of symptomatology and cognitive function in schizophrenia. Schizophrenia Research, 25, 21-31.
- Walker, E., Davis, D., & Baum, K. (1993). Social withdrawal. In C. G. Costello (Ed.), Symptoms of schizophrenia (pp. 227-260). New York: John Wiley & Sons.
- Wang, P. L. (1990). Assessment of cognitive competency. In D. E. Tupper & K. D. Cicerone (Eds.), The neuropsychology of everyday life: Assessment and basic competencies (pp. 219-228). Norwell, MA: Kluwer.

- Wang, P. L. & Ennis, K. E. (1986). Competency assessment in clinical populations: An introduction to the Cognitive Competency Test. In B. Uzzell & Y. Gross (Eds.), Clinical neuropsychology of intervention (pp. 199-133). Boston: Martinus Nijhoff.
- Wang, P. L., Ennis, K., & Copland, S. (1987). Cognitive Competency Test. Toronto: Mount Sinai Hospital, Psychology Department.
- Weinberger, D. R., Aloia, M. S., Goldberg, T. E., & Berman, K. F. (1994). The frontal lobes and schizophrenia. Journal of Neuropsychiatry and Clinical Neurosciences, *6*, 419-427.
- Weinberger, D. R., & Berman, K. F. (1996). Prefrontal function in schizophrenia: Confounds and controversies. Philosophical Transactions of the Royal Society of London: Basic and Biological Sciences, *351* (1346), 1495-1503.
- Weinberger, D. R., & Berman, K. F., & Zec, R. F. (1986). Physiological dysfunction of dorsolateral prefrontal cortex in schizophrenia, I: Regional cerebral blood flow evidence. Archives of General Psychiatry, *43*, 114-124.
- Williamson, P. (1987). Hypofrontality in schizophrenia: A review of the evidence. Canadian Journal of Psychiatry, *32*, 399-404.
- World Health Organization. (1993). The International Classification of Diseases (10th ed.). Geneva: Author.
- Young, D. A., Davila, R., & Scher, H. (1993). Unawareness of illness and neuropsychological performance in chronic schizophrenia. Schizophrenia Research, *10*, 117-124.
- Zahn, T. P., & Carpenter, W. T., Jr. (1978). Effects of short-term outcome and clinical improvement on reaction time in acute schizophrenia. Journal of Psychiatric Research, *14*, 59-68.
- Zec, R. F. (1995). Neuropsychology of schizophrenia according to Kraepelin: Disorders of volition and executive functioning. European Archives of Psychiatry and Clinical Neurosciences, *245*, 216-223.

Appendix A

RESEARCH STUDY INFORMATION

PROJECT TITLE: The neuropsychology of schizophrenia.

INVESTIGATORS: Karin Christensen, M.Sc. (Doctoral student) 721-8595
Catherine Mateer, Ph.D. (supervisor) 721-8590
Richard Williams, M.D. (co-investigator)

Dear patient:

A research project on schizophrenia is currently underway. The study will investigate the causes of some of the symptoms suffered by people with schizophrenia. The study will also help us to understand the consequences of having schizophrenia. We are especially interested in learning about how people with schizophrenia think when they are solving a problem. To learn about these aspects of your illness, we are asking for the participation of any patients who are interested in helping.

Your participation in the project would involve about 2 hours of your time. You will be asked questions about your symptoms. You will also be asked to perform some tasks such as reading, remembering information, making constructions using blocks, and telling stories. Most people find these tasks quite enjoyable. These tasks will be completed at your treatment facility, at a time convenient for you.

If you decide to participate, you are free to withdraw at any time and for any reason.

Your participation in this study will not affect your medical treatment in any way. A decision not to take part, or to withdraw from the study, also will have no impact on your treatment.

Some of the tasks you perform will be audio- or videotaped. These tapes will be erased immediately after your responses have been coded in written form.

If you decide to participate, your privacy will be respected. Your name will not appear in connection with the information that you provide, but will be kept separately and confidentially. Only the investigators identified above, and possibly a research assistant, will have access to identifying information such as names and audio/videotapes. All data will be kept in a locked cabinet within a locked office. Anonymous data will be kept for 5 years after completion of the project, and will then be destroyed by shredding.

There are no risks associated with performing the tasks in this study. On the other hand, although you may enjoy taking part in this research, you will not derive any direct benefit from participating.

If you have any questions about your rights as a participant, you may contact Dr. E. Higgs, Medical Director, at 727-4110.

If you agree to be contacted, the researcher will contact you about being part of this study.
Thank you!

Appendix B

INFORMED CONSENT

PROJECT TITLE: The neuropsychology of schizophrenia.

INVESTIGATORS: Karin Christensen, M.Sc. (Doctoral student) 721-8595
Catherine Mateer, Ph.D. (supervisor) 721-8590
Richard Williams, M.D. (co-investigator)

Written Consent:

I, (name) _____, have read the "research study information," and understand fully the nature and purpose of the study in which I have been asked to take part. The explanation I have been given has indicated that there are no risks or benefits associated with my participation. I understand that I can withdraw at any time and that this will not affect my medical treatment. I agree to participate in this study.

Patient's Name: _____

Signature: _____

Witness: _____

Signature: _____

Date: _____

Appendix C

Test Reliability and Validity Data

Quick Test

There is extensive evidence supporting the validity of the QT as a measure of general, and especially verbal, intelligence. High and significant correlations between QT scores and WAIS-R Full Scale IQ have been reported in various populations, including disability applicants (Craig & Olson, 1988), prisoners in psychiatric care (DeCato & Husband, 1984), and undergraduate students (Traub & Spruill, 1982). The correlation coefficients in these studies tend to fall in the range between 0.65 and 0.90. The QT appears to be more strongly related to Verbal IQ than to Performance IQ (e.g., DeCato & Husband, 1984). Dizzone and Davis (1973) found that QT IQs were highly correlated with WAIS Full Scale IQs (0.80, 0.79) and WAIS Verbal IQs (0.86, 0.79) in separate groups of brain-injured and schizophrenic male subjects. These researchers concluded that the QT provides a valid, reliable estimate of intelligence which is useful in variable populations. Although there are limited data, the QT's reliability has generally been supported (Dizzone & Davis, 1973; Rotatori, 1978; Vance, Prichard, & Jehle, 1980).

SANS and SAPS

Psychometric studies of these instruments have supported their interrater reliability in diverse cultural settings (Andreasen & Grove, 1986). In addition, internal consistency was found to be quite high for the SANS, and lower for the SAPS (meaning that negative symptoms are more highly related to one another than are positive symptoms), as judged by correlations of individual items with subscale scores or overall positive symptom measures. Coefficient alpha ranged from 0.63 to 0.83 for the SANS, and from 0.66 to 0.79 for the SAPS (Andreasen & Grove, 1986). For the SANS, validity is supported by the high values of Cronbach's alpha (Andreasen, 1989). No other reports pertaining to validity are available to date.

Mammoth Hunt Picture

The reliability of the five TOWL-2 subtest scores in which the written story is used, averaged across ages, have been reported as follows (Hammill & Larsen, 1988). Interrater reliability ranges from 0.94 to 0.98; internal consistency coefficients range from 0.75 to 0.96; test-retest reliability with alternate test forms ranges from 0.61 to 0.74; and in summary, all reliability coefficients assessing interrater, content sampling, and time sampling exceed 0.80.

Cognitive Competency Test

A pilot study (Wang & Ennis, 1986) evaluated the CCT's ability to differentiate an

independent living group from a dependent living group. Eleven of the 12 subcategories of test items discriminated the two groups at the 0.01 or 0.05 levels. In addition, the ATS was one of the most robust variables in differentiating the two groups. The ATS correlated significantly with all subcategories of the CCT, which themselves shared common variance; these features of the CCT suggest that some general cognitive ability level is required to perform each subtest, and that the CCT may indeed measure a common cognitive skill. These data provide preliminary evidence for the CCT's validity. Based on this pilot study and clinical impressions, Wang and Ennis (1986) adopted criteria for classifying CCT performance into three functional categories, ranging from totally independent to totally dependent.

A preliminary study of test-retest reliability, using a subset of the normative sample of healthy older adults, revealed no significant differences in the scores obtained during two test sessions on any variables except those involving verbal memory. For the latter item groups, the scores upon retesting were significantly higher than on initial testing, due to practice effects.

Scale to Assess Unawareness of Mental Disorder

A study of the psychometric properties of this scale (Amador et al., 1993) revealed interrater intraclass correlation coefficients (ICCs) of 0.89, 0.75, and 0.68 for current awareness items 1 to 3, respectively. ICCs for the retrospective items were 0.78, 0.89, and 0.67, respectively. The three general items assessing insight into current illness had moderate to strong correlations with insight ratings on two other measures. These ranged from 0.43 to 0.89, all being significant at the 0.05 or 0.001 levels. Past awareness had weaker convergent validity (correlation coefficients ranging from 0.05 to 0.60).

Appendix D

PATIENT NAME:	PIN #:	DATE: <u> </u> / <u> </u> / <u> </u> D M Y	RATER:
------------------	--------	---	--------

SCALE FOR THE ASSESSMENT OF NEGATIVE SYMPTOMS (SANS)

0=None 1=Questionable 2=Mild 3=Moderate 4=Marked 5=Severe

AFFECTIVE FLATTENING OR BLUNTING

- | | | |
|---|--|-------------|
| 1 | Unchanging Facial Expression
The patient's face appears wooden - changes less than expected as emotional content of discourse changes. | 0 1 2 3 4 5 |
| 2 | Decreased Spontaneous Movements
The patient shows few or no spontaneous movements, does not shift his or her position, move extremities, etc. | 0 1 2 3 4 5 |
| 3 | Paucity of Expressive Gestures
The patient does not use hand gestures, body position, etc. as an aid in expressing his or her ideas. | 0 1 2 3 4 5 |
| 4 | Poor Eye Contact
The patient avoids eye contact or "stares through" interviewer even when speaking | 0 1 2 3 4 5 |
| 5 | Affective Nonresponsivity
The patient fails to laugh or smile when prompted | 0 1 2 3 4 5 |
| 6 | Inappropriate Affect
The patient's affect is inappropriate or incongruous, not simply flat or blunted | 0 1 2 3 4 5 |
| 7 | Lack of Vocal Inflections
The patient fails to show normal vocal emphasis patterns, is often monotonic | 0 1 2 3 4 5 |
| 8 | Global Rating of Affective Flattening
This rating should focus on overall severity of symptoms, especially unresponsiveness, eye contact, facial expression, and vocal inflections | 0 1 2 3 4 5 |

ALOGIA

- | | | |
|----|--|-------------|
| 9 | Poverty of Speech
The patient's replies to questions are restricted in <u>amount</u> , tend to be brief, concrete, unelaborated | 0 1 2 3 4 5 |
| 10 | Poverty of Content of Speech
The patient's replies are adequate in amount but tend to be vague, overconcrete, or overgeneralized, and convey little in information | 0 1 2 3 4 5 |
| 11 | Blocking
The patient indicates, either spontaneously or with prompting, that his or her train of thoughts was interrupted | 0 1 2 3 4 5 |
| 12 | Increased Latency of Response
The patient takes a long time to reply to questions, prompting indicates the patient is aware of the question | 0 1 2 3 4 5 |
| 13 | Global Rating of Alogia
The core features of alogia are poverty of speech and poverty of content | 0 1 2 3 4 5 |

AVOLITION - APATHY

14. **Grooming and Hygiene** 0 1 2 3 4 5
The patient's clothes may be sloppy or soiled, and he or she may have greasy hair, body odor, etc.
15. **Impersistence at Work or School** 0 1 2 3 4 5
The patient has difficulty seeking or maintaining employment, completing school work, keeping house etc. If an inpatient, cannot persist at ward activities, such as OT, playing cards, etc.
16. **Physical Anergia** 0 1 2 3 4 5
The patient tends to be physically inert. He or she may sit for hours and not initiate spontaneous activity.
17. **Global Rating of Avolition - Apathy** 0 1 2 3 4 5
Strong weight may be given to one or two prominent symptoms if particularly striking.

ANHEDONIA - ASOCIALITY

18. **Recreational Interests and Activities** 0 1 2 3 4 5
The patient may have few or no interests. Both the quality and quantity of interests should be taken into account.
19. **Sexual Activity** 0 1 2 3 4 5
The patient may show decrease in sexual interest and activity, or enjoyment when active.
20. **Ability to Feel Intimacy and Closeness** 0 1 2 3 4 5
The patient may display an inability to form close or intimate relationships, especially with opposite sex and family.
21. **Relationships with Friends and Peers** 0 1 2 3 4 5
The patient may have few or no friends and may prefer to spend all of his or her time isolated.
22. **Global Rating of Anhedonia - Asociality** 0 1 2 3 4 5
This rating should reflect overall severity, taking into account the patient's age, family status, etc.

ATTENTION

23. **Social Inattentiveness** 0 1 2 3 4 5
The patient appears uninvolved or unengaged. He or she may seem "spacey"
24. **Inattentiveness During Mental Status Testing** 0 1 2 3 4 5
Tests of "serial 7s" (at least five subtractions) and spelling "world" backwards (score 2 = 1 error, score 3 = 2 errors, score 4 = 3 errors)
25. **Global Rating of Attention** 0 1 2 3 4 5
This rating should assess the patient's overall concentration, clinically and on tests

PATIENT NAME:	PIN#:	DATE: <u> </u> / <u> </u> / <u> </u> D M Y	RATER:
------------------	-------	--	--------

SCALE FOR THE ASSESSMENT OF POSITIVE SYMPTOMS (SAPS)

0=None 1=Questionable 2=Mild 3=Moderate 4=Marked 5=Severe

HALLUCINATIONS

- | | |
|---|-------------|
| 1. Auditory Hallucinations
The patient reports voices, noises, or other sounds that no one else hears | 0 1 2 3 4 5 |
| 2. Voices Commenting
The patient reports a voice which makes a running commentary on his/her behaviour or thoughts. | 0 1 2 3 4 5 |
| 3. Voices Conversing
The patient reports hearing two or more voices conversing | 0 1 2 3 4 5 |
| 4. Somatic or Tactile Hallucinations
The patient reports experiencing peculiar physical sensations in the body. | 0 1 2 3 4 5 |
| 5. Olfactory Hallucinations
The patient reports experiencing unusual smells which no one else notices | 0 1 2 3 4 5 |
| 6. Visual Hallucinations
The patient sees shapes or people that are not actually present. | 0 1 2 3 4 5 |
| 7. Global Rating of Hallucinations
This rating should be based on the duration and the severity of the hallucinations and their effects on the patient's life | 0 1 2 3 4 5 |

DELUSIONS

- | | |
|--|-------------|
| 8. Persecutory Delusions
The patient believes he is being conspired against or persecuted in some way. | 0 1 2 3 4 5 |
| 9. Delusions of Jealousy
The patient believes his/her spouse (significant other) is having an affair with someone | 0 1 2 3 4 5 |
| 10. Delusions of Guilt or Sin
The patient believes that he or she has committed some terrible sin or done something unforgivable | 0 1 2 3 4 5 |
| 11. Grandiose Delusions
The patient believes he or she has special powers or abilities. | 0 1 2 3 4 5 |
| 12. Religious Delusions
The patient is preoccupied with false beliefs of a religious nature | 0 1 2 3 4 5 |
| 13. Somatic Delusions
The patient believes that somehow his or her body is diseased, abnormal, or changed | 0 1 2 3 4 5 |
| 14. Delusions of Reference
The patient believes that insignificant remarks or events refer to him or her or have special meaning | 0 1 2 3 4 5 |
| 15. Delusions of Being Controlled
The patient feels that his or her feelings or actions are controlled by some outside force | 0 1 2 3 4 5 |
| 16. Delusions of Mind Reading
The patient feels that people can read his or her mind or know his or her thoughts | 0 1 2 3 4 5 |
| 17. Thought Broadcasting
The patient believes that his or her thoughts are broadcasted so that he/she or others can hear them | 0 1 2 3 4 5 |

- 18 **Thought Insertion** 0 1 2 3 4 5
The patient believes that thoughts that are not his or her own have been inserted into his or her head
- 19 **Thought Withdrawal** 0 1 2 3 4 5
The patient believes that thoughts have been taken away from his or her mind.
- 20 **Global Rating of Delusions** 0 1 2 3 4 5
This rating should be based on the duration and persistence of the delusions and their effect on the patient's life

BIZARRE BEHAVIOUR

- 21 **Clothing and Appearance** 0 1 2 3 4 5
The patient dresses in an unusual manner or does other strange things to alter his or her appearance.
- 22 **Social and Sexual Behaviour** 0 1 2 3 4 5
The patient may do things inappropriate according to usual social norms (e.g. masturbating in public)
- 23 **Aggressive and Agitated Behaviour** 0 1 2 3 4 5
The patient may behave in an aggressive, agitated manner, often unpredictably.
- 24 **Repetitive or Stereotyped Behaviour** 0 1 2 3 4 5
The patient develops a set of repetitive actions or rituals that he or she must perform over and over.
- 25 **Global Rating of Bizarre Behaviour** 0 1 2 3 4 5
This rating should reflect the type of behaviour and the extent to which it deviates from social norms.

POSITIVE FORMAL THOUGHT DISORDER

- 26 **Derailment** 0 1 2 3 4 5
A pattern of speech in which ideas slip off track onto ideas obliquely related or unrelated.
- 27 **Tangentiality** 0 1 2 3 4 5
Replying to a question in an oblique or irrelevant manner
- 28 **Incoherence** 0 1 2 3 4 5
A pattern of speech which is essentially incomprehensible at times.
- 29 **Illogicality** 0 1 2 3 4 5
A pattern of speech in which conclusions are reached which do not follow logically
- 30 **Circumstantiality** 0 1 2 3 4 5
A pattern of speech which is very indirect and delayed in reaching its goal idea.
- 31 **Pressure of Speech** 0 1 2 3 4 5
The patient's speech is rapid and difficult to interrupt, the amount of speech produced is greater than that considered normal
- 32 **Distractible Speech** 0 1 2 3 4 5
The patient is distracted by nearby stimuli which interrupt his or her flow of speech
- 33 **Clanging** 0 1 2 3 4 5
A pattern of speech in which sounds rather than meaningful relationships govern word choice
- 34 **Global Rating of Positive Formal Thought Disorder** 0 1 2 3 4 5
This rating should reflect the frequency of abnormality and degree

Appendix E

Guidelines for Scoring MHP Transcripts

From audiotape, type subject's response, word for word. Begin after researcher has ensured that S understands what to do; researcher will say "OK" as a cue to begin when picture is presented. Stop transcribing as soon as researcher interrupts to tell S to stop.

Write "..." to indicate pauses of about one second or more.

Include all vocalizations (e.g., coughs, throat clearing, laughing) *except* "um" and "uh"

Include comments during timing that are not story-related (e.g., "I'm running out of things to say.")

Include neologisms.

Word count: count words as indicated above, except incomplete words.

Transcribing fast or unclear speech:

Only write separate words if they are clearly heard. For example, many instances will be transcribed "they're" BUT if "they are" was heard, then separate the words in the transcript.

Similarly, write "gonna" and "I dunno" if these are heard rather than the complete words.

If a word is not heard in its entirety, but seems to fit the sentence and does not seem to be a neologism, then write the whole word in the transcript, e.g., write "...they're not bothering with him" when "with" actually sounded like "wi" or "wid".

Often hear "an" or "n" when S intends "and" - in these cases, write the whole word "and".

If a word is clearly incomplete/broken off, then write the spoken part of the word with a dash where it was broken, e.g., "...the North American *pla-* continent..."

If a part of the tape is totally incomprehensible, then write "???" and do not count as a word.

Missing information references (MIRs):

References which seem to assume that the listener has prior information that he/she does not have, and should not be expected to have. References to things unknown to the listener. In other words, a MIR is a noun phrase that 1) refers to something *not seen* in the picture, and 2) *would require additional information* to be understood. This information is not provided, either in the verbal or the situational context.

MIRs: 2 types and examples:

1. Unqualified references to persons, places, or things unknown to the listener:

I like to work all right. Some of *those shops* were filthy. I liked *the bakeries*, some of *the shops* are clean. (No prior mention of any shops or bakeries.)

They let *George* go home, so why not me? (No prior mention of George, and listener does not know him.)

A donkey was crossing *the other river*. (No prior mention of any river.)

2. Comparative references for which the basis of comparison is unknown:

I love the place best in the winter. It's just a total *opposite* then. (Missing information comparison; "opposite" in what way? Listener does not know the basis of comparison.)

References to features of the picture cannot be scored as MIRs, since the S can reasonably assume that the listener has prior knowledge of them. However, if S refers to something which involves some departure from objective features of the picture and cannot be understood using the information given, these should be scored as MIRs. Example from MHP: "There are weapons" *is not* a MIR, since spears are clearly depicted; "There is a vicious game" (with no prior mention of any game) *is* a MIR since the listener would require more information to understand. Similarly, references to mountains/valleys *are not* MIRs; but references to sacrifices *are* MIRs.

MIRs are *distinct from* the type of unclear reference which could refer to one of at least two clear-cut alternative referents; this type of unclear reference is often a pronoun, e.g., "*They* tried to stop *them* and then *they* ran away." (The pronouns are ambiguous in that they can refer to either the people or the mammoths in the picture.)

Intermingling:

Scored when it appears that the S has blended material from his/her own experience into the response, or when the S's response was obviously influenced by these experiences. Intermingled material is *disruptive/intrusive* to the flow of the story (does not "fit" neatly), and adds a *bizarre, inappropriate, or idiosyncratic* element to the speech.

Intermingling examples:

1. "...he's a big tough woolly mammoth, and that would be his wife and his baby, *'cause of course they have a nuclear family.*"

2. In response to a task involving the explanation of proverbs:

(Q) Don't cast pearls before swine.

(A) Don't give your good things to bad people. They might turn around and use your good things to make you sad. *That's a good poem for this hospital 'cause people are depressed.*

Intermingling examples (cont'd):

3. "...there's no building or manmade stuff to *make this beautiful scene sterile...*"

4. The S may become overtly self-referential and used pronouns such as "I" or "my" (BUT if the whole story is in first person, don't use this rule alone):

"I just don't think a mammoth could be that large; I've never heard of that."
 "I don't see why the people in the picture are unhappy."

5. Less direct references to the self, or instances in which the content appears to be of personal relevance but is stated more generally (i.e., second or third person statement) are also scored as intermingling:

"You shouldn't switch decisions or switch friendships with another just because he seems friendly - he might not be. May only be pretending. Stand your ground."

Perseveration:

1. Inappropriate repetitions of words, groups of words, phrases, or ideas.

Example: "...down by the *by the...*"
 "It was really...*really* dark and..."

Score expressions of the same idea as perseverations, even when they are not consecutive.)

Example: "I don't know what else I can tell you" and later: "There's not much else to say"
 Or: "That's it" and later: "That's about all"

Score when the word form is changed but the idea is the same.

Example: "...it's apparent that- *apparently...*"

Do not score when words are repeated but the idea associated with them has been changed.

Examples: "...they moved- they tried to move the furniture..."
 "...after- just after..."

Do not score *appropriate* repetitions.

Example: "They were very very happy." (Look for no pause between the 2 "very"s.)

BUT if there are *more than 2* utterances of the same word, score perseveration starting with the third utterance of the word.

2. Clanging: inappropriate intrusion of rhyming words or non-words, in consecutive order.

Example: "...wearing a hat *bat dat...*"

** The same words/phrase can have more than one score associated with it (e.g., can be scored as both intermingling and MIR).

Appendix F

Guidelines for Tinkertoy Test ScoringFalse starts:

Scored if S *touches* a piece, but does not pick it up;
also scored if S *picks it up briefly* and then puts it down again in or very near the same location.

NOT scored if S moves a piece in any direction.

NOT scored if S seems to be moving pieces out of the way.

Disconnections:

Scored if a piece was clearly attached and is then disconnected. Also, S must have let go of the piece after attaching it (don't score if S's hand is still on the piece and they then disconnect it).

NOT scored if the pieces did not fit together properly, or if they weren't clearly attached.

NOT scored if a piece drops off or falls out.

Odd actions:

Should be bizarre and not task-related, e.g., placing a piece on S's head; speaking to construction; placing a piece exceptionally far away on the table.

Appropriateness:

If S makes more than one construction, and one or more are named inappropriately, score the name as *inappropriate*. (To score as appropriate, all constructions must match their names.)

If S does not name their construction, score as inappropriate.

If the name given is bizarre (e.g., "a device to communicate with Martians"), score as inappropriate.

If a response is queried by examiner, score only the name given before the query.

Complexity score:

Movement: working wheels/moving part as *part of* a construction.

Symmetry: 2 symmetrical halves or 4 symmetrical quadrants.

3-D: must have pieces built on top of each other.

Free-standing: able to stand unassisted, even if precarious.

In cases with >1 construction: all constructions must meet a criterion in order to give the score.

III. PICTURE INTERPRETATION:

2,1,0

1. CHRISTMAS i. socializing ii. Christmas time	2. WINDOW i. boy broke window ii. girl wrongly accused
3. DROWNING i. person in trouble ii. others to aid.	4. HUNTER i. dog should ... ii. changed to ...
5. BONE i. dog chases bone ii. harmful consequences	

P.I.
max 10

IV. MEMORY: 0, 1/2

V. PRACTICAL READING SKILLS:

0,1

1. GROCERY LIST	IM	DE
SOAP	<input type="checkbox"/>	<input type="checkbox"/>
CHICKEN	<input type="checkbox"/>	<input type="checkbox"/>
TOMATOES	<input type="checkbox"/>	<input type="checkbox"/>
CEREAL	<input type="checkbox"/>	<input type="checkbox"/>
2. PRICES		
() BUS	<input type="checkbox"/>	<input type="checkbox"/>
() STAMPS	<input type="checkbox"/>	<input type="checkbox"/>
3. APPOINTMENT		
DENTIST	<input type="checkbox"/>	<input type="checkbox"/>
WEDNESDAY	<input type="checkbox"/>	<input type="checkbox"/>
3:30	<input type="checkbox"/>	<input type="checkbox"/>
CENTRAL CLINIC	<input type="checkbox"/>	<input type="checkbox"/>

1. HALL	<input type="checkbox"/>
2. GREEN APPLES	<input type="checkbox"/>
3. AMBULANCE	<input type="checkbox"/>
4. NORTHBOUND	<input type="checkbox"/>
5. STORE ENTRANCE	<input type="checkbox"/>
6. MOVIE PRICE	<input type="checkbox"/>
7. OFFICES	<input type="checkbox"/>
8. FEB. 26	<input type="checkbox"/>
9. PILLS	<input type="checkbox"/>
10. TO TORONTO	<input type="checkbox"/>

MIM MDE

MEM: max 10

P.R.S.

max 10.

VI. FINANCIAL:

1. SORT		2,1,0
2. TOTAL	0,1	<input type="checkbox"/>
3. PAYABLE	0,2	<input type="checkbox"/>
4. BALANCE	0,1	<input type="checkbox"/>
5. CHEQUE (written)		0,2
6. GROCERY COUPON	0,1	<input type="checkbox"/>
7. CREDIT CARD	0,1	<input type="checkbox"/>

FIN.

max 10.

VII. VERBAL REASONING

2.1.0

1. SMOKING (personal danger)	<input type="checkbox"/>	6. NEIGHBOUR (short; notify)	<input type="checkbox"/>
2. SNOWSTORM (warnch)	<input type="checkbox"/>	7. OPEN DOOR (avoid; notify)	<input type="checkbox"/>
3. GAS (personal safety; responsible action)	<input type="checkbox"/>	8. APPOINTMENT (before 11:00)	<input type="checkbox"/>
4. FOOD (smell, look, taste, texture)	<input type="checkbox"/>	9. SLIPPERY (alternative strategy)	<input type="checkbox"/>
5. CUT (cleanse; protect)	<input type="checkbox"/>	10. BAD DAY (refrigerator-bulb-cup)	<input type="checkbox"/>

V.R.

VIII. ROUTES:

i. LIST (RL1)

0, 1/2

R:L
max 5

ii. LOCATE (RL2)

0, 1

R:LO
max 10

<input type="checkbox"/>	1. SHOPS	<input type="checkbox"/>	6. PARK	<input type="checkbox"/>
<input type="checkbox"/>	2. HOUSE (2nd)	<input type="checkbox"/>	7. CHURCH	<input type="checkbox"/>
<input type="checkbox"/>	3. RESTAURANT	<input type="checkbox"/>	8. HOSPITAL	<input type="checkbox"/>
<input type="checkbox"/>	4. BANK	<input type="checkbox"/>	9. RAILWAY	<input type="checkbox"/>
<input type="checkbox"/>	5. BUS	<input type="checkbox"/>	10. HOUSE (own)	<input type="checkbox"/>

iii. ORIENTATION

	RL3	RL4	RL5	RL6	RL7	
	HOUSE TO BANK	MACS TO HOUSE	HOUSE TO HOSP	MACS TO PARK	PARK TO HOUSE	
REHEARSE FIRST/SECOND	I II	I II	I II	I II	I II	R:O
IMM. RECALL PASS/FAIL	(P) 1 (F) 0	(P) 1 (F) 0	(P) 1 (F) 0	(P) 1 (F) 0	(P) 1 (F) 0	
DIGIT SPAN	542 378 961	821 594 673	481 326 759	958 627 143	281 764 539	max 5
DELAY RECALL PASS/FAIL	(P) 2 (F) 0	(P) 2 (F) 0	(P) 2 (F) 0	(P) 2 (F) 0	(P) 2 (F) 0	
scoring notes:						max 10

iv. PATHFINDING ...

	RL8	RL9	RL10	RL11	RL12	
DIRECT (0, 1)						R:P
ALTER (0, 1/2)						max 5

Appendix H

Awareness of Illness Questionnaire1. Awareness of mental disorder

Do you believe that you have a mental disorder, psychiatric problem, or emotional difficulty?

Have you ever had such a problem in the past?

<u>Current</u>	<u>Past</u>	
0	0	Cannot be assessed.
1	1	Aware: clearly believes that he/she has a mental disorder.
2	2	Somewhat: is unsure whether he/she has a mental disorder, but can entertain the idea.
3	3	Unaware: believes he/she does not have a mental disorder.

2. Awareness of achieved effects of medication

I understand that you are taking some medication. What are the effect of this medication on you?

Do you feel that the medication has lessened the intensity or frequency of your symptoms?

Has medication ever helped you with your symptoms in the past?

<u>Current</u>	<u>Past</u>	
0	0	Cannot be assessed or item not relevant.
1	1	Aware: clearly believes that medication has lessened the intensity/frequency of symptoms.
2	2	Somewhat: is unsure whether medication has lessened the intensity/frequency of symptoms, but can entertain the idea.
3	3	Unaware: believes that medication has not lessened the intensity/frequency of symptoms.

3. Awareness of the social consequences of mental disorder

What do you believe is the reason that you are in hospital/ seeing a psychiatrist regularly?

Have you ever been in hospital/treated by a psychiatrist in the past? What was the reason at that time?

<u>Current</u>	<u>Past</u>	
0	0	Cannot be assessed or item not relevant.
1	1	Aware: clearly believes that the relevant social consequences are related to having a mental disorder.
2	2	Somewhat: is unsure whether the relevant social consequences are related to having a mental disorder.
3	3	Unaware: believes that the relevant social consequences have nothing to do with having a mental disorder.

Descriptive Information for Psychometric and Demographic Measures

	Range	Minimum	Maximum	Mean	SD
Initiation component	4.22	-1.58	2.64	0.00	1.00
MHP # words	384.00	63.00	447.00	197.98	101.54
MHP latency	41.00	1.00	42.00	9.78	11.41
TT # pieces	41.00	9.00	50.00	39.70	12.34
TT latency	30.00	0.00	30.00	5.10	5.88
Inhibition aggregate	0.18	0.01	0.19	0.08	0.04
TT error ratio	0.11	0.00	0.11	0.04	0.02
MHP error ratio	0.12	0.00	0.12	0.04	0.03
Intermingling ratio	0.59	0.00	0.59	0.07	0.15
Psychomotor poverty syndrome	12.25	0.00	12.25	4.76	3.39
Disorganization syndrome	13.00	-1.03	11.97	3.13	2.85
Awareness of illness	12.00	6.00	18.00	8.45	4.00
Cognitive Competency Test score	45.00	51.00	96.00	81.63	10.16
Age (years)	34.00	17.00	51.00	30.75	9.02
Education (years)	13.00	5.00	18.00	11.65	2.30
Onset Age (years)	28.00	15.00	43.00	23.80	6.37
Illness duration (years)	21.00	0.00	21.00	6.95	7.10
Quick Test IQ	38.00	82.00	120.00	98.28	9.49

Note. Initiation component includes MHP # words, MHP latency, TT # pieces, and TT latency. Inhibition aggregate = MHP error ratio + TT error ratio. Awareness of illness = current + retrospective awareness. SD = standard deviation.

Intercorrelations of Psychometric Measures

	Initpca	MHP # words	MHP latency	TT # pieces	TT latency
Initpca	1.00	-0.78 **	0.65 **	-0.43 **	0.74 **
MHP # words	-0.78 **	1.00	-0.41 **	0.27	-0.30
MHP latency	0.65 **	-0.41 **	1.00	0.16	0.33 *
TT # pieces	-0.43 **	0.27	0.16	1.00	-0.30
TT latency	0.74 **	-0.30	0.33 *	-0.30	1.00
Inhibagg	0.16	-0.40 *	0.01	0.05	-0.01
TT error ratio	0.06	-0.33 *	0.01	0.10	-0.16
MHP error r.	0.19	-0.31	0.01	-0.01	0.11
IM ratio	0.04	0.00	-0.27	-0.31	0.14
PPS	0.14	-0.20	0.10	-0.28	-0.12
DS	0.03	-0.09	-0.36 *	-0.43 **	0.05
Awareness	-0.11	-0.09	-0.21	-0.10	-0.24
CCT	-0.39 *	0.38 *	0.08	0.48 **	-0.32 *

	Inhibagg	TT error r.	MHP error r.	IM ratio
Initpca	0.16	0.06	0.19	0.04
MHP # words	-0.40 *	-0.33 *	-0.31	0.00
MHP latency	0.01	0.01	0.01	-0.27
TT # pieces	0.05	0.10	-0.01	-0.31
TT latency	-0.01	-0.16	0.11	0.14
Inhibagg	1.00	0.74 **	0.85 **	0.27
TT error ratio	0.74 **	1.00	0.28	-0.15
MHP error r.	0.85 **	0.28	1.00	0.50 **
IM ratio	0.27	-0.15	0.50 **	1.00
PPS	-0.07	0.10	-0.17	-0.17
DS	0.19	-0.09	0.34 *	0.58 **
Awareness	0.21	0.09	0.23	0.24
CCT	-0.41 **	-0.13	-0.49 **	-0.38 *

	PPS	DS	Awareness	CCT
Initpca	0.14	0.03	-0.11	-0.39 *
MHP # words	-0.20	-0.09	-0.09	0.38 *
MHP latency	0.10	-0.36 *	-0.21	0.08
TT # pieces	-0.28	-0.43 **	-0.10	0.48 **
TT latency	-0.12	0.05	-0.24	-0.32
Inhibagg	-0.07	0.19	0.21	-0.41 **
TT error ratio	0.10	-0.09	0.09	-0.13
MHP error r.	-0.17	0.34 *	0.23	-0.49 **
IM ratio	-0.17	0.58 **	0.24	-0.38 *
PPS	1.00	0.09	-0.09	0.00
DS	0.09	1.00	0.41 **	-0.57 **
Awareness	-0.09	0.41 **	1.00	-0.50 **
CCT	0.00	-0.57 **	-0.50 **	1.00

Note. Initpca = initiation component = MHP # words, MHP latency, TT # pieces, and TT latency; Inhibagg = inhibition aggregate = MHP error ratio + TT error ratio; IM ratio = intermingling ratio; PPS = psychomotor poverty syndrome; DS = disorganization syndrome; Awareness = current + retrospective awareness of illness; CCT = Cognitive Competency Test score. Significance tests are 2-tailed.
* $p < .05$; ** $p < .01$