INHIBITION OF RETURN IN SCHIZOPHRENIA

THESIS

Presented to the Graduate Council of the
University of North Texas in Partial
Fulfillment of the Requirements

For the Degree of

MASTER OF SCIENCE

Ву

Jeffrey D. Hinds, B.S.

Denton, Texas

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The present study was designed to look at inhibition of return within a schizophrenic population for the first time. Inhibition of return is an attentional phenomenon that has been studied with a number of populations, and has been shown to be present in normal individuals. Based on the disattention hypothesis put forth by Cromwell and colleagues (e.g., Cromwell & Dokecki, 1968), it was hypothesized that patients with schizophrenia would show an impaired inhibition of return. Twenty-eight inpatients with schizophrenia, and 19 normal comparisons were evaluated on a visual inhibition of return task. Consistent with hypotheses, schizophrenia patients have significant impairments in inhibition of return compared to normal comparison participants. Further, the relative lack of inhibition of return in the schizophrenic group was found to be strongest to stimuli in the left visual field. results provide initial support for a reconceptualization of the disattention hypothesis.

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CHAPTER I

INTRODUCTION

A primary focus of research with individuals with schizophrenia and those at risk for developing schizophrenia has been attention and information processing (AIP). Noting AIP deficiencies among persons with schizophrenia can be traced back to the time of Bleuler (1950) and Kraeplin (1919). Since that time, investigators have been busy finding methods to quantify these deficiencies. This effort has proven successful in enhancing the understanding of schizophrenia. However, there has not been agreement in this literature as to the underlying processes that might account for the deficiencies in task performance that have been demonstrated in individuals with schizophrenia. This paper will first examine the relevance of information processing studies in gaining understanding of schizophrenia. A review of the literature concerning visual information processing in schizophrenia and the techniques that have commonly been used to measure visual processing deficits will be presented. Next, an hypothesis proposed to explain such deficits will be presented, followed by a description of the task that was used in the present study to examine this hypothesis. Finally, the study's results and implications are discussed.

Due to the realization that schizophrenia evolves over many years prior to the onset of clinical symptoms, there has been a recent push in the schizophrenia literature to identify markers for the disorder (Chapman & Chapman, 1989). Currently, much research in the area is geared towards the "at-risk" populations relative to schizophrenia. Although a clearly superior hypothesis regarding etiology has not emerged, studies have been helpful in identifying those individuals who may be at risk for developing the disorder. For example, much of the research regarding at-risk individuals has come from studies of twins (de Marchi, 1991; Murray, Reveley, & McGuffin, 1986; Torrey, 1987). From these studies, concordance rates have been calculated for monozygotic and dizygotic twins, first-degree relatives of schizophrenia patients and others.

Other research has examined at specific cognitive functions or abilities that appear to deteriorate in individuals with schizophrenia, in hopes that these "markers" will help (a) to indicate imminent onset of the disorder, (b) to predict outcome of the disorder, (c) to represent bridges between the genetic/environmental origins and the later clinical symptoms, and/or (d) to point toward possible prevention of the disorder. Understanding one of these markers alone may not be sufficient for the delineation of the underpinnings of schizophrenia. However, the study of a number of these markers would ideally lead to

a better understanding of the disorder, as well as identify possible interventions for those individuals at risk for developing the disorder. The present study involves a phenomenon--inhibition of return--that could be a candidate for the marker research discussed above.

The term "information processing" has been used to explain the operations of the central nervous system (CNS) in handling stimuli. Various theories have emerged to propose how information processing works. For example, early theories asserted that there are a number of "stages" of processing that a stimulus undergoes. These stages are linked to the various functions of the CNS. information processing was thought to enable an individual to process stimuli in the most accurate and efficient way. Schizophrenia patients, then, were thought to have some malfunction in the early stages of processing, which eventually led to problems in thought, as well as to deficiencies in interacting with the external world. complex theories have since been proposed to explain information processing (Hoffman & Dobscha, 1989; Servan-Schreiber & Cohen, 1992), due to questions raised concerning the stage model (Edelman, 1987).

To summarize, studies examining information processing have shown that schizophrenics' deficits in processing are intensified when such things as multiple tasks and distractors require rapid processing of information (Dawson

& Nuechterlein, 1984; Kietzman, Spring, & Zubin, 1985). In addition, similar deficits have been identified in other schizophrenia-related groups, such as schizotypal patients, unaffected family members, and high-risk children (Siever, 1991).

Techniques Used to Assess Deficits in Processing

A number of the techniques used to measure information processing and attentional deficits will be discussed below. This discussion will by no means exhaust the techniques being utilized currently or in the past. However, those techniques discussed below have been used more widely and thus, have been the foci of more extensive reviews on the topic (cf., Braff, 1985; Braff, 1993; Dawson & Nuechterlein, 1984).

The term "gating" has been used to describe the inability of schizophrenia patients to "gate" or screen out irrelevant stimuli. Gating deficits of individuals with schizophrenia have been measured through the use of two different techniques: (1) gating of the human startle response; and (2) the gating of the P50 evoked response potential (ERP). In the first technique, a startle response is elicited by a sudden intense stimulus, such as a loud tone or a bright light. When the intense stimulus is preceded by a similar stimulus of weaker magnitude, however, the startle response is inhibited (i.e., not as strong). This phenomenon is known as prepulse inhibition, and is

present when the weak pre-stimulus precedes the intense stimulus by 30 to 500 milliseconds. Prepulse inhibition has been shown to be impaired in schizophrenic patients, who, despite the prestimulus, exhibit strong startle responses (Braff et al., 1978, 1992). The ability of the prestimulus to induce a buffering effect in schizophrenic participants, is extremely poor when the interval between the two stimuli exceeds 60 to 120 milliseconds.

Analogous to prepulse inhibition, the P50 ERP paradigm has shown that individuals with schizophrenia show a loss of a normal inhibitory process (Freedman et al. 1983). The procedure involves presenting two rapid-click stimuli separated by 500 milliseconds, and recording the observed response of the P50 wave, which is a specific electroencephalogram (EEG) wave elicited by an auditory evoked potential. In normal participants, the response to the first stimulus is quite large, but the response to the second stimulus is attenuated by the presentation of the first stimulus. Again, this attenuation effect is relatively lacking in schizophrenia.

Another area of interest in schizophrenia is performance on measures of ocular motor function. There is evidence that schizophrenic patients display abnormalities on such tasks (Iacono & Clementz, 1993). These deficits have also been shown to correlate with other markers of schizophrenia, as well as clinical symptoms. Probably one

of the most consistent findings in this area is that individuals with schizophrenia tend to perform poorly on an eye-tracking task. The task most commonly used to measure eye tracking involves measuring a subject's eye movements while following a swinging pendulum. Schizophrenia patients lack smooth-pursuit tracking (Abel et al., 1991; holzman, 1987); that is, their eye movements while following the pendulum tend to be rather discontinuous or "choppy". Smooth-pursuit eye movement dysfunctions have also been found in first-degree relatives of schizophrenia patients (Iacano & Clementz, 1993). Furthermore, among the major psychotic disorders, smooth-pursuit eye movement dysfunction has been shown to be specific to schizophrenia (Muir et al., 1992).

Two additional techniques that have often been utilized, and extensively reviewed are: (1) span of apprehension, and (2) backward masking. The task most typically used in span of apprehension involves requiring participants to identify target letters in a quickly presented visual stimulus array. The target letter is most commonly the letter T or F. Participants are asked to look into a tachistoscope, or to view a computer monitor, on which stimuli are presented in brief exposures. The stimulus array may or may not contain the target letter; the subject is instructed to report the target letter when it is present. Both normal comparison participants and

schizophrenia patients show reduced hit-rates on this measure as the number of non-relevant stimuli in the array increases; however, schizophrenia patients show a dramatic deficit across a number of tasks that tap this function, especially when the number of stimuli in the array is large (Asarnow, Granholm, & Sherman, 1991).

Visual backward masking also uses tachistoscopic or computer-driven stimuli. This task involves presenting a stimulus, again usually a target letter, and then presenting an intense but meaningless stimulus (e.g., a series of overlapping lines) at variable intervals following the target presentation. The meaningless stimulus is found to interfere with the subject's ability to identify the target. When the masking stimulus follows the initial presentation by 100 milliseconds or less, performance is only at chance levels. As the interval increases, participants' ability to recognize the target stimulus increases until it reaches the level that would be obtained if no masking stimulus was presented. Schizophrenia patients continue to show deficits, however, when the masking stimulus is presented up to 350 milliseconds after the target presentation (Saccuzzo & Braff, 1981; Saccuzzo, Hirt, & Spencer, 1974).

Recently, an interesting study by Elkins and Cromwell (1992) examined span of apprehension in a group of schizophrenic patients as a function of distractor masking. The task involved the typical span of apprehension task

discussed earlier; however, in this case the distractor stimuli were backward masked. Because studies of backward masking have indicated that the masking of target letters tends to result in impaired performances, the authors examined the effects of masking the distractors rather than the targets. Results of the study demonstrated that backward masking of the distractor letters did not improve schizophrenic patients' performances, but actually made them worse. The authors suggested that the performance of participants with schizophrenia might be related to an adaptive attentional process that is deficient in schizophrenia. Such a process is necessary to eliminate attention to prior information in order for a person to be ready to process new information. Masking of the irrelevant elements in this paradigm seems to aid the attentional shift in normal and depressed individuals because they scan for a target letter in a serial manner. The authors suggested the possibility that masking directs attention away from the target letter or adds new (irrelevant) information from which to be disengaged. Because individuals with schizophrenia have been described as being deficient in the adaptive function of disattending (Cromwell & Dokecki, 1968; Salzinger, 1971), the masking may create increased difficulties. The relevance of the disattention function for the present study will be examined in greater detail later in the present paper.

Studies of simple reaction time have shown that schizophrenia patients and normals differ in their responses to the regularity of the duration of the interval between a warning signal and the signal for them to respond (preparatory interval). The task involves the repeated presentation of a given preparatory interval (PI) over a block of trials (the regular condition), and a second condition involving the same PI randomly presented within a block of trials with PIs of other durations (the irregular condition). Whereas normals tend to display faster performances as the regular condition goes along, schizophrenia patients seem to lose this advantage when the PI is greater than 2 seconds (Huston, Shakow, & Riggs, 1937; Tizard & Venables, 1956). This phenomenon has been termed redundancy-associated deficit (RAD; Bellissimo & Steffy, 1971). Further, some authors (Shakow, 1963; Sutton & Zubin, 1965) have noticed a "crossover" of schizophrenics' performance in regular and irregular conditions. For schizophrenic patients, once the PI is greater than about 4 seconds, they actually show an impaired performance on regular trials rather than reaction times just being reduced to the same level as irregular trials.

On many of the tasks discussed above, the deficits that have been identified in schizophrenia patients, have also been evident in other schizophrenia-related groups. For example, first-degree relatives of schizophrenia patients

who are not symptomatic, have also displayed increased rates of inhibitory and gating failures (Freedman, Adler, Waldo, et al., 1991). Further, first-degree relatives of schizophrenia patients and patients with schizotypal personality disorder, have been found to have abnormal ocular motor performance (Blackwood et al., 1991; Clementz et al., 1992; Holzman et al., 1973; and Siever et al., 1990) Deficits in visual backward masking have been demonstrated in psychosis-prone and schizotypal individuals (Merritt & Balogh, 1990).

The Disattention Hypothesis

More than two decades ago, Cromwell and Dokecki (1968) presented a disattention hypothesis regarding much of schizophrenic behavior. They stated that schizophrenic behavior (particularly speech) might be the result of an inability to disattend from irrelevant sensory input. Consequently, individuals with schizophrenia were unable to disengage from prior events in order to respond appropriately to upcoming ones. This disattention can be thought of in relation to internal stimuli as well as The schizophrenic's inability to disengage sensory input. from internal phenomena, such as ideation experienced as voices and alien thoughts, might account for much of the behavior that is typically present in the active phase of Thus, by failing to disengage from previous the disorder. stimuli, the schizophrenic is able to process only a subset of the stimuli that influence the behavior of those who <u>are</u> able to disattend.

The disattention hypothesis has received some empirical support from various investigations. Salzinger's (1971) work on his immediacy hypothesis revealed schizophrenic behavior to be influenced more by immediate stimulation than by either remote context or a priori instructions and goals. Salzinger's hypothesis that schizophrenia patients respond only to immediate stimuli, internal or external, seems to be compatible with the notion of disattention. Salzinger (1983) describes this mode of responding to stimuli as a tendency to respond to stimuli in seclusion. The failure to respond to stimuli within their context can be shown to account for many schizophrenic symptoms, such as delusions.

Referential communication work in schizophrenia (Cohen, Nachmani, & Rosenberg, 1974) indicated that schizophrenic patients were unable to self-edit and reject an idea once it was sampled. When asked to describe a stimulus so that others would be able to identify it based on the description, normal individuals proceed through a process of selecting possible responses, and then comparing these responses based on their appropriateness. Finally, a choice is made from among these responses, rejecting those deemed not appropriate via self-editing. The authors discussed what they referred to as the perseverative-chaining model of communication in individuals with schizophrenia. The

perseverative portion of the model refers to the failure of a patient to disregard a rejected response to a stimulus, despite the fact that he or she recognizes the poor quality of the response as communication. The chaining component of the model refers to the finding that each successive utterance of a schizophrenic is a response chosen from the patient's pool of possible responses to the just-prior stimulus, not the current stimulus. The primary notion in this model is that schizophrenia patients are unable to disregard an already-sampled response. Thus, the perseverative-chaining model of schizophrenic communication was also viewed as support for disattention.

Mathysse (see Wynne, Cromwell, & Mathysse, 1978, p. 154) used the disattention formulation to explain why methylphenidate (Ritalin) and related amphetamines have opposing effects on the attention deficits of schizophrenia patients versus attention-disordered hyperactive children. Heilbrun et al. (in press) have recently used the disattention notion to study auditory hallucinations.

In spite of these data, the disattention formulation has shown little utility in schizophrenia research, with the sources of empirical support being indirect. More recently, Cromwell (1993) has elaborated on the theoretical position, theorizing that schizophrenia is composed of two interacting dysfunctions: one that leaves the schizophrenic overexcited by the physical intensity of novel stimuli; the

other leaves the schizophrenic over-inhibited by the meaningfulness of redundant stimuli. Cromwell introduced this position in relation to contradictory views put forth by Shakow (1962) and Zubin (1975). These two latter investigators found differences in how attention was characterized in schizophrenics. Shakow stressed the inability of schizophrenia patients to sustain a focus of attention; Zubin focused on their persistence of attention. Similarly, individuals with schizophrenia were described as both over-excitatory (supersensitive) and over-inhibitory. Cromwell proposed "that each tendency exists separately and is under its own genetic and environmental control" (p. 4). To illustrate, in a reaction time procedure it is found that by introducing a stimulus with minor informational value during the forewarning period, deficits in reaction time are found when this period is short (1-3 sec.) and when it is long (9+ sec.), but not when the forewarning period is between the two (5-7 sec.). Cromwell proposed that these early and late deficits represent two independent phenomena, the early associated with the over-excitatory function, and the late with the over-inhibitory function.

Inhibition of Return

Single methodologies have typically not allowed the assessment of the over-excitatory and over-inhibitory dysfunctions separately. However, Posner and his co-workers (Posner, Cohen, Choate, & Vaughn, 1984) have identified and

measured a phenomenon of potential use here. The process has been referred to as "inhibition of return."

Inhibition of return is conceptualized as follows. If an individual processes information from an area A of the visual field, and then the attention is shifted to area B; then, new information may be accessed more easily at a third area C than back at area A. The relative disadvantage in responding to the first area, as compared to the new area, is interpreted as an inhibition at that original location in the visual field. This inhibition occurs with normal participants, and appears to be independent of eye position, competing auditory information, particular area of the visual field, or type of visual stimulus used (Posner & Cohen, 1984; Posner, Cohen, Choate, Hockey, & Maylor, 1984; and Posner, Rafal, Choate, & Vaughn, 1985).

Posner et al. (1984) have demonstrated that patients with certain brain lesions demonstrate a loss of inhibition of return. Specifically, it was found that patients with severe midbrain lesions displayed an absence of inhibition of return. The authors concluded that because inhibition of return appears to be abolished by midbrain lesions, evidence suggests that the phenomena evolved alongside those brain systems implicated in the eye movement system.

The Present Study

The aim of this study was to examine inhibition of return for the first time in schizophrenic patients.

Because schizophrenia patients fail to disengage from repeated information from the same source, it was hypothesized that their inhibition of return would be impaired (of a shorter duration, thus allowing them to return unusually quickly to a previously attended area) compared to normal individuals. The hypotheses of the proposed study were:

- 1) Individuals in the schizophrenic group would show faster reaction times for trials in which the target appears in the same location as the stimulus that was first cued, relative to a third location demonstrating an impaired inhibition of return.
- 2) Individuals in the normal comparison group would show faster reaction times when the target stimulus appears in the location opposite that which was first cued, relative to the cued location demonstrating an inhibition of return.
- 3) The schizophrenic group would differ significantly from the comparison group in the measured inhibition of return.

CHAPTER II

METHOD

Participants

The participants of the proposed study were 28 schizophrenic in-patients and 19 normal comparison participants of ages and genders comparable to that of the clinical populations. Attempts were made to obtain a comparison group that was similar in these respects to the clinical samples; however, due to the nature of these populations, it did not appear feasible to match the groups exactly on these variables. See Table 1 for demographic characteristics of each group.

Participants in the inpatient sample were classified through results obtained from administration of the Schedule of Affective Disorders and Schizophrenia-Change Version (SADS-C; Spitzer & Endicott, 1978). The SADS-C is based upon criteria of the Diagnostic and Statistical Manual Third Edition-Revised (DSM-III-R; APA, 1987). All participants were assessed for history of psychiatric hospitalizations in the family. Normal comparison participants found to have either personal or family history of psychiatric hospitalization were excluded, as were participants with disabilities that precluded valid participation in the experiment.

Table 1

Demographic Characteristics of the Schizophrenia Patients

and Normal Comparison Group

Variable	Schizophrenia			Normal		
<u>Mean Age</u>	37.2			38.2		
<u>Sex</u>						
Male	26	(93%)		18	(95%)	
Female	2	(07%)		1	(05%)	
<u>Race</u>						
White	13	(46%)		13	(68%)	
African-American	10	(36%)		2	(11%)	
Hispanic	5	(18%)		4	(21%)	
<u>Handedness</u>						
Right	28	(100%)		19	(100%)	
Left	0			0		

<u>Instruments</u>

The diagnoses of each participant was determined by the SADS-C mentioned above. The SADS-C is a semi-structured diagnostic interview designed primarily for the assessment of mood and psychotic disorders. The SADS-C consists of 45 symptoms selected from the Schedule of Affective Disorders and Schizoprenia Part I (SADS; Spitzer & Endicott, 1978b). Research has demonstrated the SADS-C to have a high degree of reliability for both symptoms (median ICC = .88) and summary scales (median ICC = .93; McDonald-Scott & Endicott, 1984). In addition, validity studies of the SADS-C have established the discriminability of SADS-C scales for depressed and schizophrenic patients.

The inhibition of return task was conducted through the use of an IBM compatible computer. The computer ran the protocol via a program written by the experimenter. The program was designed to present the necessary stimuli, as well as to record the response and reaction time of the participant for each of the trials.

Inpatients meeting the study criteria of schizophrenia, and who chose to participate, were further evaluated with regards to a number of additional variables. This information was recorded on a demographic/status variables form for each individual participant. These additional variables were: process versus reactive schizophrenia, paranoid versus nonparanoid subclassification, family

history of psychiatric illness, acute versus chronic course, positive symptoms, negative symptoms, medication type, and medication dosage. Positive and negative symptoms among the schizophrenia patients were assessed via the Scale for the Assessment of Positive Symptoms (SAPS; Andreasen, 1983) and the Scale for the Assessment of Negative Symptoms (SANS; Andreasen, 1983). Studies of interrater reliability of the SANS and SAPS have found reliability coefficients to range from .42 to .99 for negative symptoms, and between .63 and .99 for positive symptoms (Andreasen & Flaum, 1991). Validity studies of these scales have also produced good results (e.g., Chaturvedi, 1986).

The most recent Abnormal Involuntary Movement (AIM)

Examination results was recorded from the patients' chart as was the number of days since the AIM was administered. This information was used to determine the presence of any abnormal movements that may have prevented the participants' valid participation in the study. Tables 2 and 3 show summary statistics for the status variables relevant to the schizophrenia group.

Procedure

Individuals were first introduced to the study, at which time those who chose to participate read and signed an informed consent form (see Appendix A). Participants were then interviewed by the investigator, using the SADS-C, to determine the diagnosis of schizophrenia or mood disorder.

Table 2
Summary of Status Variables for Schizophrenia Patients

			Mean (SD)		
Variable	N (9	ኔ)	SAPS	SANS	AIMS
<u>Type</u>					
Process	28	(100%)	36.7 (22.4)	24.5 (16.5)	.12 (.33)
Reactive	0	(0%)			
Subclassificat	<u>ion</u>				
Paranoid	14	(50%)	45.9 (20.8)	24.9 (16.3)	.29 (.49)
Non-Paranoid	14	(50%)	27.6 (20.8)	24.2 (17.2)	0
Family History					
Present	80	(29%)	40.1 (19.0)	29.9 (14.9)	.14 (.38)
Absent	20	(71%)	35.4 (23.9)	22.4 (16.9)	.10 (.32)
<u>Course</u>					
Chronic	27	(96%)	37.5 (22.4)	24.9 (16.7)	.12 (.33)
Acute	1	(4%)	15.0	14.0	0
<u>Total</u>	28		36.7 (22.4)	24.5 (16.5)	.12 (.33)

Table 3

Medication Type and Dose for Schizophrenia Group

Medication Type	N	Average Daily Dose(mg)
Clozaril	5	533
Haldol	4	440
Prolixin	4	101
Navane	3	400
Loxitane	2	-633
Mellaril	1	200
Risperdol	7	5
Klonopin	1	8

Note. All daily dosages are reported in chlorpromazine equivalents with the exception of Risperdol and Klonopin, which could not be converted due to their belonging to a different class of drugs.

Those individuals who met the criteria for either disorder, based on the SADS-C, and whose AIMS tests were within normal ranges (i.e., total AIMS < 2), were then offered the opportunity to participate in the complete protocol.

Before presenting the protocol, participants were assessed for eyesight accuracy using a visual test chart. Assessment of vision accuracy revealed all participants to have adequate vision, and thus, no participants were removed from the study due to inadequate vision.

Participants were then seated in front of a 14-inch color monitor at a standard eye distance of 24 inches. Instructions were provided, via the program, corresponding to the experimenter's oral instructions. If a participant deviated from the instructions in a manner that rendered the trial invalid, then that trial was discarded, and the number of trials were prorated at the end of the data collection. Practice trials were presented and performance on these trials was recorded. The computer screen displayed three circles. The three circles were centered on the display and placed horizontally, with two circles placed on either side of a central circle at a distance of five degrees in visual angle from the central circle. Each participant was administered eight conditions as described below.

The inhibition of return task. On each of 200 trials, participants were instructed to focus their attention on a small fixation dot located within the central circle.

Participants were then asked to depress a reaction time key (the spacebar) with the forefinger of the preferred hand as quickly as possible whenever a target (a large "+" sign) appeared in one of the non-central figures. Participants proceeded through a number of practice trials before the onset of the actual experimental trials, to clear up any misunderstood directions.

Trials: Experimental. The experimental trials served to operationalize inhibition of return. There were 40 trials in each of four experimental conditions. In each condition, the appearance of the target followed a cue (the brightening of the inside of the circle) in variable intervals of 100 milliseconds (ms), 200ms, 500ms, or 750ms.

Condition E1--Right-Center-Right (RCR): The first cue consisted of a brightening of the right peripheral figure that remained for 150ms. At 500ms after the onset of the first cue, the subject's attention was directed to the central figure through its brightening for 150ms. Following a variable interval, a target appeared within the right peripheral figure for 150ms. Participants were instructed to press the spacebar as quickly as possible following the onset of the target.

Condition E2--Right-Center-Left (RCL): This condition followed the same procedure as condition E1. However, the brightening cue first appeared in the right figure, then

attention was directed to the central figure, following which the target appeared in the left peripheral figure.

Condition E3--Left-Center-Left (LCL): Again, the same procedure was used as in the first two conditions, with the brightening cue first appearing in the left peripheral figure, followed by direction of attention to the central figure, and the target then appeared in the left peripheral figure.

Condition E4--Left-Center-Right (LCR): This condition consisted of the left figure being used as the brightening cue, followed by direction of attention to the center, with the target appearing in the right peripheral figure.

Trials: Control. In order to explore any cueing effects or differences between left or right visual field preference, 10 trials in each of the following control conditions were included. These trials examined whether a facilitation (shorter reaction time) or deficit (longer reaction time) could be explained by a preference of one visual field over the other. In order to examine visual field preferences, these control trials did not redirect the subject's attention to the central circle as in the experimental trials described above. In each of the conditions, the interval varied between the cueing (brightening) and the presentation of the target. These intervals were 100ms, 200ms, 500ms, and 750ms.

Condition C1--Right-Right (RR): The cue consisted of a brightening in the right peripheral figure for 150ms. After a 100ms, 200ms, 500ms, or 750ms interval, the target stimulus was presented in the right peripheral figure for 150ms.

Condition C2--Right-Left (RL): The cue consisted of a brightening in the right peripheral figure for 150ms. After a 100ms, 200ms, 500ms, or 750ms interval, the target were displayed in the left peripheral figure for 150ms.

Condition C3--Left-Left (LL): The cue consisted of a brightening in the left peripheral figure for 150ms. After a 100ms, 200ms, 500ms, or 750ms interval, the target was presented in the left peripheral figure for 150ms.

Condition C4--Left-Right (LR): The cue consisted of a brightening in the left figure for 150ms. After an interval of 100ms, 200ms, 500ms, or 750ms, the target stimulus appeared in the right peripheral figure for 150ms.

In summary, there were a total of 200 trials among four experimental conditions and four control conditions. Within each of the eight conditions there was an equal representation of trials at each of the four millisecond intervals (100, 200, 500, 750).

CHAPTER III

RESULTS

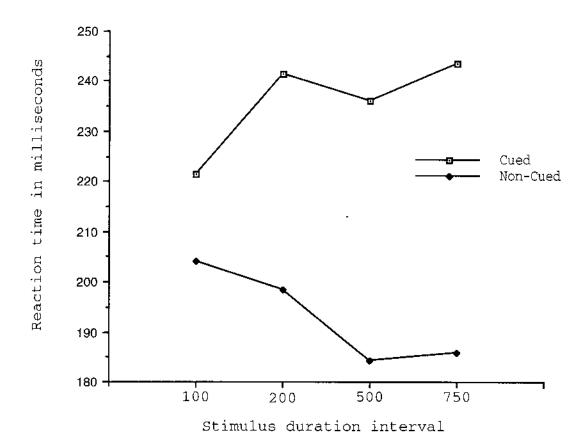
Control trials were initially examined to determine if any group differences were present regarding preferences for visual field or visual field movement. Groups did not differ in either visual field preference or visual field movement. The groups were found to be similar with respect to age, $\underline{F}(1,32) = .10$, $\underline{p} = .75$. Chi square analyses revealed no significant differences between groups with respect to gender, $X^2(1, \underline{N} = 43) = .003$, $\underline{p} = .95$; and race, $X^2(2, \underline{N} = 43) = 2.52$, $\underline{p} = .28$.

An initial overall 2 X 2 X 4 mixed MANOVA was performed with group (schizophrenic vs. normal) as a between subjects factor and both cueing (cued, non-cued) and interval (100ms,200ms,500ms,750ms) as within subject factors. The dependent variable was reaction time. A significant main effect was found for group, E(1,45) = 18.58, p < .001, with schizophrenia patients showing longer reaction times than normal comparisons in all conditions. Significant main effects were also found for cueing, E(1,45) = 64.32, p < .001; and for interval, E(3,135) = 6.84, p < .001. Both groups displayed an advantage of non-cued conditions over cued conditions; thus, some amount of inhibition of return was observed in each group. In addition to the main

effects, a significant two-way interaction was found between cueing and interval, $\underline{F}(3,135) = 3.62$, $\underline{p} = .015$, with both groups displaying faster reaction times to later, non-cued intervals. Most importantly, a significant three-way interaction between group, cueing, and interval was also found, F(3.135) = 3.17, p = .026. Figures 1 and 2 illustrate the interaction found. In order to test the primary hypthesis that schizophrenia patients would display advantages of the cued loaction relative to a third location, this three-way interaction was broken down. Regarding the interaction of cueing with group and interval, schizophrenia patients displayed an advantage of non-cued targets over cued targets for the 200ms, 500ms, and 750ms intervals, average $\underline{t} = 5.43$, average $\underline{p} < .001$; and not to the 100ms interval, $\underline{t} = 1.18$, $\underline{p} = .249$ (see Figure 1). Testing the second proposed hypothesis that normal comparisons would display faster reaction times to targets in the non-cued loaction relative to the cued location, normal comparisons displayed a significant advantage of noncued targets over cued targets at each of the four intervals, average $\underline{t} = 10.94$, average $\underline{p} < .001$ (see Figure 2).

The reaction times of normal comparisons in the cued conditions as a function of interval were found to be significantly smaller for the 100ms interval as compared to the 200ms interval, \underline{t} = -7.30, \underline{p} < .001. Reaction times to

Figure 1. The cuing x interval interaction for schizophrenia patients.



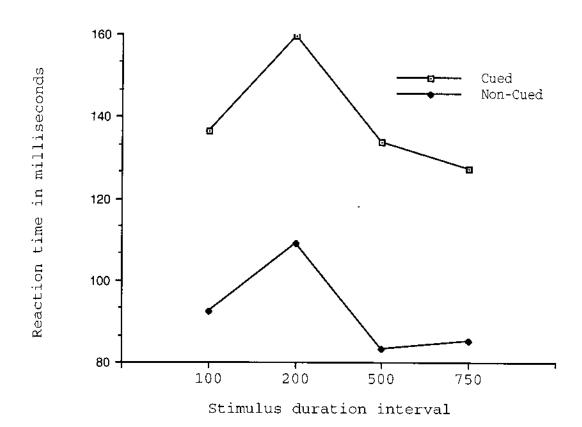
Note: For cued conditions:

```
100ms (\underline{M} = 221.48, \underline{SD} = 103.31)
200ms (\underline{M} = 241.49, \underline{SD} = 84.85)
500ms (\underline{M} = 236.02, \underline{SD} = 90.48)
750ms (\underline{M} = 243.55, \underline{SD} = 95.49)
```

For non-cued conditions:

```
100ms (\underline{M} = 203.99, \underline{SD} = 131.79)
200ms (\underline{M} = 198.36, \underline{SD} = 100.23)
500ms (\underline{M} = 184.35, \underline{SD} = 106.79)
750ms (\underline{M} = 185.75, \underline{SD} = 99.46)
```

<u>Figure 2</u>. The cuing x interval interaction for the normal comparison group.



Note: For cued conditions:

```
100ms (\underline{M} = 136.65, \underline{SD} = 38.57)
200ms (\underline{M} = 159.72, \underline{SD} = 37.19)
500ms (\underline{M} = 133.88, \underline{SD} = 36.25)
750ms (\underline{M} = 127.22, \underline{SD} = 35.24)
```

For non-cued conditions:

```
100ms (\underline{M} = 92.57, \underline{SD} = 35.02)
200ms (\underline{M} = 109.16, \underline{SD} = 33.51)
500ms (\underline{M} = 83.16, \underline{SD} = 34.45)
750ms (\underline{M} = 85.22, \underline{SD} = 34.78)
```

the 200ms interval were also found to be significantly larger than the 500ms interval, $\underline{t} = 6.63$, $\underline{p} < .001$; and the 750ms interval, $\underline{t} = 5.20$, $\underline{p} < .001$. Normal comparison reaction times to non-cued targets as a function of interval was identical to that found for cued targets, with the 200ms interval displaying significantly larger reaction times than either of the other three intervals, average t = 4.69, average p < .001 (see Figure 2). The reaction times of schizophrenia patients (Figure 1) to cued targets as a function of interval, revealed significantly smaller reaction times for the 100ms interval than both the 200ms interval, \underline{t} = -2.37, \underline{p} = .025; and the 750ms interval, \underline{t} = -2.12, p < .05. Reaction times of schizophrenia patients to non-cued targets, however, revealed only a significant advantage of the 500ms interval as compared to the 200ms interval, $\underline{t} = 2.37$, $\underline{p} = .025$ (see Figure 1).

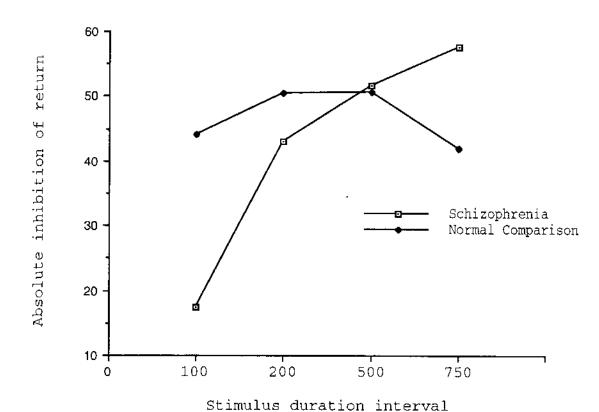
The third hypothesis, that schizophrenia patients would differ significantly from normals in the measured inhibition of return, was examined next. In order to directly operationalize inhibition of return, mean reaction times of non-cued trials were subtracted from mean reaction times of cued trials, with higher resulting numbers then representing greater inhibition of return. A MANOVA was performed using group as the between subjects factor and both visual field (target appearing in right visual field vs. left visual field) and interval as within subject factors.

Schizophrenia patients and normals did not differ significantly on overall levels of inhibition of return, $\underline{F}(1,45) = .15$, $\underline{p} = .70$. However, significant effects were found for interval, $\underline{F}(3,135) = 3.62$, $\underline{p} = .015$; group x interval, $\underline{F}(3,135) = 3.17$, $\underline{p} = .026$; and group x visual field, $\underline{F}(1,45) = 4.33$, $\underline{p} = .043$.

Regarding the group x interval interaction (see Figure 3), schizophrenia patients displayed increasing amounts of inhibition of return as the stimulus interval was lengthened. Normal comparisons, on the other hand, showed an increase in inhibition of return up to the 500ms, after which they displayed a significant decrease, $\underline{\mathbf{t}} = 2.48$, $\underline{\mathbf{p}} = .023$. The interaction between group and visual field (Figure 4) indicates that individuals in the schizophrenia group display significantly less inhibition of return to targets in the left visual field, $\underline{\mathbf{t}} = 2.34$, $\underline{\mathbf{p}} = .027$. Normal comparisons, on the other hand, do not display a significant advantage of either visual field, $\underline{\mathbf{t}} = -.70$, $\underline{\mathbf{p}} = .493$.

One additional finding regarding this inhibition of return data was a significant correlation between the measured inhibition of return and the overall reaction time, $\underline{r} = -.32$, $\underline{p} < .05$. Based on this correlational finding, it may be better to view the amount of inhibition of return as a ratio of cued and non-cued reaction times rather than an

Figure 3. The group x interval interaction of absolute inhibition of return.



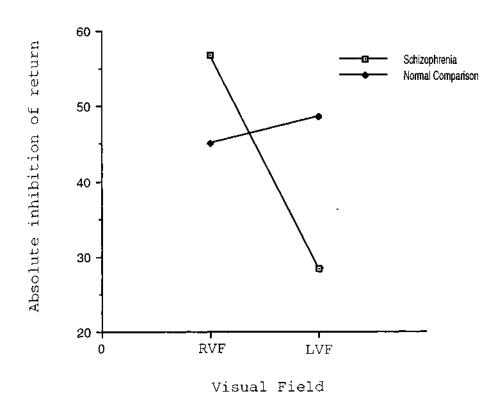
Note: For schizophrenia group:

```
100ms (\underline{M} = 17.49, \underline{SD} = 78.59)
200ms (\underline{M} = 43.13, \underline{SD} = 40.61)
500ms (\underline{M} = 51.67, \underline{SD} = 47.64)
750ms (\underline{M} = 57.79, \underline{SD} = 61.83)
```

For normal comparison group:

```
100ms (\underline{M} = 44.08, \underline{SD} = 19.35)
200ms (\underline{M} = 50.57, \underline{SD} = 24.86)
500ms (\underline{M} = 50.72, \underline{SD} = 19.08)
750ms (\underline{M} = 42.00, \underline{SD} = 13.68)
```

Figure 4. Group x visual field interaction for absolute inhibition of return.



Note: For schizophrenia group:

RVF ($\underline{M} = 56.72$, $\underline{SD} = 34.23$) LVF ($\underline{M} = 28.33$, $\underline{SD} = 73.01$)

For normal comparison group:

RVF ($\underline{M} = 44.99$, $\underline{SD} = 18.43$) LVF ($\underline{M} = 48.70$, $\underline{SD} = 17.43$)

absolute difference. Findings based on the analyses using absolute levels of inhibition of return revealed similar discrepancies between cued and non-cued reaction times for both groups. However, the schizophrenia patients were displaying a similar discrepancy at a much longer rate of responding and variability based on visual field. question remains as to whether both groups would show equivalent advantages of non-cued over cued targets if the schizophrenia patients did not take longer to react overall. Performing the same MANOVA mentioned above, an inhibition of return ratio was used (with a higher ratio representing more inhibition of return) as the dependent variable. The ratio was computed by subtracting the non-cued reaction time from the cued reaction time, and dividing the result by the sum of the cued and non-cued reaction times. A significant main effect for group was found, F(1,45) = 8.81, p = .005. (See Table 4 for a summary of group means and standard deviations for all main effects.) Schizophrenia patients thus show significantly smaller proportional amounts of inhibition of return compared to normals. Again, a significant main effect was found for interval, F(3,135) = 4.91, p = .003. Although a group x visual field interaction was not found to be significant $(\underline{F}(1,45) = 3.65, \underline{p} = .062)$, there was an evident trend. When collapsed across interval, a oneway ANOVA revealed that schizophrenia participants displayed a significantly smaller inhibition of return effect for the

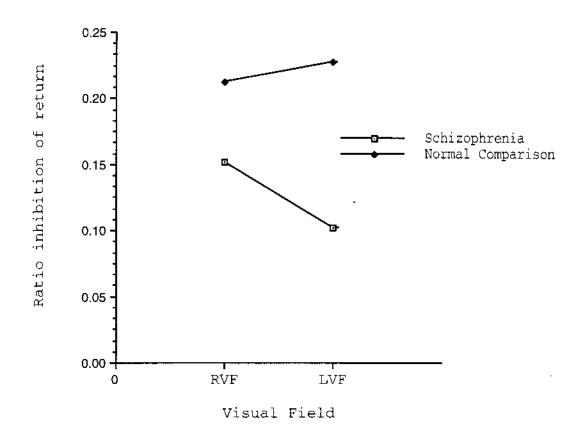
Table 4

Mean Reaction Times for Schizophrenic and Normal Comparison

Group Main Effects

Mean Reaction Time (Sd)			
Effect	Schizophrenia	Normal Comparison	
Initial MANOVA			
Overall Cued Non-cued 100ms interval 200ms interval 500ms interval 750ms interval	214.37 (95.64) 235.63 (89.39) 193.11 (106.83) 212.74 (111.70) 219.92 (90.61) 210.18 (96.06) 214.65 (92.46)	115.95 (32.10) 139.37 (33.98) 92.53 (31.62) 114.62 (35.05) 134.44 (33.14) 108.52 (34.05) 106.22 (34.33)	
Absolute Inhibition	of Return		
Overall 100ms interval 200ms interval 500ms interval 750ms interval	42.52 (47.09) 17.49 (78.59) 43.13 (40.61) 51.67 (47.64) 57.79 (61.83)	46.84 (13.71) 44.08 (19.35) 50.57 (24.86) 50.72 (19.08) 42.00 (13.68)	
Ratio Inhibition of	Return		
Overall 100ms interval 200ms interval 500ms interval 750ms interval Right Visual Field Left Visual Field	.13 (.12) .08 (.16) .12 (.11) .15 (.15) .16 (.14) .15 (.11) .10 (.15)	.22 (.08) .21 (.09) .20 (.11) .26 (.13) .22 (.10) .21 (.11) .23 (.09)	

<u>Figure 5</u>. The group x visual field trend for ratio inhibition of return.



Note: For schizophrenia group:

RVF (
$$\underline{M} = .152$$
, $\underline{SD} = .110$)
LVF ($\underline{M} = .103$, $\underline{SD} = .152$)

For normal comparison:

RVF (
$$\underline{M} = .212$$
, $\underline{SD} = .107$)
LVF ($\underline{M} = .228$, $\underline{SD} = .087$)

left visual field than the comparisons, F = 10.49, p = .002 (see Figure 5). Further, within the schizophrenia group, participants' levels of inhibition of return were found to be significantly smaller for the LVF, t = 2.14, p < .05; whereas the normal comparisons did not show this within group difference, t = -.68, t = .51 Regarding the interval effect, both groups generally display more proportional inhibition of return in the later intervals than in the early intervals. The schizophrenia group displayed significantly higher amounts of inhibition of return at the 500ms interval and the 750ms interval than at the 100ms interval, mean t = -2.87, mean t = .004. The normal comparisons display significantly higher proportions of inhibition of return at the 500ms interval when compared to the 200ms interval, t = -2.13, t = .048.

Subject status variables were explored via correlations within the schizophrenia group. Significant relations were not found between inhibition of return and subclassification (paranoid vs. nonparanoid), family history of mental illness, course of illness (acute vs. chronic). A significant negative correlation was found, however, between total score on the SADS-C and the inhibition of return (ratio) at the 100ms interval to targets in the right visual field only, $\underline{r} = .44$, $\underline{p} < .05$. In addition, total score on the SAPS was correlated with the amount of absolute

inhibition of return at the 100ms interval to targets in the right visual field only, \underline{r} = .52, \underline{p} < .01.

CHAPTER IV

DISCUSSION

The main concern of this study was whether schizophrenic patients would display the inhibition of return effect that has been reported in studies with nonschizophrenic participants (Posner & Cohen, 1984; Posner, Cohen, Choate, & Vaughn, 1985). Schizophrenia inpatients were significantly less inhibited to return attention to stimuli that were initially cued as compared to normals. This finding confirms the primary hypothesis set forth in this study. The results of the normal comparison participants appear to be analogous to those from previous studies (Abrams & Dobkin, 1994; Huey & Wexler, 1994; Posner, Rafal, Choate, & Vaughn, 1985; and Tipper, Driver, & Weaver, 1991;). Normal comparison participants respond more quickly to targets when they appear at a non-cued location than they do to targets occurring at cued locations. Schizophrenia patients, on the other hand, show a relative advantage of cued targets over non-cued targets when compared to the normal comparisons. The current findings appear to be consistent with a recent study by Huey and Wexler (1994; appearing since proposal of the present study) examining inhibition of return in schizophrenics. The Huey and Wexler (1994) study used very few participants (\underline{n} = 11) and the

procedure differed substantially from that used in the present study. However, the authors similarly reported a "blunted inhibition of return" effect for schizophrenia participants.

Though not included in the proposed hypotheses, differences in inhibition of return with respect to visual field were discovered. Although displaying less inhibition of return overall, individuals with schizophrenia showed the most significant differences to targets in the left visual field (LVF). Compared to normals, schizophrenia patients were significantly less inhibited to targets in the LVF. Schizophrenia has often been considered a disorder involving left-hemispheric impairment (Karny & Nachson, 1995; Levin, Yurgelun-Todd, & Craft, 1989). On the other hand, Tucker and Williamson (1984) have suggested that portions of the right hemisphere are largely responsible for the integration of perceptual input or preattentional activity. To the extent that LVF tasks are tapping right hemisphere activity in this study, the present findings would seem to provide support for theories of right-hemispheric impairment in schizophrenia. Such theories (e.g., Cromwell, 1987; Venables, 1984) have proposed that the right hemisphere has a relative advantage for preattentional screening, and this screening is impaired in individuals with schizophrenia. The study by Elkins, Cromwell, and Asarnow (1992), mentioned earlier, found results consistent with the present findings.

Although it was a study of span of apprehension and not inhibition of return, the authors discovered schizophrenia patients to be deficient, in comparison with normals, on tasks involving stimuli in the LVF. It appears that individuals with schizophrenia have maladaptive attentional screening processes which may be the result of right hemisphere damage/dysfunction.

The findings of this study provide support for a reconceptualized disattention hypothesis regarding schizophrenic behavior. Based on these results, individuals with schizophrenia are relatively unable to disengage from, in this case, a stimulus cue, in order to attend to a target occurring in a non-cued location. However, this lack of disengagement appears to be underlain by the relative absences of inhibition to returning attention to a stimulus even after disengagement is attempted. As mentioned earlier, empirical support for the disattention hypothesis proposed by Cromwell and Dokecki (1968) has been limited and indirect. Given the nature of the inhibition of return task, these results with schizophrenia would seem to offer more direct support to this reformulation of Cromwell and Dokecki's (1968) hypothesis.

Examining the pattern of overall responding (see Figure 3), the schizophrenic group showed a more extreme lack of inhibition to early intervals and an increasing inhibition at later intervals. Normal comparisons, on the other hand,

generally showed more inhibition to early trials with the inhibition of return effect significantly diminished at the 750ms interval. The results of the normal comparisons' performance were consistent with those of previous studies. One would expect the inhibition effect to continue to diminish if additional, longer intervals were included in the design. The schizophrenia patients, however, appeared to become more deficient with time, after showing an initial advantage of the cued stimuli at early intervals. schizophrenia participants displayed an initial deficit in inhibition of return at the 100ms interval, followed by a relative recovery at the 200ms and 500ms intervals. Unlike normals, however, individuals with schizophrenia then display increasing levels of inhibition of return at the 750ms interval, a finding not reported in the Huey and Wexler (1994) study. It appears as though there could be two separate factors that are active in this task for schizophrenics. The first of these factors results in the schizophrenia patients reacting with increased sensitivity to the brightening cue; the second factor leaves the subject over-inhibited by the attentional shift at later intervals. This finding is consistent with the recent elaboration of the disattention hypothesis put forth by Cromwell (1993).

As discussed earlier, Cromwell (1993) has proposed that individuals with schizophrenia display both a persistence of attention and an increased sensitivity to physical stimulus

input. According to Cromwell (1993), the increased sensitivity (Factor S) and what he labeled the overinhibitory factor (Factor R) exist separately and are under their own genetic and environmental comparison. Factor S is proposed to be associated with the perceived novelty of stimulus input. Such overfacilitation may result in quick responses to intense stimuli or extensive scanning, both of which could be considered adaptive. On the other hand, such a response may be maladaptive in the sense that it may result in the schizophrenia patient being unable to release attention from the novel stimulus to respond to an upcoming event. Factor R is hypothesized to result in an exaggerated tendency to "over-conceptualize" the meaning and action sequence implication of a stimulus input. This tendency is characterized by a progressive slowing of reaction time or an increased inhibition. Factor S, then, appears to be reflected by the relatively smaller absolute amounts of inhibition of return for those in the schizophrenia group to the 100ms interval (see Figure 3), while Factor R can be seen in the same figure as the increase in absolute inhibition of return at the 750ms interval.

The inhibition of return task was proposed in this study as an appropriate method of assessing the existence of two such separate factors. The current findings provide tentative support for the existence of over-excitatory and over-inhibitory factors among schizophrenia patients.

Regarding the differences in inhibition of return to stimuli in the LVF versus RVF, it may be that Factor S and Factor R exhibit their tendencies differentially to the different visual fields. Based on these differences, it may be that Factor S is more strongly associated with right hemisphere activity, while Factor R is related to left hemisphere activity. Alternatively, given that Cromwell (1993) suggests that it is the R X S interaction that constitutes what we recognize as schizophrenia, it could be that the two factors are not localized in separate hemispheres; rather, the R X S interaction may manifest differently to LVF and RVF tasks.

Because Factor S is proposed to be associated with positive symptoms and, therefore, more amenable to neuroleptic treatment, a relative lessening of Factor S deficits in the RVF may reflect the effectiveness of drug treatment for left hemisphere positive symptoms. Attempts were made to examine the possible effects that neuroleptics had on the measured inhibition of return. Significant correlations were not found between the level of inhibition of return and drug dosage suggesting that the findings are not accounted for by drug treatment. However, it is difficult to accurately assess drug effects when comparing dosages of drugs with varying actions. Approximately seven different anti-psychotic drugs were being used for treatment within the schizophrenic group. Although dosages of these

drugs were converted into chlorpromazine equivalents, this procedure is not definitive. For example, drugs with actions different from the standard neuroleptics in terms of being dopamine receptor blockers (e.g., Risperdol, which also has action on 5-HT₂ receptors), are very difficult to directly compare.

It is difficult to explain the correlations found with respect to right visual field inhibition of return indices and both overall and positive symptomatology. Attempts to explain such an unexpected finding would involve a significant degree of speculation; they might, in fact, be simple artifacts. This is especially the case, given that inhibition of return group differences were the weakest in the RVF 100ms condition—the only condition in which these correlations are significant. Additional research into visual field differences on inhibition of return tasks with schizophrenia patients is necessary.

Problems with the Present Study

Efforts to obtain an appropriate psychiatric comparison group were not successful. The diagnostic make-up of the inpatient facility was such that a vast majority of patients either had prior histories of psychosis or were currently displaying some level of psychotic symptomatology. For this reason, it was not possible to test hypotheses concerning a psychiatric comparison. The lack of a psychiatric comparison group limits the present study in that the

findings regarding schizophrenia patients may be due to the presence of pathology and not specific to schizophrenia itself. In addition, a vast majority of the patients with schizophrenia were reported to have a history of substance abuse. Currently, no studies exist regarding the effects of substance use on inhibition of return tasks; therefore, the current findings could partially be accounted for by this variable.

A majority of both groups studied, consisted of male participants. The small number of female participants did not allow for valid comparisons between genders.

Directions for Future Research

Ideal future investigations of this type would examine the presence of inhibition of return in three to four groups. In addition to a group of schizophrenia patients and normal comparisons, future investigations would include a second psychiatric comparison group with whom to compare the findings of the schizophrenia group. This psychiatric comparison group would consist of mood-disordered patients who have no prior history of psychosis. A potential fourth group might include a group of unmedicated schizophrenia patients, in order to assess for effects of neuroleptic medication. Alternatively, or in addition to, a group of non-symptomatic, first-degree relatives of schizophrenia patients might be included. Each group would ideally contain 30-50 participants so that within-group comparisons

could be made. The procedure of future studies would vary somewhat from the present study. The actual inhibition of return task, as presented by the computer, could remain as it is in the current study. However, a chin rest might potentially be utilized to keep the participants' eyes level with the visual stimuli and to assist the participants in remaining focused on the center fixation point. Finally, in addition to the four millisecond intervals used in the current study, intervals of longer duration (e.g., 1000ms, 1250ms) would be included to examine the course of the inhibition of return effect; especially for the schizophrenia patients, given that in the current study they unexpectedly displayed increasing levels of inhibition of return as a function of time.

Several important questions are raised by the current findings. Are schizophrenia patients' deficits in inhibition of return specific to the group studied here, or can they be shown in other schizophrenia groups? Is this deficit specific to schizophrenia? Does an impairment in inhibition of return occur only in medicated schizophrenics, or would unmedicated schizophrenia patients show similar deficits? Can deficits in inhibition of return be shown to be present in other schizophrenia boundary groups (e.g., first-degree relatives, schizotypal patients)? Further research on such issues should be conducted.

Summary and Conclusions

The results of the present study have contributed to the understanding of information processing in schizophrenia. It appears as if the relative lack of inhibition of returning attention in schizophrenia patients may be at least in part responsible for much of the behavior exhibited by those with the disorder. Further, the present results suggest a differential presence of this lack of inhibition to each cerebral hemisphere. Such a finding, upon necessary replication, may help in understanding the physiological aspects of schizophrenia and ultimately provide additional insight into the etiology of the disorder.

Despite contributing to a better understanding of the processing involved in schizophrenia, the current study is only the second study to examine the inhibition of return phenomenon in a schizophrenia sample. Similar research, not only concerning inhibition of return itself, but also in conjunction with other information processing variables, is necessary.

APPENDIX

INFORMED CONSENT

Informed Consent

You are invited to participate in a study in which the response speed of adults will be examined. You are being asked to participate in this study because you fall into one of the groups which will be studied. This study is being conducted by myself, Jeffrey D. Hinds, under the supervision of Kenneth W. Sewell, Ph.D., and in association with the University of North Texas. If you choose to participate in this study it will be necessary for you to spend about 45 minutes of your time with the researcher. First, you will be interviewed briefly. Following the brief interview, you will be asked to sit in front of a computer screen and release one of the keys when you see a particular target appear on the screen. You will be asked to react as rapidly as possible to this target.

Information gathered from your performance as well as any information obtained from your hospital records will be kept in strict confidence at all times. Only the investigator and supervising professor will have access to the information. Following the collection of the data, your name and other identifying information will be removed from all records. In this way, information regarding your participation will be kept confidential.

There is only minimal to no risk to you in participating in this study. Your participation in this research is completely voluntary. Your signature below indicates that you have decided to participate in this study

and that you have read and understood the information that is written here. If you decide to participate, you are free to withdraw from the study at any time. A copy of this consent form will be provided for you. If you have any questions regarding this study, you may contact Jeffrey D. Hinds at (817) 380-4032.

I HAVE READ AND UNDERSTAND THE ABOVE INFORMATION AND AGREE TO PARTICIPATE IN THIS RESEARCH. I UNDERSTAND THAT I MAY WITHDRAW FROM THIS STUDY AT ANY TIME, AND IF I CHOOSE TO DO SO, NEITHER MY RELATIONS WITH THE HOSPITAL STAFF NOR MY TREATMENT WILL BE HARMED AS A CONSEQUENCE OF MY WITHDRAWAL.

the state of the s	Date	
Your Signature		
	Date	
Investigator's Signature		
	Date	
Guardian or legal representative		
(if applicable)		

REFERENCES

Abel, L.A., Friedman, L., Jesberger, J., Malki, A., & Meltzer, H.Y. (1991). Quantitative assessment of smooth pusuit gain and catch-up saccades in schizophrenia and affective disorders. <u>Biological Psychiatry</u>, 29, 1063-1072.

Abrams, R.A., & Dobkin, R.S. (1994). Inhibition of return: Effects of attentional cuing on eye movement latencies. <u>Journal of Experimental Psychology-Human</u>

<u>Perception and Performance</u>, 20, 467-477.

American Psychiatric Association (1987). <u>Diagnostic and statistical manual of mental disorders</u> (3rd ed.-revised). Washington, DC: Author.

Andreasen, N.C. (1984a). The scale for the assessment of negative symptoms (SANS). Iowa City, IA: University of Iowa.

Andreasen, N.C. (1984b). The scale for the assessment of positive symptoms (SAPS). Iowa City, IA: University of Iowa.

Andreasen, N.C., & Flaum, M. (1991). Schizophrenia: The characteristic symptoms. <u>Schizophrenia Bulletin</u>, 17, 27-49.

Asarnow, R.F., Granholm, E., & Sherman, T. (1991). Span of apprehension in schizophrenia. In H.A. Nasrallah, J. Zubin, J., S. Steinhauer, and J.H. Gruaelier (Eds.), Handbook of schizophrenia: Experimental psychopathology,

neuropsychology, and psychophysiology. Amsterdam, The Netherlands: Elsevier.

Bellissimo, A., & Steffy, R.A. (1971). Redundancy-associated deficit in schizophrenic reaction time performance.

Bleuler, E. (1911). Dementia praecox or the group of schizophrenias. Translated by J. Zinkin. New York, NY: International Universities Press (1950).

Blackwood, D.H., St. Clair, D.M., Muir, W.J., & Duffy, J.C. (1991). Auditory P300 eye tracking dysfunction in schizophrenic pedigrees. Archives of General Psychiatry, 48, 899-909.

Braff, D.L. (1985). Attention, habituation, and information processing psychiatric disorders. In R. Michaels, J.O. Cavenar, H.K. Brodie, A.M. Cooper, S.B. Guze, L.L. Judd, G.L. Klerman, & A.J. Solnit (Eds.), <u>Psychiatry.</u>
Vol. 3. Philadelphia, PA: J.B. Lippincott.

Braff, D.L. (1993). Information processing and attention dysfunctions in schizophrenia. <u>Schizophrenia</u> Bulletin, 19, 233-259.

Braff, D.L., Grillon, C., & Geyer, M.A. (1992). Gating and habituation of the startle reflex in schizophrenic patients. Archives of General Psychiatry, 49, 206-215.

Braff, D.L., Stone, C., Callaway, E., Geyer, M.A., Glick, I.D., & Bali, L. (1978). Prestimulus effects on human

startle reflex in normals and schizophrenics.

Psychophysiology, 14, 339-343.

Chapman, L.J. & Chapman, J.P. (1989). Strategies for resolving the heterogeneity of schizophrenics and their relatives using cognitive measures. <u>Journal of Abnormal Psychology</u>, 98, 357-366.

Chaturvedi, S.K. (1986). Assessing negative symptoms in depression. <u>Journal of Clinical Psychology</u>, 13, 127-131.

Clementz, B.A., Grove, W.M., Iacono, W.G., & Sweeney, J.A. (1992). Smooth pursuit eye movement dysfunction and liability for schizophrenia: Implications for genetic modeling. Journal of Abnormal Psychology, 101, 117-129.

Cohen, B.D., Naachmani, G., & Rosenberg, S. (1974).

Referent communication disturbances in acute

schizophrenia. <u>Journal of Abnormal Psychology</u>, 83, 1-13.

Cromwell, R.L. (1987). An argument concerning schizophrenia: The left hemisphere drains the swamp. In A. Glass (Ed.) <u>Individual differences in hemispheric</u> specialization. London: Spectrum Press.

Cromwell, R.L. (1993). A summary view of schizophrenia.

In R.L. Cromwell and C.R. Snyder (Eds.), Schizophrenia:

Origins, processes, and treatments. New York: Guilford.

Cromwell, R.L., & Dokecki, P. R. (1968). Schizophrenic language: A disattention interpretation. In S. Rosenberg and J. H. Koplin (Eds.), <u>Developments in applied psycholinguistic research</u>. New York: Macmillan.

Dawson, M., & Nuechterlein, K.H. (1984).

Psychophysiological dysfunctions in the developmental course of schizophrenic disorders. <u>Schizophrenia Bulletin, 10</u>, 204-232.

de Marchi, N. (1991). The genetics of schizophrenia.

<u>Developmental Medicine and Child Neurology</u>, 33, 452-458.

Edelman, G. (1987). <u>Neural Darwinism</u>. New York, NY: Basic Books.

Elkins, I.J. & Cromwell, R.L. (1992). Span of apprehension in schizophrenic patients as a function of distractor masking and laterality. <u>Journal of Abnormal Psychology</u>, 101, 53-60.

Endicott, V., & Spitzer, L. (1978). A diagnostic interview: the Schedule for Affective Disorders and Schizophrenia. Archives of General Psychiatry, 35, 837-848.

Freedman, R., Adler, L.E., Waldo, M.C., Pachtman, E., & Franks, R.D. (1983). Neurophysiological evidence fo a deficit in inhibitory pathways in schizophrenia: Comparison of medicated and drug- free patients. Biological Psychiatry, 18, 537-551.

Heilbrun, A.B. (in press). Strategies of disattention in schizophrenics and auditory hallucinations. <u>Journal of Abnormal Psychology</u>.

Hoffman, R.E., & Dobscha, S.K. (1989). Cortical pruning and the development of schizophrenia: A computer model.

Schizophrenia Bulletin, 15, 477-490.

Holzman, P.S. (1987). Recent studies of psychophysiology in schizophrenia. <u>Schizophrenia Bulletin</u>, 13, 49-76.

Holzman, P.S., Kringlen, E., Mathysse, S., Flanagan, S. D., Lipton, R.B., Cramer, S., Levin, S., Lange, K., & Levy, D.L. (1988). A single dominant gene can account for eye tracking dysfunctions and schizophrenia in offspring of discordant twins. Archives of General Psychiatry, 45, 641-647.

Holzman, P.S., Proctor, L.R., & Hughes, D.W. (1973).

Eye tracking patterns in schizophrenia. Science, 181, 179
180.

Huston, P.E., Shakow, D., & Riggs, L.A. (1937). Studies of motor function in schizophrenia. II. Reaction time.

<u>Journal of General Psychology</u>, 16, 39-82.

Iacano, W.G., & Clementz, B.A. (1993). A strategy for elucidating genetic influences on complex psychopathological syndromes (with special reference to ocular motor functioning and schizophrenia). In: L.J. Chapman, J.P. Chapman, & D.C. Fowles (Eds.), <u>Progress in experimental psychopathology research</u>. New York, NY: Springer.

Karny, N., & Nachson, I. (1995). Abnormal lateralization in schizophrenia: Empirical evidence for an integrated model. <u>European Psychiatry</u>, 10, 75-84.

Kietzman, M.L., Spring, B., & Zubin, J. (1985).
Perception, cognition and attention. In: H.I. Kaplan, A.M.

Freedman, & B.J. Sadock (Eds.), <u>Comprehensive textbook of psychiatry</u>. Baltimore, MD: William & Wilkins.

Kraeplin, E. (1921). <u>Clinical psychiatry: A textbook</u>

<u>for students and physicians.</u> Translated by A.R. Desfendorf.

New York, NY: Macmillan.

Levin, S., Yurgelun-Todd, D., & Craft, S. (1989).

Contributions of clinical neuropsychology to the study of schizophrenia. Journal of Abnormal Psychology, 98, 341-356.

McDonald-Scott, P., & Endicott, J. (1984). Informed versus blind: The reliability of cross-sectional ratings of psychopathology. <u>Psychiatry Research</u>, 12, 207-217.

Merritt, R.D., & Balogh, D.W. (1990). Backward masking as a function of spatial frequency: A comparison of MMOI-identified schizotypics and control subjects. <u>Journal of Nervous and Mental Disease</u>, 88, 186-193.

Muir, W.J., St. Clair, D.M., Blackwood, D.H., Roxburgh, H.M., & Marshall, I. (1992). Eye-tracking dysfunction in the affective psychoses and schizophrenia. <u>Psychological</u>

Medicine, 22, 573-580.

Murray, R.M., Reveley, A.M., & McGuffin, P. (1986).

Genetic vulnerability to schizophrenia. <u>Psychiatric Clinics</u>

of North <u>America</u>, 9, 3-16.

Posner, M.I., & Cohen, Y. (1984). Components of visual orienting. In H. Bouma & D. Bowhuis (Eds.), <u>Attention and performance X</u>. Hillsdale, NJ: Erlbaum, 531-556.

Posner, M.I., Cohen, Y., Choate, L., Hockey, R., & Maylor, E. (1984). Sustained concentration: Passive filtering or active orienting? In S. Kornblum and J. Requin (Eds.). Preparatory states and processes. Hillsdale, N.J.: Lawrence Erlbaum Associates Inc.

Posner, M.I., Cohen, Y., Choate, L.S. & Vaughn, J. (1985). Inhibition of Return: Neural basis and function. Cognitive Neuropsychology, 2, 211-228.

Posner, M.I., Rafal, R.D., Choate, L., & Vaughn, J. (1985). Inhibition of return: Neural basis and function. Cognitive Neuropsychology, 2, 211-228.

Rafal, R.D., Calabresi, P.A., Brennan, C.W., & Sciolto, T.K. (1989). Saccade preparation inhibits reorienting to recently attended locations. <u>Journal of Experimental</u>

Psychology-Human Perception and Performance, 15, 673-685.

Saccuzzo, D.P., Hirt, M., & Spencer, T. (1974).

Backward masking as a measure of attention in schizophrenia.

Journal of Abnormal Psychology, 83, 512-522.

Salzinger, K. (1971). The immediacy hypothesis and schizophrenia. In H.M. Yaker, H. Osmand, and F. Cheek (Eds.), Man's place in time. New York: Doubleday Doran.

Salzinger, K. (1983). The immediacy hypothesis in a theory of schizophrenia. Nebraska Symposium on Motivation, 31, 231-282.

Shakow, D., (1962). Segmental Set: A theory of the formal psychological deficit in schizophrenia. Archives of General Psychiatry, 6, 1-17.

Shakow, D. (1963). Psychological deficit in schizophrenia. Behavioral Science, 8, 275-305.

Servan-Schreiber, D., & Cohen, J.D., (1992). Neural Networks. <u>Psychiatric Annals</u>, 22, 112-148.

Siever, L.J. (1991). The biology of the boundaries of schizophrenia. In: C.A. Tamminga, & S.C. Schulz (Eds.),

Advances in neuropsychiatry and psychopharmacology:

Schizophrenia research. New York, NY: Raven Press.

Siever, L.J., Keefe, R., Bernstein, D.P., Coccaro, E.F., Klar, H.M., Zemishlany, Z., Peterson, A.E., Davidson, M., Mahon, Horvath, T., & Mohs, R. (1990). Eye tracking impairment in clinically identified patients with schizotypal personality disorders. American Journal of Psychiatry, 147, 740-745.

Spitzer, R.L. & Endicott, J. (1978). Schedule for

Affective Disorders and Schizophrenia-Change Version, 3rd

ed. New York: New York State Psychiatric Institute,

Biometrics Research.

Sutton, S., & Zubin, J. (1965). Effects of sequence on reaction time in schizophrenia. In A.T. Welford & J.E. Birren (Eds.), <u>Behavior</u>, aging and the <u>nervous system</u>. Springfield, Ill.: Charles C. Thomas.

Tipper, S.P., Driver, J., & Weaver, B. (1991). Object-centered inhibition of return of visual attention. <u>Ouarterly Journal of Experimental Psychology-Human Experimental</u>
Psychology, 43, 289-298.

Tizard, J., & Venables, P.H. (1956). Reaction time responses by schizophrenics, mental defectives, and normal adults. American Journal of Psychiatry, 112, 803-807.

Torrey, E.F. (1987). Prevalence studies in schizophrenia. <u>British Journal of Psychiatry</u>, 150, 598-608.

Tucker, D.M., & Williamson, P.A. (1984). Asymmetric neural control systems in human self-regulation.

Psychological Review, 91, 185-215.

Venables, P.H. (1984). Cerebral mechanisms, autonomic responsiveness, and attention in schizophrenia. In W.D. Spaulding & J.K. Cole (Eds.) <u>Nebraska Symposium on</u> Motivation. Lincoln: University of Nebraska Press.

Wynne, L.C., Cromwell, R.L., & Mathysse, S. (Eds.)

(1978). The nature of schizophrenia. New York: John Wiley and Sons.

Zubin, J. (1975). Problem of attention in schizophrenia. In M.L. Kietzman, S. Sutton, & J. Zubin (Eds.), Experimental approaches to psychopathology. New York, NY: Academic Press.