Relations of Symptoms to Cognitive Deficits in Schizophrenia

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Abstract

Schizophrenia is characterized by a variety of cognitive dysfunctions. Information-processing dysfunctions differ between clinical subtypes such that nonparanoid schizophrenia patients attend less than paranoid schizophrenia patients to connotative or contextual aspects of stimuli. The positive and negative symptom dimensions are also associated with distinct cognitive deficits. In general, positive symptoms are related to auditory-processing deficits and negative symptoms to visual/motor dysfunctions. The interaction of frontal and septohippocampal brain systems, and failures of information-processing automaticity and self-monitoring, have been proposed as the bases of positive symptoms. Negative symptoms are thought to arise from abnormalities in the complex interactions of frontal and striatal systems. Recent theoretical analyses have recommended a focus on the cognitive and neuropsychological analysis of specific symptoms (e.g., hallucinations and delusions) instead of on the more heterogeneous symptom clusters or dimensions. Studies of specific symptoms indicate that patients with hallucinations have deficits in discriminating the source of information. Delusions have been related to abnormal inference processes as well as abnormal perceptual experiences. Studies should now examine the links between information-processing abnormalities and symptoms over time, as the latter change, within the framework of explicit, disconfirmable theoretical models.

People with schizophrenia generally perform inefficiently on information-processing tasks. The cognitive dysfunction of schizophrenia, or "psychological deficit" (Hunt and Cofer 1944), has been the subject of extensive empirical and theoretical analysis over the last 50 years (Hunt and Cofer 1944; Buss and Lang 1965; Lang and Buss 1965; Chapman and Chapman 1973; Cromwell 1975). Advances in cognitive psychology, neuropsychology, and neuroscience now allow us to describe mechanisms of the cognitive dysfunctions typically observed in schizophrenia, as well as possible neurobiological substrates (Steinhauer et al. 1991).

Schizophrenia is an experiential and behavioral disorder that requires analysis at biological, psychological, and social levels. The study of cognitive processes has been an important avenue for linking the biological and social aspects of the illness (Hemsley 1991). Three principal approaches have been used to join the phenomenology and cognitive psychology of schizophrenia. The first was the comparison of Kraepelinian subtypes to determine whether there were differences in cognitive deficits. In the second, positive and negative symptom dimensions were studied, following on the suggestion by Strauss et al. (1974) that these independent dimensions might reflect disturbances in different underlying processes or mechanisms. The comparison of

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cognitive deficits in patients classified as Type I ("positive schizophrenia") and Type II ("negative schizophrenia"; Crow 1985) is a variant of this approach. Most recently, interest emerged in the cognitive psychology of specific symptoms themselves. This article reviews these approaches to the study of symptom-cognition relations in schizophrenia and describes principal findings, methodological and conceptual issues, and promising trends.

Psychological Deficits in Schizophrenia Subtypes

The psychological deficits of schizophrenia are pervasive; patients perform less well than other groups on most cognitive or attentional tasks (Hemsley 1991). And, as was evident in the first review of this field (Hunt and Cofer 1944), these deficits are also quite variable in groups of patients. Indeed, the variability of performance in a group of patients with schizophrenia may exceed the average difference between them and controls (Cromwell 1975).

The great variability of schizophrenia patients' performance on psychological tasks was demonstrated in Shakow and colleagues' early work on the mechanisms of psychological deficit in schizophrenia (Shakow and Huston 1936; Huston et al. 1937; Rodnick and Shakow 1940; Shakow 1962). In the course of their research, Shakow et al. discovered substantial correlations (r = 0.4–0.6) between the patients' performance and their motivation, cooperativeness, and interest.

While reduced motivation contributes to the poor performance of schizophrenia patients on psychological tasks, it is by no means the only cause. Some recent cognitive studies have demonstrated that the performance of schizophrenia patients on cognitive tasks can be *better* than that of controls. For example, schizophrenia subjects perform better than controls on visual estimation of numbers because they have a deficit in their ability to group them (Schwartz-Place and Gilmore 1980). Patients with schizophrenia also may learn an association more rapidly than controls because of dysfunctions in inhibitory or selective attention processes, as in the latent inhibition studies described below.

Reduced motivation and lack of interest are certainly important contributors to the variability of performance seen within groups of schizophrenia subjects. Other factors more specific to schizophrenia have also been shown to contribute to variability in schizophrenic psychological deficit. The three that have been investigated most extensively are symptom pattern, stage of illness (chronicity), and premorbid adjustment (Cromwell 1975).

Like cognitive deficits, symptom patterns are variable in schizophrenia, as the disorder is not defined by a conjunctive symptom set. In both Kraepelinian and Bleulerian formulations, the common feature(s) among patients was something other than symptoms. For Kraepelin (1919/1971), the common features were onset (early) and course (progressive deterioration), while for Bleuler (1911/1950) the commonality was in underlying mechanism (breaking of associative threads; Neale 1987). Some symptomatic homogeneity was achieved through classifying patients into clinical subtypes (e.g., paranoid, catatonic, hebephrenic). The investigation of relations between these subtypes and others (chronic vs. acute, process vs. reactive) and both pattern and severity of psychological deficit was the first approach taken to relate symptoms to cognitive disorder in schizophrenia.

In seminal papers, Silverman (1964) and Venables (1964) reviewed the already large literature on relations of psychological deficit to Kraepelinian subtype (particularly paranoid vs. nonparanoid), premorbid adjustment, and chronicity (Schooler and Feldman 1964). Both Silverman (1964) and Venables (1964) noted that subclassifying patients conjointly on the three dimensions of subtype, premorbid adjustment, and chronicity reduced the variability of performance in schizophrenia subgroups. Furthermore, this tripartite subclassification identified distinct patterns of cognitive and psychophysiologic deficits. For instance, nonparanoid, poor premorbid, and chronic patients were found to perform more poorly than controls. Paranoid patients, especially those who had made adequate psychosocial adjustments before the onset of the illness and who had been ill for only a short time, often did not differ from normals and sometimes performed better than control groups on cognitive/attentional tasks (Cromwell 1975).

The study of relationships between cognitive dysfunctions and clinical characteristics appeared to be an avenue for rationalizing the cognitive variability in schizophrenia. It permitted the identification of subgroups whose information-processing characteristics were different from each other as well as from controls. Three related principles emerged from this research to organize our under-

standing of cognition in schizophrenia: (1) fundamentally different patterns of information processing or cognitive style are associated with paranoid and nonparanoid schizophrenia; (2) the pattern of cognitive characteristics observed in schizophrenia is tempered by the premorbid history of the patients; and (3) these cognitive styles appear to change over the course of the illness, possibly as an adaptive process (Venables 1964; Silverman 1972).

The study of subgroup differences was the basis of a number of hypotheses about the mechanisms of schizophrenia. Silverman's (1964, 1967, 1972) theory of relations between cognition and clinical features of schizophrenia focused on three aspects of attention: the modulation of intensity of input ("stimulus intensity control"), breadth of attention ("scanning control"), and relative focus on sensory rather than conceptual attributes of stimuli ("sensory input processing/ideational gating"). Silverman (1967) saw cognitive characteristics changing over the course of the disorder, as did Venables (1964), who suggested that chronic patients developed a cognitive style of narrowing attention in response to states of hyperarousal. These theories were based on cross-sectional data rather than longitudinal studies of patients, and there was little direct support for their hypotheses of cognitive changes over the course of schizophrenia (Strauss 1973).

Silverman (1967) also suggested that nonparanoid and poor premorbid schizophrenia patients attended less to connotative or contextual aspects of stimuli and more to sensory/perceptual properties than normal subjects. This idea was elaborated by Magaro (1980) in a comprehensive theory differentiating the cognitive style of paranoid from other schizophrenia subjects. Magaro (1980) proposed that the paranoid schizophrenia patient is biased toward the use of conceptual rather than perceptual processing strategies; that is, his or her information processing is more governed by schemata and expectations than by perceptual analysis and inference.

Interest in subtype, premorbid, and chronicity constructs waned in the late 1970s, when the schizophrenia concept was being reformulated along more Kraepelinian lines. The narrower construct of schizophrenia as specified by Feighner et al. (1972), Research Diagnostic Criteria (RDC; Spitzer et al. 1978), and *DSM–III* criteria (American Psychiatric Association 1980), which include chronicity (poor outcome/duration) as part of the diagnostic criteria, led to reduction in the variability of premorbid adjustment and chronicity (Westermeyer and Harrow 1984). In the 1980s, symptom patterns of schizophrenia and their relation to neurocognitive dysfunctions came to be studied in terms of the positive and negative symptom dimensions.

Psychological Deficit in Relation to Positive and Negative Symptoms

Crow (1985) proposed two distinct forms of schizophrenia. Type I was characterized by positive or productive symptoms caused by reversible neurotransmitter abnormalities, while Type II was marked by the presence of cognitive deficits, negative symptoms, and structural brain abnormalities. Crow's hypothesis, the development of frameworks (e.g., Strauss et al. 1974), and methods for the reliable assessment of these symptoms (e.g., Schedule for Assessment of Negative Symptoms [SANS; Andreasen 1984*a*] and Schedule for Assessment of Positive Symptoms, [SAPS; Andreasen 1984*b*]) stimulated research on the positive and negative symptom typology (Walker and Lewine 1988).

Crow (1985) hypothesized a relation between cognitive dysfunction and negative symptoms, seeing both as mediated by structural brain impairment. The association between cognitive dysfunction and negative symptoms was confirmed in a number of studies that used clinical assessments of mental status to assess cognition. However, when more detailed assessments of cognitive processes were made, the relationships between positive and negative symptoms and cognition were equivocal (Walker and Lewine 1988). The measurement and conceptualization of positive and negative symptoms also proved more problematic than anticipated (Andreasen and Grove 1986; Carpenter et al. 1991).

Negative symptoms form a more coherent construct than do positive symptoms (Andreasen and Grove 1986). The internal consistency reliability (α) for the SANS (0.85, Andreasen and Grove 1986) is substantially higher than for the SAPS (0.48, Andreasen and Grove 1986; see also Andreasen and Olsen 1982). Internal consistency reliability (α) is evidence of the construct validity of a dimension and has important consequences for empirical research. Reliability sets an upper bound on the correlation of a measure with other variables and so affects what *can* be found in a study. Scale reliability coefficients, or interitem

correlations among positive and negative symptom items, are rarely reported in studies on cognitive correlates of symptom patterns. Although interrater reliability is typically reported, it is not a substitute for scale reliability.

The importance of examining scale reliability is illustrated by Addington and Addington's (1991; Addington et al. 1991) recent longitudinal study. Moderate interitem correlations were found among negative symptoms ($0.45 \leq r \leq 0.57$), but interitem associations for positive symptoms were so low ($-0.01 \leq rs \leq 0.35$) that the investigators felt it inappropriate to sum the SAPS item scores into a single scale. These investigators found a number of significant relations between negative symptom total and neuropsychological measures, but virtually none with the positive symptom items, each of which was considered separately. Single item "scales" are generally unreliable, so the absence of correlations between each positive symptom and neuropsychological measures is not surprising. This is an extreme case, but it demonstrates the importance of the psychometric properties of symptom measures in studies of their correlates.

Green and Walker (1986) examined the relation between the severity of positive and negative symptoms and a wide range of neuropsychological functions in an inpatient sample. (Unless otherwise indicated, the studies reviewed evaluated positive and negative symptoms with the SAPS and the SANS, respectively.) The data were evaluated by (1) comparing the performance of patients classified into positive type, negative type, and mixed type, depending on the preponderance of symptoms and (2) multiple regression models. In the latter, positive and negative symptom scores were treated as continuous variables so that variation in cognitive processing could be related to individual differences on both dimensions as well as on their interaction. Green and Walker (1986) found few consistent trends in the group comparisons, but an interesting pattern emerged in the correlations. Higher negative symptom levels were associated with poorer performance on visual memory (Benton Visual Retention Test; Lezak 1983) and motor speed and dexterity tasks (Perdue Pegboard; Lezak 1983). On the other hand, higher positive symptom levels were associated with deficits on verbal memory (Buschke's selective reminding procedure [Buschke and Fuld 1974]) and language comprehension (Token Test) deficits.

On the basis of the Green and Walker (1986) study and others, Walker and Lewine (1988) offered the generalization that visual-processing and motor deficits are associated with negative symptoms, while auditory-processing deficits are linked with positive symptoms. Nuechterlein et al. (1986) found negative symptoms (assessed with the Brief Psychiatric Rating Scale [BPRS; Overall and Gorham 1962]) linked with poor performance on tasks that make heavy demands on visual and motor processes, visual vigilance on the Continuous Performance Test (CPT; Rosvold et al. 1956), and target discrimination on the Span of Apprehension (Neale 1971) tasks; this has been partially replicated by Strauss et al. (in press), who found a correlation between BPRS negative symptoms and Span of Apprehension, but not CPT. Studies of backward masking also indicate that negative symptoms are associated with deficits on tasks involving rapid processing of visual information (see Green and Walker 1984; Braff 1989; and Merriam et al. 1990, who used the Positive and Negative Syndrome Scale [PANSS; Kay et al. 1989] to assess symptoms).

Difficulty in processing auditory verbal information, especially on tests of distractibility, is a robust correlate of positive symptoms. In a study that assessed selective attention by measuring the decrease in digit recall caused by distraction, Oltmanns and Neale (1975) demonstrated that auditory distractibility was a specific, differential deficit. Using this task with groups of positive symptom, negative symptom, and mixed symptom schizophrenia subjects, Walker and her colleagues (Green and Walker 1986; Walker and Harvey 1986) found significant selective attention deficits only for the positive symptom group. Unfortunately, correlational analyses were not reported in these studies. Green and Walker (1986) also studied dichotic shadowing alone with digit-span distraction. There were no differences between symptoms-type groups on the shadowing task, although there were on auditory distraction. So, the association between positive symptoms and auditory processing deficits is not seen consistently.

The effects of distractions on an auditory task were also studied by Cornblatt et al. (1985). They investigated the effects of two kinds of distraction on the performance of a task in which auditorily presented names had to be matched to pictures. One distraction condition consisted of irrelevant noises that partially overlapped with the critical auditory stimuli. In the sec-

ond distraction condition a story was recited by a male voice, while the target words were spoken by a female voice. Negative symptoms were not correlated with performance in any of the conditions. Positive symptoms were unrelated to scores in the baseline no-distraction condition but were correlated with increased errors with both kinds of distractions.

The partially masking distraction condition was a selective attention task because the sounds were irrelevant information. The narrative text condition, however, was a divided attention task. The auditorily presented text was not irrelevant for the subjects: they were warned that a memory test for the narrative material would follow the word-picture task. Although positive symptoms were related to performance on the name-picture match task in both selective- and divided-attention conditions, they were not related to memory for the narrative material. Instead, performance on the multiple choice memory test was related to negative symptoms. The "overload condition" memory task, as Cornblatt et al. (1985) termed the narrative distraction condition, is psychologically complex, so it is difficult to attribute the symptom association to any specific psychological process.

It is noteworthy that positive symptoms predict performance on tasks in which language stimuli are presented auditorily because hallucinations in schizophrenia are predominantly auditory and verbal. It would be interesting to evaluate the differential relationship of auditory and visual verbal learning tasks and of tone and shape discrimination tasks to positive and negative symptoms. This would clarify whether positive symptoms are related to processing in the auditory modality or to language stimuli.

The hypothesis that stimulus modality accounts for relations of cognitive tasks with positive and negative symptoms may be correct, but it does not address the processes that connect performance deficits and symptom manifestations. Identifying the mechanisms in single tasks is itself problematic since there is no isomorphism between a performance deficit and a single processing impairment. The deficits that schizophrenia subjects have on even "simple" tasks such as backward masking and visual vigilance may indicate dysfunctions at sensory/input processing or later cognitive levels.

Isolating deficient neurocognitive processes requires an integrated series of studies that use distinctly different, theoretically linked tasks or measures. Knight (1987) argued that such focused, analytic experimental designs are also necessary for the study of cognitive differences between schizophrenia subgroups or of relations between cognitive dysfunctions and symptoms. In his view, theoretical models should guide the design of a set of information-processing paradigms to measure the cognitive function of interest. The test of a hypothesized relationship between a symptom, or symptom cluster, and information processing then is in the pattern of performance deficits across a series of measures. Knight (1987) suggests that this approach is conceptually more powerful than the psychometric task-matching strategy proposed by Chapman and Chapman (1978). His approach to testing theories depends on the availability of cognitive models that are precise enough to permit the design of a set of converging experimental paradigms. A statistical approach to more explicit specification of hypothesized mediating processes is offered by latent trait modeling (Bentler 1980). This technique has been useful in some areas of psychological research but may have only limited applicability in psychopathology research. The design requirements of latent trait studies, particularly multiple measures of each construct, large samples, and cross-validation, are difficult to implement in clinical studies of schizophrenia (see Breckler [1990] for a review of uses and misuses of this approach). The cognitive science strategy of deriving experimental manipulations from connectionist models may be a more feasible approach to theory development and testing. This is illustrated in Cohen and Servan-Schreiber's work (1991, 1992), which is discussed below.

The interpretation of relations between cognitive and symptom measures is plagued by a number of issues. As with symptom measures, insufficient attention has been paid to the psychometric properties of test batteries. It is difficult to isolate constructs with tests that are factorially or componentially complex, as are most neuropsychological measures (Lezak 1983). Further, relations between clinical measures and cognitive tasks can be psychometric artifacts caused by the differences among tasks in discriminating power in the specific samples studied (Chapman and Chapman 1978; Strauss and Allred 1987). Studying symptom-performance associations using test batteries may not permit the identification of links to specific deficits. The correlational patterns may reflect general performance deficiencies, especially if the tasks

have differential sensitivity (Chapman and Chapman 1978; Knight 1987).

Measures that are independent in some populations may be correlated in patients with schizophrenia, raising the possibility that in schizophrenia the cognitive task measures a generalized deficit rather than a specific ability or process (Chapman and Chapman 1978). An example of this may be found in Addington et al.'s (1991) study of the relations between neuropsychological deficits and positive and negative symptoms. They found significant relations between negative symptoms and both general intellectual ability (IQ) and executive functions (as assessed by the Wisconsin Card Sorting Test [WCST; Heaton 1981] and Category Instances Fluency Test [Lezak 1983]) both at index hospitalization and at a 6-month followup. Factor analysis showed that the executive function measures and IQ loaded on the same dimension. Executive function measures, such as the WCST and Category Fluency, and general IQ tend to be independent in other groups (Stuss and Benson 1986). This suggests that all three tests may be measures of general intellectual inefficiency in schizophrenia. Wagman et al. (1987), using a test battery selected to tap independent functions, also found a general cognitive performance factor in their factor analysis of a neuropsychological battery in a sample of outpatients with schizophrenia.

As noted earlier, the measurement of positive and negative symptoms has also been problematic. The constructs themselves are not without ambiguity. There are several overlapping but far from isomorphic conceptualizations of positive and negative symptoms and measures thereof (McGlashan and Fenton 1992). Initially, positive and negative symptoms were construed as unidimensional (Andreasen and Olsen 1982), giving rise to the classification of patients into positive, mixed, or negative symptom types. Other investigators reported that the two dimensions were independent, conforming to Strauss et al.'s (1974) earlier formulation (Walker and Lewine 1988).

Neither a unidimensional nor a two-factor model appears to account for the correlation patterns typically observed between SANS and SAPS component scales. Arndt et al. (1991) recently confirmed the findings of Bilder et al. (1985) and Liddle (1987) that the SANS and SAPS subscales are best summarized by a three-factor model. The SANS components all load on one factor. The SAPS scales divide into two independent dimensions, one defined by delusions and hallucinations and the other by positive thought disorder and bizarre behavior. Arndt and associates' (1991) study applied multiple approaches to factor analysis to three reasonably large samples (n's = 55–93). The findings of this study, combined with the findings from samples in New York (Bilder et al. 1985) and England (Liddle 1987), indicate that the three-component structure of negative symptoms, delusions/hallucinations, and thought disorder is robust. Addington and Addington (1991) reported this same structure in a sample of clinically stable patients, although the structure was not as clear-cut when the symptom ratings were obtained while the patients were acutely psychotic.

The studies reviewed have assessed negative symptoms cross-sectionally and without regard to origins, an approach criticized by Carpenter et al. (1991), who argue that transitory negative symptoms must be differentiated from persistent (present longitudinally), primary (independent of episode, depression, or medication) negative symptoms, which constitute their deficit state of schizophrenia. The latter should be the focus of study of neurocognitive correlates of negative symptoms.

As noted above, two approaches have been used to analyze the relations between positive/negative symptoms and cognitive measures: (1) comparing patients grouped as positive, negative, or mixed, and (2) correlating symptom scores and cognitive scores. Which one is preferable is partly a psychometric issue. More information is used in the correlational analyses (Green and Walker 1986), and any group comparison analysis of variance can be modeled with regression using the full range of scores (Cohen and Cohen 1983). Furthermore, grouping patients into positive, negative, and mixed types is appropriate only if positive and negative symptom clusters are poles of a single dimension, as Andreasen and Olsen (1982) and Crow (1985) initially proposed. Unidimensionality is indicated by a negative correlation between positive and negative symptom scores, which is not commonly found. The correlation between positive and negative symptoms varies with the population studied (Walker and Lewine 1988).

In a cross-sectional study, Bilder and associates (1985) found that the thought disorder/bizarre behavior factor and, to a lesser extent, the negative symptom factor were correlated with neuropsychological deficits. Hallucinations and

delusions did not correlate with any neuropsychologial measures. Some of the cognitive deficits in schizophrenia appear to be state markers that wax and wane with episodes of psychosis, while others are more enduring trait-like features (Zubin and Spring 1977). Negative symptoms seem to be more trait-like than hallucinations and delusions and are less responsive to neuroleptic treatment (e.g., Lewine 1990). Consequently, it would be interesting to determine whether the cognitive correlates of negative symptoms are more persistent than those of the psychotic symptoms. Longitudinal studies of cognitive deficits in schizophrenia in relation to clinical state are needed to answer this question.

In a rare longitudinal study, Addington et al. (1991) examined 38 patients while they were acutely ill and again at 6-month followup. Negative symptoms were related to poor performance on measures of verbal reasoning both during hospitalization and at followup. Poor verbal fluency was associated with high levels of negative symptoms and low ratings of thought disorder on both occasions. At initial testing, better verbal reasoning was associated with presence of delusions. Bizarre behavior was related to poor verbal reasoning during hospitalization, as Bilder et al. (1985) also found. The association between delusions and cognitive performance was not seen 6 months later, but the relation of cognitive performance with bizarre behavior continued. Substantial change in positive symptoms occurred in the 6-month period. Improvement in these symptoms was related to improvements in cognitive functions. Conversely, negative symptoms changed little, and as expected this small change was not associated with change in neuropsychological performance. Additional studies of the relationship of symptoms and cognitive deficits across time would be useful.

Neuropsychological Analysis of Positive and Negative Symptoms

The current status of the positive and negative symptom constructs from descriptive, genetic, and treatment approaches was the subject of an international conference (Marneros et al. 1991). In addition, two novel, provocative, conceptual analyses of the neuropsychological substrates of positive and negative symptoms have also been advanced. The first is by Frith, who, in two essays (Frith 1987; Frith and Done 1988), proposed that a failure to monitor actions internally results in the positive symptoms of hallucinations and delusions, while defects in the initiation of spontaneous action underlie negative symptoms. Both deficits are metacognitive processes, that is, monitoring or executive systems, rather than processing mechanisms. Indeed, Frith explicitly rejected his earlier theory, which emphasized selective attention deficits in the genesis of positive symptoms (Frith 1987; Frith and Done 1988). He links his current model to neuropsychological evidence for two separate routes to action, one that is stimulus-driven by environmental events and a second that is self-generated ("willed intention"). More specifically, this model proposes that information about self-generated acts fails to reach a monitor system, resulting in the experience of "alien forces" as in delusions of control, unintended thoughts, the experience of passivity, and other delusions and hallucinations.

In a study of performance on two arcade-like shooting tasks, Frith and Done (1989) tested the hypothesis that experiences of alien control reflect a deficit in a self-monitoring system. In both tasks the subject had to "shoot down" a bird that appeared on either the left or right of the screen by firing the "gun" on the opposite side of the screen. Subjects could correct errors at any point in a trial. In one task, there was immediate visual feedback: the "bullet" emerged from the gun. In the second, a wall hid the trajectory of the "bullet" for the first 2,000 msec after the response. Four groups were compared: schizophrenia patients with delusions of alien control, schizophrenia patients without this symptom, affective psychotic patients, and normal controls. The groups did not differ in error rates when there was immediate visual feedback, and virtually all errors were corrected within 2,000 msec. However, when the trajectory of the "bullet" was hidden from view, patients with experiences of passivity and alien control were significantly less able to correct their errors. They caught up once the "bullet" became visible in the last 800 msec of a trial, so the deficit in error correction was restricted to the period in which error correction depended on internal monitoring of an action.

The authors suggest that a similar deficit in monitoring one's own responses can account for anomalies of auditory event-related potentials (ERPs) in schizophrenia. In normal controls, the P300 component of the auditory ERP is

smaller when subjects trigger stimuli than when the tones are externally controlled. This expectation effect is attenuated in schizophrenia (Braff et al. 1977). Interestingly, problems in both monitoring and initiation of behavior have been demonstrated in animals following lesions of the frontal cortex (Fuster 1989). Frith and Done have proposed that frontal damage is the basis of both positive and negative symptoms (Frith 1987; Frith and Done 1988). In their theory, positive symptoms and associated cognitive deficits reflect prefrontal cortical impairments in interaction with the septohippocampal system. Negative symptoms and associated cognitive deficits are due to disruptions in frontal-striatal connections.

The second new neuropsychological analysis of symptoms of schizophrenia comes from Gray et al. (1991). Their detailed, admittedly somewhat speculative, model focuses on positive symptoms of schizophrenia, which, following Crow (1985), they refer to as Type I schizophrenia. The key concept in their model is that automaticity in information processing fails to develop fully in Type I schizophrenia. Consequently the behavior of patients with positive symptoms is less influenced by regularities of past experience than is the behavior of normal controls.

One way that immediate past experience influences learning and memory in humans and other mammals is shown in the phenomenon of latent inhibition (LI) (Lubow 1989). LI refers to the retardation of association learning by preexposure to the stimulus that is to be conditioned. During preexposure, the stimulus is repeatedly presented but has no signal value. Normally, animals habituate to repetitive, uninformative signals. Consequently, when contingencies change and the stimulus becomes informative, it takes longer for preexposed subjects to learn the contingency than subjects not previously exposed to the stimulus. There is a dopaminergic basis for LI, and preliminary evidence indicates that preexposure does not produce LI in symptomatic Type I patients (Gray et al. 1991), who are presumably in a hyperdopaminergic state. Acutely ill patients do not show the preexposure effect in a number of experimental paradigms, while chronic patients (i.e., those with fewer positive symptoms) and patients with short histories of illness who are effectively treated with neuroleptics do show LI (Gray et al. 1991).

Prepulse inhibition of eye-blink startle (Braff et al. 1991) and sensory gating evoked-potential paradigms (Freedman and Mirsky 1991) also reveal abnormalities in the responses of schizophrenia subjects because an immediately preceding stimulus fails to control information processing. Schizophrenia patients show less inhibition of a startle response preceded by a weak stimulus trial and show less attenuation of the ERP P50 to the second of a pair of auditory signals. The failure of selective attention produces the typical outcome in these studies of schizophrenia: the performance of patients is poorer than that of controls. In LI paradigms, on the other hand, the failure of selective attention in positive symptom patients results in better learning than in controls.

Gray et al. (1991) submit that the genesis of positive symptoms and cognitive abnormalities in Type I patients lies in the interactions of corticolimbicstriatal brain systems and the septohippocampal complex, which serves as a monitor or comparator of current stimuli, memory, plans, and motor programs. As previously discussed, the septohippocampal system is also central to Frith and Done's (1988) account of self-monitoring disturbances in positive symptom schizophrenia.

There are differences as well as similarities in these two recent theories. Frith's theory focuses on internal monitoring and planning of action (Frith 1987; Frith and Done 1988). In contrast, the Gray et al. (1991) theory emphasizes input disturbances; that is, disruptions in the attentional mechanisms that facilitate the development of automatic processing (see also Hemsley 1991, in press). Both theories are exciting integrations of psychological and neuroscience perspectives on schizophrenia. They offer explicit, testable models of the mechanisms of symptoms and cognitive deficits. Both the Frith-Done and Gray-Hemsley theories provide more detail on positive than on negative symptoms, although each offers hypotheses about the neurocognitive bases of the latter as well. The Gray-Hemsley model has treated positive symptoms as a more global construct than has the Frith and Done model, which focuses more on discrete symptoms and specific associated cognitive abnormalities. As discussed earlier, there is substantial psychometric evidence of the independence of delusions and hallucinations from thought disorder. Consequently, a focus on specific symptoms rather than on the positive symptom category would be beneficial in the further development of neuropsychological theories.

Relations of Cognitive Functions to Specific Symptoms

Persons (1986) and, more recently, Costello (1992) have discussed advantages of focusing psychological (or biological) analysis on the explanation of discrete symptoms instead of syndromes. Both of these authors noted the heterogeneity in patient groups that is inevitable when disjunctive criteria are used to define a diagnostic construct. Persons (1986) also posited that a focus on the cognitive analysis of specific symptoms rather than symptom clusters (syndromes) can benefit research. Focusing on symptoms underscores the continuity between normal and clinical phenomena and encourages more careful definition and analysis of symptoms themselves. Much of the research on symptom-cognition relations is based on plausibility rather than on firmly grounded hypotheses (Neale et al. 1985). This makes inconsistent findings difficult to reconcile.

Certainly, a focus on discrete symptoms can facilitate theoretical development by fostering more elaborate, tighter explanatory links between proposed mechanisms and the clinical phenomena (Persons 1986; Costello 1992). Knight (1987), however, noted a potential pitfall in this approach. If symptoms are studied independently of diagnosis, one must assume that the mechanisms of symptoms are the same across syndromes. Although there may be a final pathway that is common to delusions in schizophrenia and, for example, mania, there may be different processes involved as well (Neale 1988). Knight (1987) refers to the possibility of different symptom-psychological mechanism relations across disorders as symptom-process equivocality.

As discussed above, subtyping schizophrenia into paranoid-nonparanoid, premorbid adjustment, and chronicity subgroups reduced within-group variability and isolated some distinctive patterns of cognitive deficits. The subgrouping variables thus were "markers" for cognitive differences within schizophrenia. Theoretical analysis in this literature, as that of Type I/Type II group differences, tended to deal with the mechanisms of cognitive deficits rather than the mechanisms of the symptoms or the processes that might mediate both (Silverman 1964, 1972; Venables 1964; Cromwell 1975). Persons' (1986) and Costello's (1992) articles emphasize a different tack in the study of cognitive processes in conjunction with symptomatic characteristics of schizophrenia: the psychological analysis of the symptoms themselves. The Frith-Done and Gray-Hemsley theories are examples of highly generalized studies of this sort of analysis. Two specific symptoms, hallucinations and delusions, have been the subject of recent empirical study and theoretical analysis (Persons 1986; Oltmanns and Maher 1988; Slade and Bentall 1988; Bentall 1990).

Hallucinations. A number of cognitive mechanisms of hallucinations have been proposed, and four principal theoretical approaches were reviewed by Bentall (1990): (1) classical conditioning of hallucination-like experiences (e.g., visual afterimages); (2) "seepage" of preconscious mental processes or contents, either through overarousal or dysfunction in executive controls over access of preconscious processing to consciousness (Frith 1979); (3) abnormally vivid mental imagery in conjunction with defective reality testing, or deficits in imagery with misattribution of those images that are experienced; and (4) subvocalization theories. This last group of theories posits that hallucinations are caused by dysfunctions in the neural mechanisms of the subvocalizations that typically accompany thought. Bentall (1990) discussed a number of limitations of each of these approaches and elaborated the view that hallucinations represent a breakdown in the metacognitive processes of reality discrimination. Reality discrimination refers to the idea "that hallucinators mistake their own internal, mental, or private events for external, publicly observable events" (Bentall 1990, p. 88). That is, hallucinations result from deficits in the ability to discriminate between real and imaginary events.

Failures in reality discrimination have been demonstrated by Heilbrun and his associates in studies of patients with histories of auditory hallucinations. In the first study, Heilbrun (1980) contrasted the performance of hallucinating and nonhallucinating inpatients on tasks that required recognizing expressed thoughts as one's own. Patients were first interviewed, and their responses to five general questions were recorded. One week later, subjects were tested for their ability to recognize their own expressed thoughts using multiple choice tests. Patients with a history of hallucinations were less able to recognize their own thoughts, although their memory, communication skill, or stability of opinions did not differ. In a subsequent study, Heilbrun et al. (1983) found that patients with a history of au-

ditory hallucinations and poor premorbid adjustment were less accurate in localizing the direction of the experimenter's voice than nonhallucinating patients or hallucinating patients with good premorbid adjustment. The patients studied included those with depressive and personality disorders as well as schizophrenia. The authors stated that comparisons of only the schizophrenia patients would have produced even stronger effects.

The reality-discriminating abilities of currently hallucinating schizophrenia patients, delusional psychotic patients without hallucinations (mainly schizophrenia subjects), and normals were studied by Bentall et al. (1991*a*). In this study, subjects were first required to provide responses to a series of cues (e.g., name a kind of dwelling that begins with H) or were provided with responses to cues (a type of footwear is a shoe). One week later, without forewarning, the subjects were required to recall which responses were self-generated and which had been provided. Hallucinators were less able to discriminate between memories of their own thoughts and memories of information given to them by the experimenter.

Hoffman (1986) also proposed that misattribution processes accounted for auditory hallucinations as part of a more comprehensive theory of language production failures in schizophrenia. In brief, Hoffman's (1986) hypothesis is that hallucinators experience alien voices because of disorders of discourse planning that lead to the experience of unintended verbal images. This model brings together speech disorganization, language processing, and hallucinations, thus directly linking specific cognitive deficits to specific symptoms. As commentators on Hoffman's (1986) article noted, there are no direct tests of this idea, but it can lead to interesting experimental studies.

Schizophrenia is characterized by hallucinations in modalities other than audition, so a focus on metacognitive processes rather than specific cognitive mechanisms such as audition or language processing may be a useful strategy. A great deal is known about the similarities, differences, and interferences between imagination and perception in normal cognition. Johnson (1988) recently reviewed the cognitive psychology of confusion between perception and imagination in both experiencing and remembering. She summarized evidence from disorders other than schizophrenia that suggested that both visual and auditory hallucinations may be the results of improper interpretation of fragmentary sensory/perceptual experiences.

Delusions. As noted earlier, factor analyses of positive symptom scales indicate that hallucinations and delusions both load on one factor and are independent of thought disorder. Thus, it might be expected that some of the cognitive processing characteristics of hallucinating schizophrenia patients would also be found in delusional patients. Apparently, however, there have been no direct comparisons of schizophrenia patients with delusions and those with only hallucinations.

There is much more theory than research on the psychology of delusions (Winters and Neale 1983; Butler and Braff 1991). Oltmanns and Maher (1988) edited a volume that presents a number of theoretical perspectives on delusions derived from cognitive psychology, hypnosis, and social psychology, as well as Maher's views, which are a jumping-off point for several of the other chapters.

A theoretical link between delusions and hallucinations was made by Johnson (1988) in terms of reality monitoring, a perspective that builds on the theoretical views of Maher (1988). Maher proposed that delusions are the product of normal reasoning processes applied to aberrant experience. He documented both the range of evidence for anomalous experience in the genesis of delusions and the absence of evidence of abnormal logical thinking in studies that compare paranoid patients with controls.

Chapman and Chapman (1988) presented findings from their high-risk group study of college students that are germane to Maher's (1988) theory. They studied college students who scored very high ($\geq$ 2 standard deviations [SDs] above the mean) on their Perceptual Aberration and/or Magical Ideation scales (Chapman and Chapman 1988). These subjects, as well as controls, were interviewed twice over a 2-year period with an expanded version of the Schedule for Affective Disorders and Schizophrenia (SADS-L; Spitzer and Endicott 1977) which permitted ratings of 80 types of deviant experiences. At the first interview, about half the psychometric high-risk group met *DSM–III* criteria for schizotypal personality disorder. In the small number of high-risk subjects who developed psychosis over the followup period (n = 3), there was similarity between the aberrant beliefs expressed in the first interview and the delusions exhibited during the psychosis. Isolated delusions were reported on followup by nine ad-

ditional high-risk subjects; these symptoms were more extreme than those manifested on the first interview. These findings suggest a continuity of aberrant beliefs and delusions in schizophrenia. Such continuity was also seen by Harrow et al. (1988) in a retrospective study of patients with schizophrenia.

Chapman and Chapman (1988) also noted that not all subjects with similar anomalous experiences developed aberrant beliefs. Likewise, some subjects developed delusional interpretations of experiences generally not considered anomalous (e.g., at a party a friend "reads your mind" and says, "Let's go"). The Chapmans suggested that the genesis of delusions lies in the interplay of anomalous experiences and two cognitive abnormalities of schizophrenia. These two abnormalities are cognitive slippage, which became apparent in their subjects when they talked about psychotic and psychotic-like experiences, and the tendency to process information in a biased manner. The Chapmans suggest that people who develop delusions assign excessive significance to certain aspects of their experiences and show constriction in the range of information they use.

Magaro (1980) also commented on the tendency of paranoid subjects to attribute meaning according to rigid conceptual expectations. Paranoid patients show a rush to closure (Neufeld 1990) and tend to discover relationships between stimuli that do not exist (Brennan and Hemsley 1984). Since the "discovery" of illusory correlations is a normal cognitive phenomenon (Chapman and Chapman 1969), it may be that paranoid patients reason like the rest of us, but to a greater extent.

In a theoretical review of the formation and maintenance of delusions, Hemsley and Garety (1986) proposed that deficits in the ability to weigh evidence may underlie delusions. They suggested that Bayesian inference could serve as a normative model against which to evaluate the inference processes of deluded and nondeluded individuals. Within a Bayesian framework, cognitive biases can occur at a number of points in the generation of hypotheses (plausible inferences), estimation of probabilities, and assessment of likelihood ratios. Garety et al. (1991) subsequently studied the reasoning of delusional patients (paranoia/delusional disorder), deluded schizophrenia patients, and anxious patients, as well as normal controls, on probabilistic inference tasks. Since delusion-relevant content can perturb cognition, two content-neutral tasks were used. As expected, they found that both delusional groups requested less information before making a decision. However, the hypothesis that delusional groups would be more overconfident in their probability judgments was not supported. In Bentall and associates' (1991*b*) a study of social attribution, delusional patients were more certain about their choices than normal controls, but they did differ from depressive patients.

Garety et al. (1991) reported that delusional patients were more likely to be extreme responders than were members of the other groups. In post hoc comparisons of extreme and nonextreme delusional responders, they found extreme responders had higher degrees of conviction in their own beliefs and were more likely to have had anomalous experiences (e.g., auditory hallucinations, strange coincidences). Thus, for at least a subgroup of patients, abnormal inferences and abnormal perceptions went hand in hand. Other delusional patients showed neither characteristic (Garety et al. 1991). Disregarding the possibility that the measurement of anomalous experience and inference processes may not have been sufficiently sensitive, Garety et al.'s (1991) findings suggest possible heterogeneity of the mechanisms of delusions. On the other hand, there were no differences between the schizophrenia patients with delusions and the nonschizophrenia groups.

Bentall and associates (Kaney and Bentall 1989; Bentall et al. 1991*b*) also studied the notion that the differences between the reasoning proclivities of delusional and nondelusional patients are mainly quantitative. Social attribution theory was the framework used to study reasoning in delusional, depressed, and normal subjects. In the first of two reports (Kaney and Bentall 1989), delusional patients, like depressed patients, expressed strong beliefs in chance events and made excessively stable and global attributions in comparison with normal subjects. The delusional patients, however, made external attributions for negative events, while the depressed patients made internal attributions for negative events and external attributions for positive events. In their second study (Bentall et al. 1991*b*), subjects selected one of three explanations for the actions of one person toward another. The choices were attributing the cause to a person, to a stimulus, or to the circumstances. The paranoid patients made more person attributions, especially for negatively val-

ued actions, although there were many similarities in performance among the groups.

The content of thought, as well as form, were considered by Hemsley and Garety (1986) in their theoretical analysis of delusions. An earlier study from their laboratory (Brennan and Hemsley 1984) had shown that there were strong perceptions of correlation between unrelated events ("illusory correlation") for stimuli related to patients' delusions. Chapman and Chapman (1988) also noted greater cognitive slippage among their high-risk subjects when they described delusional ideas.

Further evidence of the disruptive effects of personally relevant stimuli on the information processing of delusional patients has been provided by Bentall and Kaney (1989). These investigators administered a Stroop color-word interference test (Lezak 1983), a measure of selective attention, to a group of patients with persecutory delusions, to a depressed group, and to normal controls. The stimulus words were either content-neutral or had depressive or paranoid content. The subject's task was to name the color in which the word was printed. Using color-naming speed for neutral words as a referent, the researchers found that depressed patients showed greater interference with depressive words and deluded patients were slow on words with paranoid themes. Depressed and paranoid patients also showed memory biases in recall of thematic material. Depressed subjects more easily recalled depression-related stimuli, while delusional patients recalled threat-related material more easily (Kaney et al. 1991). Both symptom-specific and more general information-processing abnormalities are found among delusional patients.

Overview and Future Needs

Systematic relationships exist between clinical aspects of schizophrenia and cognitive or neuropsychological parameters whether they are studied at the level of subtypes, symptom dimensions, or specific symptoms. Individual differences among patients in the severity of negative symptoms are related to some classes of information processing, while variability in positive symptoms is related to different cognitive measures. Specifically, deficits in memory, perceptual-motor integration, and visual information processing appear to be associated with negative symptoms, while auditory information processing, especially of language stimuli, is a principal correlate of positive symptoms.

The theoretical basis of these relations is relatively undeveloped. The first wave of interest in this topic occurred at a time (the 1960s) when knowledge of neural mechanisms of cognition and cognitive theory itself were just emerging, and the boundaries of the schizophrenia construct were not well conceptualized. The second wave of research, that on positive and negative symptom dimensions, was stimulated by interest in evaluating Crow's (1985) assertions about relations between negative symptoms, brain abnormalities, and cognitive deficits. This research was driven by interest in the concept of two distinctive syndromes of schizophrenia, Type I and Type II. Research has been mainly descriptive, with few studies attempting to test directly hypotheses about mechanisms. The relationship between stimulus modality (auditory/visual) and symptom cluster (positive/negative) is open to a number of interpretations.

Two trends are apparent in the most recent work. First, specific positive symptoms rather than the positive symptom construct are being studied. Since it is now clear that positive symptoms are not a single construct, this is a valuable change. Conversely, greater coherence exists among negative symptoms, so they may usefully be treated as a construct. However, the etiology and neurobehavioral mechanisms of this negative construct remain unclear (Carpenter et al. 1991; Marneros et al. 1991). The second trend is the development of explicit neuropsychological theories of schizophrenia, such as Frith and Done's (1988), Gray et al.'s, (1991), and Hoffman's (1986). Although the specifics of each these theories are subject to criticism (see published commentaries in articles by Gray et al. [1991] and Hoffman [1986]), each has enough detail to allow clear hypotheses and tests of these hypotheses.

A common ground is emerging for the explanation of symptoms and cognition in schizophrenia in the interaction of discrete, though distributed neural systems. Cohen and Servan-Schreiber (1992) recently proposed a connectionist model of attention and language processing based on the psychobiology of the prefrontal cortex and the mesolimbic dopamine system. Their computational model successfully simulated the performance of schizophrenia patients on Stroop color-word interference, visual vigilance deficits, and lexical ambiguity tasks. Preliminary tests

of predictions of the model in other information-processing tasks are encouraging (Cohen and Servan-Scheiber 1991). As the phenomenology and cognitive psychology of schizophrenia come to be better understood in terms of neurobiology and neuropsychology, the mechanisms linking these two domains of abnormality will become clearer.

Important questions remain about the relationship between cognition and symptoms. Cognitive vulnerability markers should be present before clinical symptoms. This suggests that the neuropsychological processes of cognitive deficits affect the later development of symptoms. But how tightly linked are cognitive disorders and symptom patterns developmentally? Are they driven by common mechanism or is the link indirect?

Clinically, schizophrenia is a dynamic disorder marked by a variety of courses in symptom expression (Strauss 1987; Marneros et al. 1991). Delusions and hallucinations appear to dissipate over time (Depue and Woodburn 1975; Pfohl and Winokur 1982). But paranoid schizophrenia patients are also less likely to be rehospitalized, so they are less available for study in samples of patients with lengthy illnesses (Strauss 1973). When symptoms change, do neuropsychological or information-processing measures also change? There is scant direct evidence of change in cognitive characteristics over the course of schizophrenia. Most of our knowledge is cross-sectional, measuring differences between chronic patients and those who have not been ill for a long period at one point in time. Such differences may reflect sample biases rather than change (Strauss 1973). To learn whether changes in symptom expression are paralleled by changes in putative cognitive mechanisms, both longitudinal studies and studies of pharmacologic interventions are necessary.

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Acknowledgments

The author is grateful for the comments and suggestions of Drs. Althea M.I. Wagman and Rue Cromwell on an early draft, and also those of Dr. David Braff and Mr. Brian Carpenter.

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The Kurt-Schneider Award is sponsored by JANSSEN, GmbH, for exceptional scientific achievements. The aim of the award is to encourage psychiatric research, especially in the field of schizophrenia, including basic research (clinical psychopathology, biochemistry, physiology, pharmacology, genetics, epidemiology), diagnostics, therapy, and rehabilitation.

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