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Social Cognition in Schizophrenia

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The study of social cognition in schizophrenia may augment the understanding of clinical and behavioral manifestations of the disorder. In this article, the authors describe social cognition and differentiate it from nonsocial cognition. They garner evidence to support the role of social cognition in schizophrenia: Nonsocial information-processing models are limited to explain social dysfunction in schizophrenia, measures of social cognition may contribute greater variance to social functioning than measures of nonsocial cognition, task performance on nonsocial-cognitive measures may not parallel performance on social-cognitive tasks, and symptomatology may be best understood within a social-cognitive framework. They describe the potential implications of a social-cognitive model of schizophrenia for the etiology and development of the disorder.

There appears to be a renewed interest in the psychological and phenomenal aspects of schizophrenia (Amador, Strauss, Yale, & Gorman, 1991; Bentall, 1994; Brekke, Levin, Wolkon, Sobel, & Slade, 1993; Davidson & Strauss, 1992; Frith, 1994; Trower & Chadwick, 1995). These approaches emphasize the view of the self, the world, and others of patients with schizophrenia, as a contribution to symptomatology, psychosocial impairment, and recovery. Underlying such approaches is the notion that schizophrenia is inherently an interpersonal disorder in which problems result from faulty construction of the social environment and one's place in it. Therefore, an important level of analysis becomes the *social cognition* of patients with schizophrenia—the cognitive processes involved in how they think about themselves, other people, social situations, and interactions.

In this article, we review what we know about social cognition in schizophrenia and advocate more research in this area. We propose that the social content and context of stimuli pose particular problems for patients with schizophrenia and that models, which exclusively emphasize nonsocial-cognitive processes, do not adequately explain the social impairment and symptomatology of the disorder. We begin with a brief overview of the social-cognitive perspective, followed by a presentation of evidence that suggests that measures of social cognition contribute variance, beyond measures of nonsocial cognition, to indices of the social functioning of patients with schizophrenia. We review findings that indicate that the task performance of patients with schizophrenia may differ on social-cognitive versus nonsocial-cognitive tasks. We also review research that indicates that social-cognitive models may have particular relevance to understand the symptomatology of schizophrenia. Finally, we discuss how a social-cognitive model of schizophrenia is consistent with what is known about the etiology and developmental course of the disorder. We conclude the article by posing unanswered questions and proposing future research directions.

Before proceeding, we must make one caveat. Although many researchers have tacitly or explicitly argued that the term *schizophrenia* correctly denotes a discrete pathological entity, the classification of psychotic and other psychiatric disorders remains a matter of considerable debate. Some researchers suggested that schizophrenia can be broken down into a small number of discrete syndromes (e.g., Andreasen, Arndt, Alliger, Miller, & Flaum, 1995), others argued that psychiatric disorders in general should be classified using a dimensional approach (Clark, Watson, & Reynolds, 1995), and other researchers even advocated the abandonment of the schizophrenia concept altogether in favor of research targeted at particular symptoms of psychosis (Bentall, Jackson, & Pilgrim, 1988). These debates are beyond the scope of this article. For this reason, we use the term *schizophrenia* to denote the range of psychological and behavioral phenomena commonly associated with the diagnosis and included in diagnostic manuals such as the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed. [*DSM–IV*], American Psychiatric Association [APA], 1994), but we do not prejudge the correct

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classification of these phenomena, which might be determined by future research.

Definition of Social Cognition

Social cognition is a domain of cognition that involves the perception, interpretation, and processing of social information (Ostrom, 1984). Although social and nonsocial cognition are clearly related (Corrigan, Green, & Toomey, 1994), the former is not necessarily derivable from the latter. For example, a participant's performance on tasks that assess "social intelligence" are not strongly predicted by verbal IQ, performance IQ, or other cognitive tasks (Ford & Tisak, 1983; Frederiksen, Carlson, & Ward, 1984; Marlowe, 1986; see Sternberg, Wagner, Williams, & Horvath, 1995, for a review). Thus, nonsocial cognition likely represents a necessary but not sufficient condition for adequate social cognition. This is consistent with the "building-block" view of social cognition (see Ostrom, 1984, for a discussion). The reader, therefore, should view nonsocial cognition and social cognition as different levels of analyses rather than as orthogonal constructs (Spaulding, 1986).

Social cognition differs from nonsocial cognition in a number of specific ways. The first difference concerns the type of stimuli processed. The stimuli often used in studies of nonsocial cognition have been characterized as "numbers, words, or objects," (Corrigan & Toomey, 1995, p. 396) which tend toward being affectively neutral and static (e.g., nonsense syllables, briefly presented digits, and auditory tones). Conversely, social stimuli are typically personally relevant and mutable over time (Fiske & Taylor, 1991; Forgas, 1995). Social stimuli range from the perception of specific individuals to comprehension of the steps that comprise complex social situations (Fiske & Taylor, 1991). Furthermore, whereas the unobservable attributes of social-cognitive stimuli are vital (e.g., inferences about others' personality based on observation of interpersonal behavior), such unobservable attributes are less important for nonsocial-cognitive stimuli (Fiske & Taylor, 1991).

Nonsocial cognition and social cognition also differ with respect to the relationship of the perceiver to the stimulus. For nonsocial-cognitive stimuli, the perceiver's relationship tends to be unidirectional—the perceiver acts on nonsocial-cognitive stimuli not vice versa. In social cognition, the stimulus (e.g., another individual) can also act on, perceive, or gather information on the perceiver. For example, the perceiver can be complimented or disparaged by the social stimulus. This process is described by Fiske and Taylor (1991) as "mutual cognition." Furthermore, social-cognitive stimuli can change as a function of being observed (i.e., reflexivity), which can, in turn, affect the stimulus' effect on the observer. Thus, the relationship between perceiver and stimulus in social cognition tends to be interactive.

A final difference between nonsocial and social cognition concerns how performance is evaluated. Most of the work on nonsocial cognition in schizophrenia has focused on deficits, although there are some exceptions (e.g., Harrow, Lanin-Kettering, & Miller, 1989; Huq, Garety, & Hemsley, 1988; Knight, 1984). Such work is based, in part, on group contrast studies, in which performance on nonsocial-cognitive tasks is found to be more impaired for patients with schizophrenia relative to controls. Conversely, research in social cognition includes evaluation of biases in addition to deficits. *Biases* refer to a characteristic response style that does not necessarily indicate poor task performance. For example, mental health in individuals is associated with unrealistically positive evaluations of the self (relative to evaluation of others), an exaggerated sense of control in random situations, and an overly optimistic vision of the future (S. E. Taylor & Brown, 1988, 1994; see Colvin & Block, 1994, for an alternative view). Because social cognition often involves such information-processing biases, it may be inappropriate to judge participants' responses as either correct or incorrect against normative standards. Whereas in studies of nonsocial cognition, it is usually possible to determine the accuracy of each participant's performance; in studies of social cognition, it may only be possible to note differences between participants and conditions (i.e., we cannot state whether it is "correct" to attribute observed behavior to traits or to situations because social behavior is "multidetermined").

Social-cognitive biases have been investigated in depression (Segal, Hood, Shaw, & Higgins, 1988), posttraumatic stress disorder (McNally, Kaspi, Reimann, & Zeitlin, 1990), and social phobia (Hope, Rapee, Heimberg, & Dombeck, 1990; Mattia, Heimberg, & Hope, 1993). Application to schizophrenia, however, has been limited primarily to studies of the role of such biases in delusions and hallucinations (see Bentall, 1990; and Bentall, Kinderman, & Kaney, 1994, for reviews). Therefore, researchers of nonsocial-cognitive models may be ignoring a critical aspect of information processing in schizophrenia samples by focusing predominantly on performance deficits.

The distinctions between social and nonsocial cognition are made not only by social psychologists but also by researchers in fields as diverse as evolutionary biology (Cosmides, 1989; Cosmides & Tooby, 1989) and primate behavior (Brothers, 1990a, 1990b). For example, Cosmides and Tooby (1994) have argued that cognitive research in the social sciences is based on the (faulty) assumption that the mind comprises general, content-free processes. This model, coined the standard social science model, is incompatible with findings from evolutionary biology, which indicates that natural selection has shaped how individuals reason and that reasoning for adaptive problems (e.g., ones of social exchange) involves highly specific cognitive mechanisms. According to Cosmides (1989),

> the innate information-processing mechanisms that comprise the human mind were not designed to solve arbitrary tasks, but are, instead, *adaptations:* mechanisms designed to solve the specific biological problems posed by the physical, ecological, and social environments encountered by our ancestors during the course of evolution. (p. 188)

In a series of nine studies, Cosmides demonstrated that participants' reasoning for both familiar and unfamiliar rules (e.g., "If a man eats a cassava root, then he has a tattoo on his face") improved when the rules were embedded in a social- versus nonsocial-exchange context. These findings lend support for the hypothesis that certain cognitive processes are specialized to solve social rather than nonsocial problems.

Social cognition has also been observed in primates. For example, primatologists have observed that chimpanzees and baboons engage in "tactical deception" in various social situations

(e.g., a subordinate male chimp courting a female chimp would hide his erection only in the presence of the dominant, alpha male chimp; Premack & Woodruff, 1978; Whiten & Byrne, 1988). Other evidence, reviewed by Brothers (1990b), suggests that primates, at least to some extent, are able to make inferences about the intentions and motivations of others (e.g., primates attempting to "fix" a situation that is perceived as distressing to other members of their group). Consistent with the notion of "domain specificity" noted by Cosmides (1989) in humans, Brothers (1990b) stated that

> logical operations carried out by monkeys in a social setting, with other individuals as the 'terms,' do not seem able to be executed with inanimate objects in the laboratory. There appears to be, then, a 'social intelligence' distinguishable in primate evolution from general intelligence. (pp. 31–32)

To summarize, we have argued that social cognition and nonsocial cognition represent different levels of analyses to understand human functioning. We reviewed evidence that indicates that social cognition represents a specialized domain of cognition developed to solve social, adaptive problems. For the purpose of this article, we offer an operational definition of social cognition to provide a conceptual framework for the ensuing sections. Due to the range of characteristics included under the rubric of social cognition (described above), our definition is by intention broad. Thus, *social cognition* consists of the mental operations underlying social interactions, which include the human ability and capacity "to perceive the intentions and dispositions of others" (Brothers, 1990b, p. 28). This includes human skills in areas such as social perception, attributional analysis, and empathy and reflects the influence of social context and content on performance. This definition, and others like it (see Fiske & Taylor, 1991, for a review), places "traditional" information processing (or nonsocial cognition) within a social context. Tasks or stimuli that are void of social content or not performed in an interpersonal context are considered nonsocial-cognitive stimuli.

Limitations of Nonsocial Information-Processing Deficit Models

A plethora of researchers have investigated the role of information processing in schizophrenia. Deficits in information processing have been implicated in the development (Cornblatt, Lenzenweger, Dworkin, & Erlenmeyer-Kimling, 1992; Erlenmeyer-Kimling et al., 1993), course (Nuechterlein & Dawson, 1984b; Nuechterlein et al., 1992), clinical presentation (M. S. Strauss, Buchanan, & Hale, 1993), and social impairment of schizophrenia (Breier, Schreiber, Dyer, & Pickar, 1991; Corrigan & Toomey, 1995; Penn, Mueser, Spaulding, Hope, & Reed, 1995; Spaulding, 1978). Such deficits form the basis of the diathesis–stress model; chronic impairment in information processing interacts with stressful life events (or enduring stressors) to lower the vulnerable individual's threshold to a relapse (Cromwell & Spaulding, 1978; Nuechterlein & Dawson, 1984a; Zubin & Spring, 1977).

Nonsocial-cognitive deficits have also contributed to understand more complex aspects of functioning, such as social adjustment (Corrigan, Schade, & Liberman, 1992; Liberman et al., 1986). In this vein, nonsocial-cognitive abilities are posited to mediate acquisition of varied behavioral skills, including social skill, interpersonal problem solving, and coping. For example, to learn effective conversational skills, one must be able to attend to another individual and maintain the conversation topic in working memory. Otherwise, the thread of the conversation is lost. A number of studies support the role of nonsocial cognition in skills training (Bowen et al., 1994; Corrigan, Wallace, Schade, & Green, 1994; Kern, Green, & Satz, 1992; Lysaker, Bell, Zito, & Bioty, 1995; Mueser, Bellack, Douglas, & Wade, 1991).

The importance of nonsocial-cognitive deficits in schizophrenia is further underscored by the emergence of cognitive rehabilitation (Corrigan & Yudofsky, 1996; Green, 1993; Penn, 1991; Spaulding, Storms, Goodrich, & Sullivan, 1986; Stuve, Erickson, & Spaulding, 1991). This approach assumes that direct remediation of nonsocial-cognitive deficits may affect the nonsocial-cognitive processes that serve as either vulnerability or episodic markers. For example, researchers have attempted to remediate performance on the span of apprehension task, a measure of early information processing (Kern, Green, & Goldstein, 1995). Thus, stabilization of nonsocial-cognitive impairment should raise the threshold for future relapses to occur.

Despite its apparent ubiquity, there are limitations with nonsocial information-processing models of schizophrenia. First, although nonsocial information processing contributes significant variance to indices of social competence, the mean variance is less than 25% (Bellack, Sayers, Mueser, & Bennett, 1994; Bowen et al., 1994; Corrigan & Toomey, 1995; Dickerson, Ringel, & Boronow, 1991; Lysaker et al., 1995; Mueser et al., 1991; Penn et al., 1995; Penn, Spaulding, Reed, & Sullivan, 1996; Spaulding, 1978; Spaulding, Penn, & Garbin, in press; Wykes, Katz, Sturt, & Hemsley, 1992; Wykes, Sturt, & Katz, 1990). Table 1 summarizes the results of studies in which researchers examined the relationship between nonsocial cognition and social functioning (i.e., a laboratory-assessed social skill or a social behavior in the treatment setting). To provide a context to interpret these studies, we make the following points. First, assessment of social competence was varied, ranging from role-play assessment (e.g., Penn et al., 1995) to group activity and performance (e.g., Dickerson et al., 1991). Second, a number of these studies suffered from low power (the results in Table 1 may be an underestimate of the association between nonsocial cognition and social functioning; see Green, 1996, for a review), which may have obscured other significant correlations from being manifested. Third, there is growing evidence that the relationship between nonsocial cognition and social functioning is stronger for female than male patients with schizophrenia (Mueser, Blanchard, & Bellack, 1995; Penn, Mueser, & Spaulding, 1996). Therefore, after we collapsed the relationship across patients' gender, these studies may not be representative of the relationship of nonsocial cognition and social functioning among specific patient subgroups. Finally, although there is some evidence that nonsocial-cognitive measures also relate to outcome (see Green, 1996, for a review), we excluded them from the table because such measures tend to be interview based and too global.

It is possible that current nonsocial-cognitive measures capture only a part of the total "pool" of cognitive variance avail-

Table 1
Summary of Studies That Assess the Relationship Between Nonsocial-Cognitive Functioning and Social Competence

Study	Nonsocial-cognitive measures	Social functioning measures	Highest variance accounted for (cognitive variable)
Bellack et al. (1994)	IQ, WMS–R: digits forward, backward, and logical memory	RPT	16%[a] (WMS: logical memory)
Bowen et al. (1994)	CPT, SPAN, DSDT	PRT	36%[a] (SPAN)
Corrigan & Toomey (1995)	DS–CPT, RAVLT, DSDT, WCST	RPT	12%[a] (RAVLT–Recognition)
Dickerson et al. (1991)	LNNB, IQ	Group activities score	19%[a] (entire battery [median correlation])
Lysaker et al. (1995)	WCST, Slosson Intelligence Test, Gorham Proverbs Test	Interview-based social skills assessment	35%[b] (bsln combination of variables predicting change in social skills)
Mueser et al. (1991)	WMS	RPT	22%[a] WMS (verbal scale) 12%[b] WMS (raw score [bsln with improvement in social skills at posttest]) 26%[b] WMS (MQ [bsln with improvement in social skills at followup])
Penn et al. (1995)	COGLAB	RPT	27%[a] (vigilance)
Penn, Spaulding, et al. (1996)	COGLAB	Ward behavior	26%[a] (WCST)
Spaulding (1978)[b]	Muller–Lyer Illusion, WCST, object-sorting task	Ward behavior	41%[a] (reflects the combination of all variables, including a measure of dogmatism)
Spaulding et al. (in press)	COGLAB	Ward behavior	25%[a] (RT—Time 1) 40%[a] (RT—Time 2) 12%[b] (SE, WCST) 17%[c] (vigilance, WCST)
Wykes et al. (1990)[d]	RT	Social behavior problems	(Variance accounted for in social behavior was not reported) (Participants divided into fast and slow "processors" did not differ in behavioral problems[a])
Wykes et al. (1992)[d]	RT	Social behavior problems	9%[a] (RT)

Note. WMS–R = Wechsler Memory Scale–Revised; RPT = Role-Play Test of Social Problem Solving or Social Skill; CPT = Continuous Performance Test; SPAN = span of apprehension; DSDT = Digit Span Distractibility Test; DS–CPT = Degraded Stimulus, Continuous Performance Test; RAVLT = Rey Auditory Verbal Learning Test; LNNB = Luria–Nebraska Neuropsychological Battery; WMS–MQ = Wechsler Memory Scale–Memory Quotient; WCST = Wisconsin Card Sorting Test; COGLAB = RT, CPT, SPAN, Muller–Lyer Illusion, Size Estimation (SE) Task, and WCST; RT = reaction time; bsln = baseline.
[a] Reflects a cross-sectional correlation between nonsocial-cognitive measures and social functioning. [b] Reflects relationship between nonsocial-cognitive measures as initial assessment with changes in social functioning. [c] Reflects the relationship between changes in nonsocial cognitive functioning with changes in social functioning. [d] Samples comprised individuals with diagnosis of schizophrenia and other chronic psychiatric conditions.

able; as better nonsocial-cognitive measures are developed (or new measures used), the variance accounted for in social functioning will increase. The findings, however, raise the alternative possibility that, after we account for nonsocial-cognitive factors, other variance in social functioning among patients with schizophrenia may still be "unclaimed." As previous work indicates that symptomatology is only weakly related to social competence (Appelo et al., 1992; Bellack, Morrison, Wixted, & Mueser, 1990) and demographic variables do not appear to moderate the associations among cognitive and social competence measures (e.g., Penn et al., 1995), then factors in addition to nonsocial-cognitive processes may contribute to social functioning. We present data in the ensuing sections, suggesting that performance on social-cognitive measures may account for this unclaimed variance.

A second limitation concerns the division between theoretical models applied to schizophrenia versus nonclinical controls (i.e., individuals without a major psychiatric disorder). Theoretical constructs in schizophrenia are dominated by biologically oriented, reductionistic models, which explain disordered behavior and symptoms in terms of processes (relatively) void of social context. For example, various researchers have applied cognitive theories such as "pigeon-holing" (Broadbent, 1977), automatic versus controlled information-processing (Posner, 1982; Schneider & Shiffrin, 1977) models of conditioning (e.g., latent inhibition), language production, and probabilistic reasoning to hallucinations and delusions (Frith, 1987; Gray, Feldon, Rawlins, Hemsley, & Smith, 1991; Hemsley, 1977; Hoffman, 1986; Huq et al., 1988; Knight, 1984; Magaro, 1984). These models provide important frameworks for how anomalies in nonsocial cognition produce, or at least significantly contribute to, some of the symptoms of schizophrenia. However, they omit the social-cognitive processes often discussed in normative samples (e.g., social schemata; Fiske & Taylor, 1991).

It is widely accepted that, in controls, important interpersonal phenomena cannot be understood solely by reference to nonsocial information-processing variables. For example, the construct of prejudice is not only a function of a nonsocial-cognitive process, such as to categorize individuals into homogeneous out-groups (i.e., more of a purely cognitive perspective; e.g.,

Linville & Jones, 1980; and Park, Ryan, & Judd, 1992), but also a function of competition between groups (Sherif, Harvey, White, Hood, & Sherif, 1961); economic tension (Hovland & Sears, 1940); desire to maintain a positive self-evaluation (i.e., compared with others; Tajfel & Turner, 1986); cultural–historical factors (Dovidio & Gaertner, 1986; Duckitt, 1992); schema content and complexity (Devine, 1989; Judd & Park, 1988); affective factors (Stangor, Sullivan, & Ford, 1991); and the observation of one's attitudes and behaviors with a possible retaliation result (Crosby, Bromley, & Saxe, 1980). Therefore, a myriad of factors other than nonsocial cognition may be required to account for social behavior. Thus, it is plausible to assume that similar contextual and interpersonal factors may affect the behavioral and clinical manifestations of schizophrenia. In the following section, we present a brief historical overview of the investigation of social cognition in schizophrenia.

Historical Perspective on Social Cognition in Schizophrenia

Research on the performance of patients with schizophrenia using socially oriented stimuli dates back to the work conducted on size estimation tasks (see Cromwell & Spaulding, 1978; Neale, Held, & Cromwell, 1969; and M. Strauss, Foureman, & Parwatikur, 1974, for reviews), perception of stressful pictures (see Buss & Lang, 1965, for a review), social concepts (Whiteman, 1954), and reasoning tasks (Gillis, 1969). Findings in these areas are not straightforward. Specifically, size estimation is correlated with patient chronicity and is a function of stimulus thematic content (M. Strauss et al., 1974). For example, threatening versus neutral content engenders the greatest size estimate deviation in patients with good, rather than poor, premorbid adjustment (Neale et al., 1969). Although affective stimuli tend to produce poorer performance than neutral stimuli, the overly broad definition of affective stimuli precludes identification of the key stimulus properties that contribute to impaired performance (see Buss & Lang, 1965, for a discussion). In other words, affective stimuli can be of both a nonsocial (e.g., a loud noise) and social nature (e.g., a picture of parents and children interacting). Efforts to equate cognitive and social measures psychometrically have resulted in greater deficits on the socially relevant measures in some cases (e.g., Gillis, 1969; and Whiteman, 1954) and comparable task performance in others (e.g., Chapman & Chapman, 1975). Therefore, conclusions from these early findings are unclear because studies lack consensus on operational definitions of constructs and findings are rarely replicated with the same measures.

Early researchers also investigated how patients with schizophrenia represent social information (e.g., personal constructs) and the role of the experimental context on task performance (e.g., the "censure-deficit" model; Rodnick & Garmezy, 1957). Findings with a personal construct paradigm or close variations indicate that, relative to psychiatric and nonpsychiatric controls, the self structures of patients with schizophrenia tend to be less complex and elaborated (Gara, Rosenberg, & Mueller, 1989; Robey, Cohen, & Gara, 1989), which may be indicative of the "loosening" of associations and thought disorder (Bannister, Fransella, & Agnew, 1971). However, observations that such personal constructs were not consistent across repeated assessments led to additional hypotheses, namely, constructs are applied inconsistently by patients with schizophrenia (Livesay, 1984), the variability in personal constructs may reflect phasic changes in the disorder (Cromwell, 1984), the reliability of the repertory grid for use with patients having schizophrenia is poor (Dingemans, 1980), or all of the above. These issues cloud the interpretability of findings on self structure in patients with schizophrenia.

Researchers in the late 1950s to early 1970s examined the effects of the experimental context on task performance (e.g., auditory discrimination). They manipulated context in various ways, including the presence versus absence of the experimenter (Gelburd & Anker, 1970) and the use of censure, both of a nonsocial (e.g., a tone; e.g., Rodnick & Garmezy, 1957) and social nature (e.g., verbal feedback; Atkinson & Robinson, 1961; Cavanaugh, Cohen, & Lang, 1960). Although Rodnick and Garmezy's seminal work suggests that, relative to controls, patients with schizophrenia show a differential performance deficit after censure, later research did not support this hypothesis (e.g., Wagener & Hartsough, 1974); censure, both social and nonsocial, tends to improve task performance of patients with schizophrenia (Atkinson & Robinson, 1961; Cavanaugh et al., 1960; Frieswyk, 1977; Van Dyke & Routh, 1973). Thus, these findings indicate the experimental context does affect the performance of patients with schizophrenia on various cognitive tasks, although the issue of a differential impact (relative to controls) was not consistently supported.

Although the studies reviewed in this section lay the groundwork for research described below, it is possible to question whether they represent true tests of the role of social cognition in schizophrenia. The studies are fraught with a number of methodological and conceptual problems, including the overly broad use of the term *affective stimuli,* the minimal social component in the manipulation of social context (e.g., "that was bad, too slow"; Cavanaugh et al., 1960), and the questionable reliability of early techniques to assess self structure in patients with schizophrenia. Such problems may reflect that research in social cognition and severe adult psychopathology have been conducted in relative isolation from each other. Thus, if psychopathologists study social cognition in schizophrenia, it will be necessary to apply models and measures of social cognition that have been better validated with controls. For example, future analysis of self structure in schizophrenia should look toward well-validated methodologies from the social-cognition literature (e.g., priming and the Stroop paradigm; see Higgins & Bargh, 1987, for a review), especially those used with clinical populations (e.g., depression; Segal, 1988). The studies described in the following sections, which have focused on social-cognitive constructs such as social perception and attribution style, represent a step in this direction.

Social Dysfunction and Social Cognition in Schizophrenia

The study of social cognition in patients with schizophrenia is especially important to understand social dysfunction during the development and maintenance of the disorder. Impairments in social functioning are among the hallmarks of schizophrenia (*DSM–IV;* APA, 1994) and are thought to represent a domain

independent of positive and negative symptoms (Lenzenweger & Dworkin, 1996; Lenzenweger, Dworkin, & Wethington, 1991; J. S. Strauss, Carpenter, & Bartko, 1974). These deficits, although present in other clinical groups (e.g., bipolar disorder), are most pronounced in patients with schizophrenia (Bellack et al., 1990) and are evident in children and adolescents who later develop schizophrenia (Dworkin, et al., 1993; Hans, Marcus, Henson, Auerbach, & Mirsky, 1992; Walker, 1994). Such deficits likely contribute to premorbid social competence, perhaps the strongest predictor of outcome in patients with schizophrenia (Mueser, Bellack, Morrison, & Wixted, 1990; J. S. Strauss & Carpenter, 1977; Tien & Eaton, 1992). Moreover, social competence at discharge is inversely associated with relapse rate (Johnstone, MacMillan, Frith, Benn, & Crow, 1990; Perlick, Stastny, Mattis, & Teresi, 1992).

There is growing evidence that impairments in the social functioning of patients with schizophrenia are related to deficits in social cognition. Deficits in affect perception and social-cognitive problem solving, often observed in patients with schizophrenia (see Bellack, Morrison, & Mueser, 1989; and Morrison, Bellack, & Mueser, 1988, for reviews), are associated with social competence (Mueser et al., 1996; Reed, Penn, Spaulding, & Sullivan, 1994; Spaulding, Weiler, & Penn, 1990) and postdischarge social functioning (Sullivan, Marder, Liberman, Donahoe, & Mintz, 1990). Specifically, Mueser et al. (1996) demonstrated that performance on tasks of facial affect perception were associated with behavior of patients on the ward. Social-cognitive problem-solving skills were related to patients' ward functioning, in one study (i.e., Reed et al., 1994), and, in another study, differentiated patients who relapsed from those who did not (i.e., Sullivan et al., 1990). Perhaps the most compelling evidence for the role of social cognition in the social functioning of patients with schizophrenia is provided in two studies (i.e., Corrigan & Toomey, 1995; and Penn, Spaulding, et al., 1996).

Corrigan and Toomey (1995) compared the relationships between measures of nonsocial information processing (i.e., vigilance, verbal recall and recognition, and conceptual flexibility) and social cognition (i.e., social cue perception) with interpersonal problem-solving skills. Their results reveal that social cue perception was significantly associated with interpersonal problem reception (to identify the problem), processing (to come up with a solution), and sending skills (to role-play the solution), even after a Bonferroni correction was used. Only 3 of 15 nonsocial information-processing variables were significantly associated with interpersonal problem-solving skills, with none remaining significant at the Bonferroni-corrected alpha level. Furthermore, the correlation coefficients representing the relationships between social cue perception and interpersonal problem solving are significantly higher than the corresponding correlation coefficients between nonsocial information-processing and interpersonal problem solving for 7 of 15 comparisons. Thus, Corrigan and Toomey demonstrated that a measure of social cognition has a more consistent relationship with interpersonal problem-solving skills than measures of nonsocial information processing.

Penn, Spaulding, et al. (1996) extended the findings of Corrigan and Toomey (1995) in two ways. First, a battery of social-cognitive tasks was used, including measures of facial affect perception, empathy, and social-script sequencing (i.e., analogous to picture arrangement in the Wechsler Adult Intelligence Scale–Revised; Wechsler, 1981). The latter task is of interest because possession of culturally normative behavioral scripts (Schank & Abelson, 1977) is important for everyday social interactions to be smooth and effective. Second, social functioning was indexed as naturally occurring behavior on the ward. As in the Corrigan and Toomey study, Penn et al. included a battery of nonsocial information-processing tasks (i.e., reaction time, vigilance, and conceptual flexibility). The results largely paralleled Corrigan and Toomey's; omnibus tests of the correlation matrices reveal that only the social-cognitive tasks are significantly related to ward behavior. Therefore, in two studies, different in patient sample characteristics, social-cognitive measures, and indices of social functioning, measures of social cognition have a stronger relationship with social behavior than nonsocial information-processing tasks.

One caveat should be noted about the findings above. Because the information-processing and social-cognitive tasks were not matched for reliability or discriminability (Chapman & Chapman, 1973, 1978), the possibility cannot be ruled out that the correlational patterns reflect the differential psychometric sensitivity of the social-cognitive measures to global impairment rather than a genuinely stronger relationship with social behavior. However, as pointed out by Knight (1984), to control for task difficulty and reliability may confound the underlying processes: "To match on discrimination difficulty is to unmatch on process" (p. 124). Thus, to equate social-cognitive and nonsocial-cognitive tasks could be inappropriate because the processing of social information may be inherently more difficult than the processing of nonsocial-cognitive information. This could be due to the affect or personal relevance associated with social-cognitive stimuli. An analogy might be the following: predicting a minor league baseball player's future batting average in the major leagues based on his ability to hit fastballs and curveballs. If we assume that curveballs are more difficult to hit, then to make the fastball–curveball task equivalent by including easier curveballs and more difficult fastballs may not be ecologically valid, unless such a pattern is also found in the major leagues. In essence, to equate these types of pitches, we would "take" the curve out of curveballs.

Given this problem, what can be done to ensure that differences in performance on cognitive and social-cognitive measures represent construct rather than task characteristic differences? Knight (1984) offered some solutions that may be relevant to the performance of patients with schizophrenia on nonsocial-cognitive versus social-cognitive tasks. One strategy is to identify social-cognitive tasks on which patients with schizophrenia perform better than controls. For example, if it is hypothesized that patients with paranoid schizophrenia have a self-schema selectively sensitive to threat (e.g., Bentall, Kinderman, et al., 1994; Trower & Chadwick, 1995), then patients with schizophrenia might perform faster than controls on a task that requires making "me" or "not me" judgments on words that connote threat compared with nonthreat (see Markus, 1977, for a description of this procedure). Bentall and colleagues' work on preferential attention to and recall of threatening propositions by patients with persecutory delusions (described below) is

consistent with this approach (Bentall & Kaney, 1989; Kaney, Wolfenden, Dewey, & Bentall, 1992).

A second strategy is to identify experimental conditions that will produce a relative rather than absolute performance superiority by patients with schizophrenia. For example, it might be possible to identify experimental conditions that produce decrements in task performance of controls but that do not affect the performance of patients with schizophrenia. A likely situation is where intact processes in controls interfere with task performance. For example, in nonclinical samples, dispositional judgments about a target individual are a function of both stimulus input (e.g., the target individual's facial expressions and behavioral cues) and contextual factors (e.g., the social situation in which the behavior is embedded; Trope, 1986; Trope, Cohen, & Maoz, 1988). Trope and colleagues predicted that, as stimulus input becomes more ambiguous, there is greater reliance on situational or contextual factors. Thus, in an experimental condition where faces are ambiguous with respect to emotional expression, the presentation of erroneous or irrelevant situational information may lead to more "incorrect" dispositional judgments for controls, relative to patients with schizophrenia. A similar pattern may or may not be predicted for nonsocial stimuli, depending on the type of stimulus and the hypothesized processes involved. Therefore, besides matching nonsocial-cognitive and social-cognitive tasks for reliability and difficulty level, other strategies may be used to compare task performances of patients with schizophrenia on nonsocial-cognitive and social-cognitive measures.

Task Performance on Social-Cognitive Versus Nonsocial-Cognitive Measures

The issue of differential performance of patients with schizophrenia on social-cognitive versus nonsocial-cognitive tasks can be broken down into three areas of research: affect perception versus general perception, social versus general knowledge tasks, and biases in social information processing. The results of studies in which researchers compare the performance of patients having schizophrenia with controls on affect perception and general perception tasks (i.e., control tasks) are summarized in Table 2 (Archer, Hay, & Young, 1992; Bellack, Blanchard, & Mueser, 1996; Feinberg, Rifkin, Schaffer, & Walker, 1986; Gessler, Cutting, Frith, & Weinman, 1989; Heimberg, Gur, Erwin, Shtasel, & Gur, 1992; Kerr & Neale, 1993; Mueser et al., 1996; Novic, Luchins, & Perline, 1984; Schneider, Gur, Gur, & Shtasel, 1995; Walker, McGuire, & Bettes, 1984). At first glance, it would appear that, in most of the studies (8 out of 10), patients with schizophrenia performed lower than controls on both affect and general perception tasks. Such findings are consistent with a "generalized" performance deficit rather than a specific deficit in affect perception. However, in two of the studies (Feinberg et al., 1986; Novic et al., 1984), support for the generalized deficit was less impressive; a "differential" deficit in affect perception was found for patients having schizophrenia compared with patients having depression in Feinberg et al. (1986), whereas the generalized deficit reported by Novic et al. was manifested only after the researchers controlled for initial performance on the control task. Furthermore, Novic et al. reported that when unmatched tasks were used, patients with schizophrenia demonstrated a differential deficit in affect recognition.

The findings described earlier do not present a clear picture regarding differential performance deficits on affect perception versus general perception tasks. A factor that may have contributed to this obfuscation concerns the control measure of general perception. In most cases, the control task was either the Benton facial recognition test (Benton, Hamsher, Varney, & Spreen, 1983), an age-discrimination task, or another task that requires perception of facial displays (for exceptions, see Feinberg et al., 1986; and Kline, Smith, & Ellis, 1992, in a study of facial affect recognition memory). These control measures closely resemble the affect identification and discrimination tasks in format and content; both sets of measures use faces as the target stimuli, with stimuli representing static rather than dynamic displays. In other words, these tasks control for the affective rather than social quality of stimuli. It remains to be seen whether the observed generalized deficits hold after more social-neutral control tasks are used (Dworkin, 1992; Hellewell, Connell, & Deakin, 1994).

The representation of social knowledge relative to practical knowledge was investigated in a series of studies by Cutting and Murphy (1988, 1990). Social knowledge was assessed with such items as, "What do you think would be the most sensible thing to say if you came across two strangers having a fight in the street?" (Cutting & Murphy, 1990, p. 357). An example of a practical knowledge question is, "Why is it unsafe to drink tap water in some countries?" (p. 358). Findings reveal that patients with schizophrenia showed the greatest impairments, relative to clinical controls, on the social knowledge test. Cutting and Murphy concluded that the thematic content of the social knowledge test may be particularly responsible for the impaired performance on this task of patients with schizophrenia.

A somewhat different approach to understanding social-cognitive versus nonsocial-cognitive functioning in schizophrenia has been undertaken in studies in which researchers investigated performance biases (Bentall & Kaney, 1989; Bentall, Kaney, & Bowen-Jones, 1995; Brennan & Hemsley, 1984; Kaney et al., 1992). Specifically, using a variation of the Stroop color-naming task, Bentall and Kaney found that patients with prominent persecutory delusions demonstrated slowed color naming to threat- but not depression-related words, a finding that was replicated by Fear, Sharp, and Healy (1996). In a related study, Brennan and Hemsley found that patients with paranoid schizophrenia showed a greater tendency to make illusory correlations to paired words than patients with nonparanoid schizophrenia and controls, with group differences strongest for words related to paranoia (e.g., *spy* or *secret*). Finally, in studies of memory bias, patients with persecutory delusions showed a tendency to preferentially recall threatening propositions (Kaney et al., 1992) and words (Bentall et al., 1995).

From a deficit-model perspective, these biases are inconsistent with the traditional psychopathology findings of impairment by patients with schizophrenia on most laboratory tasks because they are specific to information with particular content. However, they are congruent with findings obtained from patients with other psychological disorders (e.g., social phobia) in which biases result from matches between stimulus content and underlying social schema. Thus, these studies suggest that the content

Table 2
Results of Studies That Compare the Performance of Patients Having Schizophrenia With Controls on Affect and General Perception Measures

Study	Tasks psychometrically matched?[a]	Results
Archer et al. (1992)	No	Face recognition, face expression, unfamiliar face matching task: C > S (chronic patients)
Bellack et al. (1996)	Yes (used two of the tasks matched by Kerr & Neale, 1993)	Facial affect identification and discrimination tasks: C = S (acutely ill patients) = BP Videotape Affect Perception Test: Angry scenes (all conditions): C = S (acute) = BP Sad scenes (video only): C = S (acute) = BP Sad scenes (audio + video): C > S (acute) = BP Happy scenes (video only): C > S (acute) = BP Happy scenes (audio + video): C = S (acute) = BP Benton Facial Recognition Test and Speech Sounds Test: C > S (acute) = BP
Feinberg et al. (1986)	No	Emotion matching task: C = D > S Emotion labeling task: C > D > S (difference between D and S approached significance) Identity matching task: C > S (S = D)
Gessler et al. (1989)	Yes (difficulty level and distribution of items)	Emotion discrimination task: C = D = S (remitted) = S (chronic) > S (acute) Age discrimination task: C = S (remitted) > S (chronic) = D = S (acute)
Heimberg et al. (1992)	Yes (reliability only)	Emotion discrimination task and age discrimination task: Compared with nonpsychiatric and depressed controls, patients with schizophrenia (mostly neuroleptic naive) showed a greater performance deficit on the emotion discrimination task compared with the age discrimination task
Kerr & Neale (1993)	Yes (difficulty only)	Facial and Vocal Emotion Identification and Discrimination Tests: C > S (unmedicated patients); Benton Facial Recognition and Speech Sounds Recognition Tests: C > S
Mueser et al. (1996)	Yes (used the same tasks matched by Kerr & Neale, 1993)	Facial Emotion Identification and Discrimination Tests: C > S; Benton Facial Recognition Test: C > S
Novic et al. (1984)	Yes (reliability and discriminability)	Facial Affect Recognition Test: C = S (chronic; after covaried out performance on the facial recognition task); C > S (without covarying); Facial Recognition Test: C = S
Schneider et al. (1995)	Yes (reliability only)	Emotion discrimination task: C > S Age discrimination task: C > S
Walker et al. (1984)	Yes (reliability and discriminative power)	Emotion labeling with multiple choice: C > A = S Emotion discrimination: C = A > S Facial discrimination: C = A = S[b]

Note. A = affective disorder; BP = bipolar disorder; C = control; D = patients with depression; S = patients with schizophrenia.
[a] Represents the development of tasks that were matched in terms of difficulty level and reliability. [b] Post hoc tests conducted at the .10 alpha level.

of stimuli affects processing and retrieval of novel information by patients with schizophrenia.

Overall, the evidence reviewed in this section lends some support to our thesis that differential performance is demonstrated on social-cognitive versus nonsocial-cognitive tasks. However, it is still unclear why differential performance occurs on some tasks (e.g., Stroop and recall tasks) and not others (i.e., facial affect recognition). The studies above differ on many factors, such as sample characteristics, measures, and constructs evaluated, so it is presently not possible to determine what other variables, if any, account for the inconsistent results.

Social Cognition and Symptomatology

Social Cognition and Content of Positive Symptoms

Both clinical observation and empirical investigation indicate that the prominent positive symptoms of schizophrenia, hallucinations and delusions, represent fairly well-organized subjective experiences that revolve around consistent themes (see Bentall, 1990; Butler & Braff, 1991; Lowe, 1973; and Winters & Neale, 1983, for reviews). Specifically, auditory hallucinations typically take the form of "voices" that direct, admonish, or comment on the patient's behavior (Chadwick & Birchwood, 1994; Chaturvedi & Sinha, 1990; Larkin, 1979; L. J. Miller, O'Connor, & DePasquale, 1993; Rogers, Gillis, Turner, & Frise-Smith, 1990). For example, Chaturvedi and Sinha reported that auditory hallucinations were the most frequent type of hallucination found in an Indian sample, with the most typical hallucination characteristics being voices conversing and running commentaries on behavior.

In a study of U. S. patients, L. J. Miller et al. (1993) found that negative themes were commonly reported but that some patients nonetheless maintained a positive attitude toward their voices; the voices were regarded as important elements in their

social world. Chadwick and Birchwood (1994) similarly observed that some patients believed their voices to be malevolent, whereas others believed their voices to be benevolent. Voices believed to be benevolent were courted, whereas those believed to be malevolent were feared and resisted. With respect to delusions, consistent interpersonal themes include social comparison (Heilbrun, Diller, & Dodson, 1986), plots and persecution from others (Winokur, 1977), peer pressures (Forgus & DeWolfe, 1974), "grandiosity" (Junginger, Barker, & Coe, 1992), and other existential themes that reflect concerns about the patient's place in the social universe (Bentall, 1994; Roberts, 1991a).

Social-Cognitive Models of Schizophrenia Symptomatology

Over the past 6 years, two broad social-cognitive accounts of schizophrenia symptomatology have been proposed by Bentall (1990, 1994; Bentall, Kinderman, et al., 1994) and Frith (1992, 1994). Both researchers have argued that attempts should be made to identify social-cognitive abnormalities associated with particular symptom classes, on the assumption that the diverse range of symptoms exhibited by patients with schizophrenia are unlikely to be explicable in terms of common nonsocial-cognitive abnormalities. Bentall's work has focused on hallucinations and delusions. In a review of the research literature on the former, he has argued that "hallucinators make hasty and overconfident judgments about the source of their perceptions and have a bias toward inappropriately attributing their perceptions to an external source" (Bentall, 1990, p. 90). On this view, patients with hallucinations mistake self-generated experiences—for example, inner speech—for sources external or alien to themselves. These misattributions partially reflect patients' beliefs about the likelihood that such experiences will be real as opposed to imaginary. Consistent with this account, experimental studies have shown that the hallucinations of patients with schizophrenia can be influenced by one manipulating their beliefs and expectations (Haddock, Slade, & Bentall, 1995; Mintz & Alpert, 1972; Young, Bentall, Slade, & Dewey, 1987).

In a series of studies that used social reasoning or "attributional" tasks, patients with persecutory delusions were shown to have an abnormal tendency to attribute negative events (e.g., failing an examination) to external causes (i.e., other people or circumstances) and positive events (e.g., getting a promotion) to internal causes (i.e., the self; Candido & Romney, 1990; Fear et al., 1996; Kaney & Bentall, 1989, 1992; Lyon, Kaney, & Bentall, 1994). A similar attributional pattern has been found in outpatients with schizophrenia (Silverman & Peterson, 1993). This response style seems to be an extreme form of the self-serving bias observed in participants without psychopathology (D. T. Miller & Ross, 1975) and is the opposite pattern to that found in patients with depression, who show an exaggerated tendency to attribute negative events to internal causes and positive events to external causes (Sweeney, Anderson, & Bailey, 1986). However, Lyon et al. showed that the exaggerated self-serving attributional style of patients with delusions is evident only on explicit measures of attributional style; on an implicit measure (i.e., disguised as a memory task), patients made attributional responses that were similar to those of patients with depression (i.e., to explain negative events in terms of internal causes and positive events in terms of external causes).

Bentall, Kindermann, et al. (1994) have suggested that the explicit attributional responses of patients with delusions reflect a strategic attempt to reduce discrepancies between self-representations and self-ideals but that their implicit attributional responses reflect a latent negative self-schema. Consistent with this account, patients with delusions showed slowed color naming to self-esteem-related words on a Stroop task (Kinderman, 1994), but, on an explicit measure of self-representations, Kinderman and Bentall (1996) reported high consistency between self-representations and self-ideals. Therefore, the persecutory delusions of patients with schizophrenia may serve an important function as the protection and support of the patient's self-esteem in a potentially hostile (or threatening) social environment.

Drawing on diverse research findings from higher primates, autism, and neurophysiology, Frith (1992, 1994) has argued that in patients with schizophrenia impairments in "willed action" (i.e., not able to spontaneously generate behavior in the absence of external cues or to suppress inappropriate behavior), and the ability to monitor these actions and the intentions of others, are the result of a general impairment in "theory of mind"—the ability to represent mental states. For example, an inability to represent the intentions of others may contribute to persecutory delusions in patients, whereas impairments in patients' ability to consider others' perspectives during conversations (e.g., by omitting the appropriate speech referents) may underlie the patient's incoherent speech. Frith also applied impaired theory of mind to the negative features of schizophrenia (e.g., social withdrawal); a complete absence of "mentalizing," rather than impaired functioning, would render a patient completely disinterested in social contact and impoverished in speech.

Due to its relatively recent formulation, direct empirical support for Frith's (1992, 1994) theory of mind hypothesis is only available from two studies. Corcoran, Mercer, and Frith (1995) compared the performance of 55 patients having schizophrenia with two groups of psychiatric and nonpsychiatric controls. Performance of the groups was assessed on a measure developed by Corcoran et al., coined "the hinting task." This comprised 10 short scenarios that depict an interaction between two characters. At the end of the scenario, one of the characters drops a very obvious hint to the other (e.g., "I want to wear that blue shirt, but it's creased"; p. 12). The patient was instructed to indicate what he or she thought the character really meant to say, what he or she was hinting at. Because the two control groups did not differ in performance on the hinting task, they were combined into one group. Analyses reveal that the patients with schizophrenia performed significantly worse than the control group on the hinting task. Furthermore, analysis of patient–symptom subgroups showed that patients with negative features, passivity symptoms, and paranoid delusions performed worse than the control groups and patients with schizophrenia but in remission.

In a second study (Frith & Corcoran, 1996), samples of patients with schizophrenia and controls (patients with anxiety and other participants) were read simple stories, accompanied by cartoons that depicted social interactions. After each story,

participants were asked one memory question and one question that required them to infer the mental state of one of the characters. Analyses indicate that, compared with the combined control sample, patients with paranoia and a subgroup of patients with positive and negative behavioral signs (e.g., poverty of speech and incongruity of affect) performed more poorly on the theory of mind questions. The findings from these two studies should be interpreted cautiously, however, because of the small sample sizes in some of the symptom groups (e.g., passivity symptoms, $n = 7$; Corcoran et al., 1995). Furthermore, they need to be replicated across different tasks, especially those with well-established psychometric properties.

Most of the research described earlier was on the social-cognitive mechanisms related to positive symptoms. Less attention has been placed on the role of social cognition in negative symptoms (e.g., anhedonia, alogia, and flat affect). This may be an important omission because negative symptoms tend to have a more consistent relationship with social competence than positive symptoms (see Mueser & Bellack, in press, for a review). One model that espouses a psychosocial perspective on negative symptoms has been described by J. S. Strauss, Rakfeldt, Harding, and Lieberman (1989). J. S. Strauss et al. hypothesized that negative symptoms may arise from a number of sources, including the fear of stressful interpersonal situations, concerns about behavioral incompetence (i.e., to avoid interactions with others because of an inability to suppress inappropriate behavior), and avoidance of stigmatization, among others.

J. S. Strauss et al.'s (1989) psychosocial model of negative symptoms has received little direct empirical support. Some indirect support is garnered from findings that indicate that patients with schizophrenia reported using withdrawal as a strategy to cope with being stigmatized (Link, Cullen, Struening, Shrout, & Dohrenwend, 1989) and that negative symptoms vary as a function of the amount and quality of social interaction on the ward (Wing, 1978). The hypothesis that fear of stressful situations is associated with negative symptoms was tested by Penn, Hope, Spaulding, and Kucera (1994), who assessed the relationship between various indices of social anxiety (i.e., self-report, information processes, and behavioral social anxiety during a role-play) and symptomatology. Evidence in support of J. S. Strauss et al.'s model was mixed; self-reported social anxiety (on a social anxiety measure) was associated with positive symptoms, whereas behavioral indices of social anxiety during a role-play were associated with negative symptoms. A similar relationship between social anxiety and the positive symptoms (i.e., paranoia) was reported by Smari, Stefansson, and Thorgilsson (1994). So future work is needed to empirically assess J. S. Strauss et al.'s psychosocial model of negative symptoms.

In this section, we have reviewed findings that indicate that symptomatology in schizophrenia, predominantly positive symptoms, may reflect distortions in social-cognitive processes. However, given that schizophrenia is a disorder punctuated by periods of symptom exacerbation and followed by relative remission, we raise the question about the integrity of social-cognitive processes throughout the course of the disorder. Unlike the voluminous work on vulnerability and episodic cognitive markers in schizophrenia (Nuechterlein & Dawson, 1984b), there has been little research on phasic changes in social cognition. Because nonsocial-cognitive and social-cognitive processes are related to one another (e.g., Penn, Spaulding, et al., 1996), a plausible hypothesis is that similar trait and state markers exist in the social-cognitive domain.

Social Cognition and Phasic Changes in Schizophrenia

There is preliminary evidence that social-cognitive impairments are stable across clinical states. Specifically, Gabel and Wolwer (1992) showed that relative to patients with depression and nonclinical controls, those with schizophrenia performed more poorly on tests of affect recognition 1 month apart, even after a significant reduction in symptomatology across assessments. Similar findings were reported in a study of social-cognitive problem solving (SCPS); despite reduction in symptomatology over a 3-month period, the SCPS of patients with schizophrenia was still impaired relative to patients with depression and nonclinical controls (Penn et al., 1993). These findings, which suggest the presence of stable, vulnerability-linked social-cognitive deficits, need to be replicated before confident conclusions can be drawn.

In lieu of a substantial body of literature on the longitudinal course of social-cognitive processes in schizophrenia, evidence from disparate sources must be garnered to shed light on clinical changes in social cognition. One possible source is to look toward findings on other disorders with known social-cognitive impairment, such as depression. For example, cognitive models of depression posit that, during symptom remission, negative schema remain available to influence information processing (Beck, 1987). In support of Beck's model, a number of studies have found evidence of depressive information processing (e.g., greater recall of negative words) in patients with former depression, compared with those never having depression (Hedlund & Rude, 1995; Ingram, Bernet, & McLaughlin, 1994). These findings have relevance for schizophrenia because negative schemata have been hypothesized to underlie some paranoid delusions (e.g., Bentall, Kinderman, et al., 1994). Along these lines, note that dysphoric symptoms often precede a schizophrenia relapse (Heinrichs & Carpenter, 1985; Herz & Melville, 1980); perhaps this occurs through activation of latent negative schemata. Thus, patients with schizophrenia who are no longer actively symptomatic may still be prone to subtle biases in social information processes, which render them vulnerable to future relapses.

A second source to draw on is cross-sectional work on the social-cognitive performance of subgroups of patients with schizophrenia in different stages of the illness (i.e., acute vs. remitted). For example, in the area of affect perception, patients in the acute phase tend to perform lower than those whose symptoms are in remission (Cutting, 1981; Gessler et al., 1989). In a study of situational social perception (i.e., perception of the components of social events), relatively remitted outpatients with schizophrenia demonstrated higher performance than symptomatic inpatients, although their performance was still impaired relative to nonclinical controls (Corrigan, Garman, & Nelson, in press). Therefore, deficits in social perception may represent vulnerability-linked impairments, which are augmented during symptom exacerbation.

Much work is needed to determine the role of social cognition across the clinical course of schizophrenia. It is clear that, for a

theory of social cognition to be comprehensive, it must consider the variability of the disorder. The research discussed earlier may, we hope, serve as the basis for future work in this area.

Speculations on Social Cognition in Schizophrenia

Developmental Implications

The development of schizophrenia is often preceded, at least in high-risk samples, by cognitive and social competence impairments that are manifested in early childhood (see Asarnow, 1988, for a review). In fact, the general pattern is one of subtle attentional–vigilance and neuromotor deficits before Age 9, followed by increasingly greater interpersonal difficulties through adolescence (e.g., poor affective control, withdrawn, or having unstable interpersonal relationships), culminating in symptom onset in adulthood (Asarnow, 1988). Thus, the evidence suggests that cognitive–attentional impairments in childhood predict later psychopathology (Walker, 1994).

There is some evidence that impairments in social cognition may predate psychotic illness. Specifically, in the Israeli High-Risk Study (Frenkel, Kugelmass, Nathan, & Ingraham, 1995), it was found that an external locus of control in adolescence was predictive of poor mental health in adulthood. This observation may be particularly significant, given that locus of control is a construct that is somewhat similar to attributional style, which, as noted earlier, is prone to particular biases among patients having schizophrenia with prominent delusions (e.g., Bentall, Kinderman, et al., 1994). Indirect evidence for the role of social cognition in the development of schizophrenia is garnered from the work of Cornblatt and colleagues (Cornblatt & Keilp, 1994; Cornblatt et al., 1992). Based on prospective, longitudinal data, Cornblatt and colleagues hypothesized that disruption of social information processing by a chronic attention deficit may be the strongest predictor of social impairment among high-risk individuals. Specifically, among clinically unaffected adult participants at risk for schizophrenia, an attentional deviance index (ADI) in childhood predicted social insensitivity and indifference (i.e., as measured by the personality disorder examination) in adulthood. This pattern of correlations was not found for either controls or participants at high risk for affective disorders. Furthermore, in a different sample of high-risk participants, the correlation between ADI (at Age 9) and social competence at Ages 9, 11, and 15 was only significant at the oldest age. Thus, nonsocial-cognitive processes do not relate to social functioning until the child reaches adolescence, when the variety and complexity of social input are likely to increase.

Although the foregoing gives some "primacy" to nonsocial-cognitive impairments, it is consistent with the aforementioned building-block view of social cognition: Nonsocial cognition represents a necessary but not sufficient condition for effective social cognition. Furthermore, it suggests that, as the social environment demands more specialized processing (i.e., the processing of information relevant to interpersonal interactions and demands; e.g., Cosmides & Tooby, 1989), faulty development of social cognition compromises the individual's ability to accurately perceive and effectively act on social situations. This sequence of events is compatible with Carter and Flesher's (1995) neurosociological model of schizophrenia development:

> It is our contention that at precisely the point in social development when a variety of processes should take place to facilitate the translation of the individual into the complex world of adult interactions, the requirements on patients to engage in those processes begin to outstrip the cognitive resources available to meet those demands. (p. 218)

At this point, with the exception of the findings of the Israeli High-Risk Study described earlier (Frenkel et al., 1995), data are not available to determine whether deficits in social cognition precede the onset of schizophrenia or result from multiple repeated episodes. As high-risk research incorporates assessment of social cognition into its designs, evidence may accumulate regarding the contribution of social-cognitive impairment to the etiology of schizophrenia. In the remainder of this section, we discuss the hypothesized neural mechanisms that underlie social cognition and attempt to integrate them with the neuropathology of schizophrenia.

Neuropathology Implications

There is some evidence that certain neural areas are implicated in the processing of social information. Specifically, in a review of studies based on animal models and individuals without schizophrenia, Kirkpatrick and Buchanan (1990) concluded that the neural circuit underlying social affiliation comprises the amygdala and prefrontal cortex. For example, bilateral lesions of the amygdala result in reduced levels of social interest and contact in animals and increased passivity, apathy, and flattened affect in humans. Lesions in the prefrontal cortex are associated with a variety of behavioral deficits, including disinhibition, aspontaneity, stereotypy, social withdrawal, and poor social judgment, among others (see Goldberg & Seidman, 1991, for a review). Similar neural mechanisms that underlie social cognition have been proposed by Brothers (1990a, 1990b). Brothers reviewed data that indicate that facial and affect perception and the ability to interact with others are influenced by functioning in the superior temporal sulcus, amygdala, and orbital frontal cortex, respectively. Furthermore, data from primates reveal that neurons found primarily in the inferotemporal cortex are particularly sensitive to the perception of faces (Brothers, 1990a). Thus, these findings are part of a growing consensus that disruption of fronto–temporo/limbic neural circuits are responsible for aspects of social cognition (E. H. Taylor & Cadet, 1989).

The hypothesized neural substrates of social cognition overlap with those implicated in the etiology and maintenance of schizophrenia (see Buchsbaum, 1990; and Gur & Pearlson, 1993, for reviews). For example, Kirkpatrick and Buchanan (1990) argued that the neural circuit that comprises the prefrontal cortex and amygdala underlies enduring negative symptoms in schizophrenia. Although evidence of structural abnormalities in the prefrontal cortex differentiating between patients with deficits (with enduring negative symptoms) from those without deficits has not been consistently supported (Buchanan et al., 1993), patients with negative deficits tend to perform more poorly than patients without negative deficits on neuropsychological tasks sensitive to frontal lobe impairment (Buchanan et al., 1994). Furthermore, a number of neurodevelopmental models suggest that dysfunctions in particular neural circuits, including fronto–limbic connections, contribute to the onset of schizo-

phrenia (Csernansky, Murphy, & Faustman, 1991; Roberts, 1991b; Waddington, 1993; Weinberger, 1987).

Of particular interest to future work is to identify the particular neural system(s) responsible for the execution of behaviors during social-cognitive tasks. For example, would one expect the hypofrontality often associated with perseverative errors on the Wisconsin Card Sorting Test (see Goldberg & Seidman, 1991, for a review) to occur during perseveration on a social problem-solving task? Note, however, that even if similar neural patterns are demonstrated for nonsocial-cognitive and social-cognitive tasks, this does not necessarily weaken our thesis that the processing of social and nonsocial information represents different levels of analyses. As noted by Brothers (1990b), "a neural system which is well-adapted for a particular purpose may under some circumstances process stimuli in other domains, without weakening the claim for a particular primacy adaptation" (p. 42). Thus, similar neural processes may not produce equivalent functions.

Brothers (1990b) and Cosmides and Tooby (1994) have recommended that a more important issue is whether the neural system is well adapted to particular functions. For example, with respect to schizophrenia, therefore, the question, "Are enlarged brain ventricles and cortical atrophy associated with social functioning?" may be reframed as, "Do such structural anomalies place limitations on the abilities of patients with schizophrenia to interact effectively in their social environment?" This particular question becomes even more meaningful in light of Dunbar's (1993) hypothesis (based on findings from primates) that group size in humans, which is predicted by the ratio of neocortical volume to the rest of the brain, is linearly associated with the amount of time that people spend on "social grooming" (e.g., gossip or talking about relationships). If this hypothesis proves empirically viable, then, among patients with schizophrenia who do have cortical atrophy, interventions should not be focused on increasing their social networks (which may overly extend their social-cognitive resources) but on helping these patients manage smaller, less overwhelming networks. Thus, from a functional social-cognitive perspective, neuropathology is more than a tool to identify etiology; it also aids in the identification of treatment-limiting and facilitating factors.

Concluding Comments and Future Directions

In this article, we have argued that the study of social cognition in schizophrenia is not redundant with traditional nonsocial cognition. In support of this assertion, the following evidence has been presented: (a) Nonsocial-cognitive models alone may be limited to explain the social and clinical impairments of schizophrenia, (b) measures of social cognition contribute independent variance to social functioning beyond measures of nonsocial cognition, (c) patients with schizophrenia tend to perform differently on certain social-cognitive versus nonsocial-cognitive tasks, and (d) the content and process of hallucinations and delusions are consistent with distortions in social cognition. These findings suggest that assessment of social cognition can contribute important insights into the psychosocial and clinical sequelae of schizophrenia.

Conclusions drawn from this review suggest a number of directions to pursue in future research. First, to better understand the role of social cognition in schizophrenia, future researchers need to assess the interrelationships among various social-cognitive domains (e.g., attributional style and affect perception). In a preliminary study that addresses this issue in controls, Kinderman, Dunbar, and Bentall (1996) found that poor performance on a theory of mind task was associated with a tendency to make external attributions that are personal (e.g., "John ignored me because he hates me") rather than external attributions that are situational (e.g., "John ignored me because he had a bad day"). In a study conducted with inpatients having schizophrenia, Penn, Spaulding, et al. (1996) found that a measure of affect perception was significantly associated with performance on a social-scripts task. These findings, which indicate shared variance among social-cognitive measures, need to be evaluated in the context of a well-validated social-cognitive battery administered to both patients with schizophrenia and controls. Such an assessment might reveal whether the underlying structure of social cognition differs for schizophrenia versus clinical (and nonclinical) controls.

Relatedly, the specificity of social-cognitive impairments to schizophrenia, rather than psychosis in general, needs to be investigated. Although a number of studies found patients with schizophrenia to be impaired relative to clinical controls on measures of social cognition (e.g., Feinberg et al., 1986), some did not (e.g., Bellack et al., 1996). Furthermore, among those studies that found similar results in patients with common symptoms but different diagnoses (e.g., paranoid schizophrenia and delusional disorder; Fear et al., 1996; Kaney & Bentall, 1989), it still needs to be determined whether the "production" of the symptom, like its presentation, is consistent across groups. For example, in a comparison of the neuropsychological factors underlying the Capgras delusion and those in paranoid schizophrenia, evidence for right-hemisphere dysfunction was found only in the former group (H. D. Ellis et al., 1993). Thus, the social cognition in disorders with overlapping symptomatology, less severe symptomatology (e.g., schizotypal personality disorder or delusional disorder), or both may have dissimilar neurological loci. This underscores the need to integrate social-cognitive theories of schizophrenia with neuropathology investigations. Progress in this area may advance the understanding of the etiology of schizophrenia and the appropriate classification of different manifestations of psychosis.

Second, the representation and use of social information in schizophrenia should be investigated. We suggest a formal study of social schemata in schizophrenia, which includes representation of information about the self, others, and social situations (Corrigan, Wallace, & Green, 1992). Social schemata could have particular importance for clinical interventions because the learning, acquisition, and application of social skills may be impeded if the "templates" for such skills are not present (Penn, 1991). Specifically, to fully use a schema, a number of conditions must be met: It must be available, it must be activated, and the individual must be willing to enact the behaviors the schema calls for.

These conditions above suggest that a patient with schizophrenia may have difficulty learning assertiveness skills in a specific situation (e.g., someone who jumps ahead of her or him on a medication line) if a series of behaviors (i.e., make good eye contact, speak in a clear voice, express needs, etc.) are not

embedded within a general social-cognitive context (e.g., an event schema). Furthermore, the learned response is effective not only to the extent that it is applied to that particular situation but also to the class of situations that require assertive responses (e.g., someone changes the channel you are watching on the television, to tell your physician that your medications are giving you untoward side effects, etc.). Therefore, paradigms that assess schema activation (e.g., priming) in schizophrenia should be explored. Thus, schema availability is a necessary but not sufficient condition for schema activation to occur. Researchers should note, however, that schema activation is complex and dependent on many factors, including the presence of cue stimuli and the goals of the individual (see Bargh, 1989; and McNally, 1995, for discussions of these issues in normative and anxiety disorder samples).

The challenge for researchers and clinicians may be to ascertain whether the patient has correctly identified the situation but chosen not to access certain behaviors (i.e., to not enact the behaviors called for by the schema) or if discrepancies in the problem situation and schema template are significant enough to result in schema inactivation. Furthermore, different intervention strategies may need to be planned for patients without schemata in a given domain, compared with those who have the schema but are unable to access them (due to impairments in social perception). With the former patient, interventions may need to focus on concept formation (e.g., Brenner et al., 1995); whereas for the latter, the focus may be on the developing of social perception skills (Corrigan & Green, 1993).

A third research direction is the assessment of, and improvement in, the ecological validity of current social-cognitive measures. Although facial affect recognition is a widely used task of social perception, in only two studies have researchers assessed its relation with social functioning (i.e., Mueser et al., 1996; Penn, Spaulding, et al., 1996). Both studies, conducted with different patient samples, found that facial affect recognition was most consistently related to neatness and grooming on the ward, suggesting that accurate affect recognition of others has implications for self-perception. However, social perception during an interaction is typically more complex than one merely identifying and discriminating facial affect; context must be taken into consideration, as should verbal and physical cues. Furthermore, an individuals' goals during an interaction is not just to correctly recognize a given emotion. Rather, it is to determine what that emotion means. For example, one may easily identify happiness due to an individual's broad smile. This cue may be misread, however, if the context of the smile is ignored (e.g., the smile is spiteful and portends a harmful comment or action). This type of perception requires reading between the lines, a process impaired in patients with right-hemisphere brain damage (e.g., Brownell, Potter, Bihrle, & Gardner, 1986) but studied in patients with schizophrenia only in the context of Frith's (1992) model of theory of mind impairments. Therefore, assessment of social perception should be broadened to include appreciation of contextual, interpersonal cues (e.g., Corrigan & Green, 1993) and self-perception (Blanchard, Mueser, & Bellack, 1992; Harrow et al., 1989).

Fourth, schizophrenia researchers should pay attention to models of social perception drawn from the social-cognition literature (Bellack et al., 1996; Gilbert, Pelham, & Krull, 1988; Trope, 1986; see Newman & Uleman, 1993, for a review). Specifically, these models posit social perception as a two-stage process. The first stage involves relatively automatic identification or categorization of behavior. Most of the current measures of social perception in schizophrenia research appear to assess social perception at this level of analysis. The second stage, which requires either an inferential or attributional analysis of behavior, is more effortful. At this stage, one determines whether the observed behavior is due to stable dispositions or situational factors. Given the difficulties in controlled information processing associated with schizophrenia (see Braff, 1993, for a review), the second stage may be especially problematic for patients with schizophrenia; they are less likely to modify initial impressions of a target individual or may not "correct" for situational parameters. Therefore, assessment of this second stage may shed further light on the extent of social perceptual impairment in patients with schizophrenia.

A fifth research direction concerns the relationship between affect and social cognition. The influence of affect on social cognition is complex (see Clore, Schwartz, & Conway, 1994, for a review). For example, negative affect can lead to greater self-focused attention (Ingram, 1990) and can reduce attentional resources by one eliciting intrusive thoughts (H. C. Ellis & Ashbrook, 1988). This latter finding has particular relevance to schizophrenia; three studies have shown that, in patients with schizophrenia, thought disorder is exacerbated (relative to controls and patients without thought disorders) when they were asked to discuss affectively laden items (Docherty, Evans, Sledge, Seibyl, & Krystal, 1994; Haddock, Wolfenden, Lowens, Tarrier, & Bentall, 1995; Uziel, Harrow, Waltz, Sands, & Miller, 1995). In a related vein, future investigations should determine whether the often-observed facilitative effects of positive affect on nonsocial-cognitive processes (e.g., creativity; Isen & Daubman, 1984) can be demonstrated on social-cognitive skills among patients with schizophrenia. Extant research that demonstrates some effects for mood induction among patients with schizophrenia is an important step in this direction (Berenbaum & Oltmanns, 1992; Blanchard, Bellack, & Mueser, 1994; Kring & Neale, 1996).

Finally, the significant association between social cognition and social functioning suggests that remediation of social-cognitive skills may be a critical component of successful psychosocial rehabilitation. For example, an affect perception training program, consisting of to identify specific facial features (e.g., lips), link features to feelings (e.g., a wrinkled nose occurs with disgust), and reinforce correct affect identification responses, has been effectively implemented with adults having developmental disabilities (McAlpine, Singh, Ellis, Kendall, & Hampton, 1992). Preliminary interventions for patients with schizophrenia are promising but have often been primarily limited to multiple case studies (see Penn & Mueser, 1996, for a review). We hope that the development of controlled group trials will reveal whether social-cognitive remediation augments the effectiveness of current psychosocial interventions.

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