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Symptom Domains and Neurocognitive Functioning Can Help Differentiate Social Cognitive Processes in Schizophrenia: A Meta-Analysis

Joseph Ventura*, Rachel C. Wood, and Gerhard S. Hellemann

Department of Psychiatry and Biobehavioral Sciences, Semel Institute for Neuroscience and Human Behavior, University of California Los Angeles, Los Angeles, CA

*To whom correspondence should be addressed; Department of Psychiatry, University of California Los Angeles, 300 Medical Plaza, Room 2243, Los Angeles, CA 90095-6968, US; tel: 310-206-5225, fax: 310-206-3651, e-mail: jventura@ucla.edu

Background: The existence of deficits in several social cognitive domains has been established in schizophrenia, and those impairments are known to be a significant determinant of functional outcome. Both symptoms and neurocognition have been linked to social cognitive deficits, but the nature and the relative strength of these relationships have not been established. Methods: A meta-analysis of 154 studies (combined $N = 7175$) was conducted to determine the magnitude of the relationships between 3 symptom domains (reality distortion, disorganization, and negative symptoms) and 6 Measurement and Treatment Research to Improve Cognition in Schizophrenia (MATRICS) domains of neurocognition with 4 domains of social cognition. Analyses were conducted to determine whether the strength of these relationships differed depending on the symptom type or neurocognitive domain under investigation. Results: The correlations between reality distortion and the domains of social cognition ranged from near zero to moderate (*r*'s range from −.07 to −.22), as compared with the moderate association for disorganization (*r*'s range from −.22 to −.32) and negative symptoms (*r*'s range from −.20 to −.26). For each of the neurocognitive domains, the relationships to social cognitive domains were mostly moderate (*r*'s range from .17 to .37), with no one neurocognitive domain being prominent. Conclusions: The effect sizes of the correlations between disorganization and negative symptoms with social cognition were relatively larger and more consistent than reality distortion. The relationship between social cognition and 6 MATRICS domains of neurocognition were mostly moderate and relatively consistent. When considering disorganization and negative symptoms, the relationship to social cognitive processes was relatively as strong as for neurocognition.

Key words: social cognition/reality distortion/disorganization/negative symptoms/neurocognition/meta-analysis

Introduction

Social cognition has recently become one of the most relevant topics for understanding functioning in the daily lives of people with schizophrenia.[1,2] The significance of social cognitive processes is supported by an extensive body of research that includes hundreds of empirical articles and well over 25 reviews.[3,4] Schizophrenia patients have clearly been shown to have social cognition deficits as compared with normal individuals in the key domains identified by the National Institute of Mental Health (NIMH) Consensus Committee[2]: Emotion Perception and Processing, Social Perception and Knowledge, Attributional Bias, and Theory of Mind (ToM). These deficits appear to be present in the prodrome (or high risk samples), in first-degree relatives of schizophrenia patient probands, in early course patients, and during periods of symptom remission.[3–10] In addition, social cognition deficits might have an underlying biological basis, which to some extent might be influenced by genes.[9,11] Social cognition deficits have been identified as a relevant component in the development of various types of symptoms and might be a mediator of the relationship between neurocognition and outcome.[12–16] In fact, social cognition appears to be a valid predictor of outcome that explains additional variance, which cannot be accounted for solely by neurocognition.[4,12,17–19] Social cognition deficits are considered so critical a domain for good functioning that a number of social cognition training programs have been developed and implemented.[20] Interestingly, social cognitive deficits have been linked to symptoms and to neurocognition separately. However, the relative magnitude and meaning of these relationships could benefit from a comprehensive review and integration.

Psychiatric symptoms are a defining feature of schizophrenia and play an important role in predicting

functional outcome.[17,21–23] Despite all that is known about symptoms, we lack a needed understanding of the relationships between major symptom dimensions such as reality distortion, disorganization, and negative symptoms and the domains of social cognitive processes.[2,24] The evidence seems to indicate that specific symptom types might be related in differential ways to separate social cognitive processes, but how these domains map onto each other is less clear.[24–27] Several investigators have suggested possible mechanisms for how social cognition processes could be associated with the development of positive symptoms.[17,26,28–30] However, studying the relationship between positive symptoms and social cognition is mired by the fact that many studies combine reality distortion and disorganization in their definition of positive symptoms, obscuring which positive symptom is most relevant to which social cognitive process. One of the most consistent findings to date seems to be an association between disorganization and deficits in ToM.[8,30,31] In contrast, there appears to be relatively weaker associations between reality distortion and social cognitive processes, eg, ToM.[32,33] Additionally, several articles have suggested that the relevance of negative symptoms in understanding various forms of social cognitive processes.[2,14,34,35] One possibility is that negative symptoms that involve reduced emotional experience (ie, anhedonia) or expression (ie, affective flattening) might be more associated with the development or maintenance of social cognition deficits.[14] Symptoms and social cognitive deficits appear linked in fundamental ways that can help explain the nature of social functioning and could add in guiding the remediation of these social cognitive deficits.

A conceptual issue is currently facing the field regarding how closely social cognitive processes overlap with neurocognition. This is important because doubts have been raised about the independence of these 2 constructs. Some research suggests that neurocognition and social cognition are largely distinct domains.[14,36,37] Yet, these 2 domains are often found to be at least moderately correlated.[4] One of the challenges is that the construct of social cognition in schizophrenia is broad and multidimensional but social cognition concepts overlap. Thus far, the most frequently studied components of social cognition in schizophrenia have been emotion perception/processing and ToM. The question remains as to how the domains of social cognition are related to the key domains of neurocognition. Assuming that social cognition is an independent construct, then are the separable neurocognitive domains differentially or uniformly correlated with the domains of social cognition?[38] Some social cognitive skills that might be of a lower order, eg, emotion recognition, might be associated with lower order (bottom-up) neurocognitive skills such as attention.[39,40] Some higher order cognitive skills (top-down), such as reasoning and problem solving ability or abstraction skills, might be associated with specific higher level social cognitive functions, eg, ToM.[31,41] Perhaps, a comprehensive examination of the separate neurocognitive domains in relationship to identified domains of social cognitive processes can help determine how these 2 key constructs are associated.

The aims of this meta-analysis were (1) to examine the relative magnitude of the relationship between key symptom domains, ie, reality distortion, disorganization, and negative symptoms, with social cognitive processes and (2) to determine if examining the correlations between 6 separate neurocognition domains with 4 identified social cognitive processes could differentiate the various components of social cognition in schizophrenia.

Methods

Procedures

We conducted a literature search of the following databases: *PsychInfo, PsychAbstracts, EBSCOhost, PubMed*, and *Google Scholar* covering the period from January 1, 1977 to February 28, 2011. Searches were restricted to articles published in the English language. The following key search terms were used (selected list, some terms were combined): social cognition, emotion recognition, affect recognition, affect prosody, emotions, ToM, attribution, social skills, schizophrenia, schizoaffective, schizophreniform, neurocognition, positive symptoms, negative symptoms, disorganization symptoms, and formal thought disorder. The reference lists of published articles identified by this method were then screened to locate additional relevant studies. Additionally, we used the search options in PubMed and Google Scholar that allow for a search of articles with related topics.

Using these search methods, 552 articles were identified as potentially relevant to this topic. These studies were then evaluated using the following inclusion criteria: (1) study must have used empirical methods and been published in a peer reviewed journal; (2) study must have contained descriptions of study measures and operational definitions of variables; (3) study must have used structured assessments of symptoms with established scales or standardized methods of symptom assessment; (4) study must have assessed neurocognitive functioning using standardized batteries; (5) study must have been cross-sectional (as defined by an assessment interval of 90 days or less); (6) all participants in the study must have been diagnosed with schizophrenia, schizoaffective disorder, or schizophreniform disorder according to *Diagnostic and Statistical Manual of Mental Disorders* criteria; (7) statistics reported must have been correlation coefficients or other statistics that could be converted into correlations so that an effect size and z score could be calculated; and (8) study data must not have been included or published previously in another article included in this analysis.

A total of 398 articles did not meet these criteria and were excluded from the study. A total of 154 studies met the inclusion criteria with a combined total sample of 7175 patients (for a comprehensive list, see online supplementary material for table S1). Studies of inpatients (n = 50), outpatients (n = 69), and combined samples (n = 35) were included (3 studies did not report patient status; see online supplementary material for table S1). Data from all of the 154 studies were compiled in a database containing: (1) the author(s) and year of publication; (2) sample characteristics; (3) demographic information; (4) description of the neuropsychological tests, eg, California Verbal Learning Test, and the neurocognitive domain assessed by each test, (5) symptom measures, eg, Scale for the Assessment of Positive Symptoms (SAPS), and the symptom domains examined by these measures; (6) description of the social cognition assessment used, ie, Social Cue Recognition Test, and the social cognition domain examined by each measure; and (7) study statistics, eg, correlation coefficients. From our database of 154 studies, we identified those studies that reported relationships between symptoms and social cognition (n = 91), studies that reported relationships between neurocognition and social cognition (n = 35), and studies reporting relationships between all 3 domains (n = 28). The aggregate sample characteristics were as follows: 68% of patients were male, the mean age was 36.5 years (SD = 5.6), and the mean education was 12.4 years (SD = 1.1).

Defining Symptoms, Neurocognition, and Social Cognition

Symptoms. The dimension of "reality distortion" included positive symptoms consisting of delusions, eg, suspiciousness, and hallucinations, eg, auditory, as measured by items from structured symptom scales, eg, Positive and Negative Syndrome Scale (PANSS), Brief Psychiatric Rating Scale (BPRS; see online supplementary material for table S2). The dimension of "disorganization" included positive symptom items such as conceptual disorganization, formal thought disorder, mannerisms and posturing, and bizarre behavior, as measured by structured assessment scales, eg, PANSS, SAPS. The "combined" category was composed of studies that used a definition of positive symptoms that included both reality distortion and disorganization, eg, by using the PANSS positive symptom scale which includes delusions, hallucinations, and conceptual disorganization or the SAPS total score which combines delusions, hallucinations, and formal thought disorder. The dimension of "negative symptoms" included symptoms such as blunted affect, emotional withdrawal, and motor retardation, as measured by items from structured symptom scales, eg, Scale for the Assessment of Negative Symptoms (SANS), BPRS.

Neurocognition. For the current study, "neurocognition" was operationally defined as cognitive functions, such as verbal memory and working memory that are objectively measurable with standardized neuropsychological tests, such as the Wechsler Adult Intelligence Scale (WAIS) Digit Span Test (see online supplementary material for table S3). As part of Measurement and Treatment Research to Improve Cognition in Schizophrenia (MATRICS) initiative, 7 major separable domains of neurocognition were identified.[38] The current study used 6 of the 7 domains for categorizing objective measures of neurocognition into the following: Verbal Learning and Memory, Visual Learning and Memory, Working Memory, Reasoning and Problem Solving, Speed of Processing, and Attention/Vigilance. Because one of the primary aims of this study was to examine separate neurocognitive domains in relationship to the various types of social cognitive processes, the MATRICS domain of social cognition was not included as a neurocognitive domain for the current analyses.

Social Cognition. For the current study, "social cognition" was operationally defined as the cognitive skills needed to process social information, as measured by standardized assessments such as the Bell-Lysaker Emotion Recognition Test (see online supplementary material for table S4). The classification of social cognition domains was based on the NIMH Consensus group categories,[2] as well as the domain grouping established by Fett et al.[4] There were 4 domains of social cognition: Emotion Perception and Processing, Social Perception and Knowledge, Attributional Bias, and ToM. The Emotion Perception and Processing domain consists of the skills needed to correctly identify, understand, and manage emotions. The Social Perception and Knowledge domain is composed of skills needed to recognize social cues and understand the roles, rules, and goals that characterize social situations. The Attributional Bias domain consists of the skills needed to appropriately infer the causes of particular positive and negative events. The domain of ToM, also known as mental state attribution, represents the ability to infer the beliefs and intentions of others, as well as the ability to interpret complex mental states such as inferring one person's belief about another person's belief and concepts such as irony, sarcasm, and humor.

Data Analysis Procedures

We examined the effect size differences between 3 symptom domains, 6 MATRICS domains of neurocognition, and 4 domains of social cognition. We were interested in examining separately the differences between reality distortion, disorganization, and negative symptoms with the 4 domains of social cognition. We also examined separately the relationship between verbal learning and

memory, visual learning and memory, working memory, reasoning and problem solving, speed of processing and attention/vigilance with the 4 domains of social cognition. The first step for these analyses was to transform the observed (published) correlations in each study using Fisher's *r*-to-*z* transformation. Where indicated, multiple results were averaged from the same domain, eg, several tests of working memory were combined into a single observation for a given study. The correlation coefficients were then combined into a single estimate of the population correlation by averaging the coefficients weighted by sample size.[42] The estimated overall effect sizes were then tested for homogeneity across studies by calculating Q statistics.

Sample Diagnostics. We found that most of the effects were homogeneous, ie, all studies have approximately the same effect size (table 1). The studies that are not homogeneous are identified. If the differences in effect sizes between studies were associated with specific study characteristics, the study characteristic and direction of the relationship are provided (table 1). In the studies with a heterogeneous effect size, we tested for effect size associations with the key study variables as follows: mean age, mean education, gender ratio, inpatient or outpatient status, sample size, and year of publication. For a few of the meta-analyses, we found significant associations between effect size and study characteristics, but none of the associations were unexpected. Working memory performance was associated with degree of education, and average disorganization with higher proportion of males in the study. Additionally, large samples tended to have smaller effect sizes with verbal memory measures. The alternative estimates for the overall effect sizes based on the homogeneous subset studies are provided (table 1). Although a few studies were identified as outliers in some of the neurocognitive domains, we found it difficult to justify excluding one study for not being a valid source of information for a given domain, and yet valid for another. Using this rationale, the study results are based on parameter estimates from all studies. For comparison purposes, parameter estimates based on the homogeneous subset studies are provided (table 1). Although the significance of the reported P values is potentially inflated, the data presented here can be considered as being a reasonably robust representation of the relationships between the variables of interest.

Results

To best interpret the results, we believe that the correlations between the variables of interest and social cognition should be understood largely as effect sizes rather than attempting multiple tests of the statistical significance of these relationships. Even if the P value examining the difference between 2 correlations was high, this would most likely reflect that this meta-analysis is well powered to detect small but not scientifically meaningful differences given the large sample sizes. We used the classification of effect sizes according to the commonly referenced standards of Cohen[43] indicating that a small $r = .10$, a medium $r = .30$, and a large $r = .50$.

Symptoms and Social Cognition

To address one of the primary questions posed in this study, we examined the cross-sectional relationships between the 3 symptom domains, separately, to the 4 identified social cognitive processes (table 2). Interestingly, the only symptom category that had sufficient studies for the meta-analysis of Attributional Bias was reality distortion. The relationship between reality distortion and the 4 domains of social cognition ranged from minimal to medium (*r*'s ranged from $-.07$ to $-.22$). The relationship between social cognition and reality distortion was medium for Emotion Perception and Processing ($r = -.22$) and Social Perception and Knowledge ($r = -.21$) but minimal for Attributional Bias and ToM ($r = -.07$ and $r = -.08$, respectively). These correlations were found to be significantly different from each other, eg, the correlation between reality distortion and Emotion Perception ($r = -.22$) was significantly different from the correlation between reality distortion and ToM ($r = -.08$, $P < .01$). In contrast, relatively consistent and medium effect size relationships were found between the 3 social cognition domains (for which there was sufficient published data) and disorganization (*r*'s ranged from $-.22$ to $-.32$) and negative symptoms (*r*'s ranged from $-.20$ to $-.26$). The pattern of effect sizes for the studies that combined reality distortion and disorganization in relationship to the 3 analyzable social cognition variables was small to medium (*r*'s ranged from $-.11$ to $-.22$). Combining the effects of these 2 types of positive symptoms resulted in intermediate relationships that on average were lower (aggregated $r = -.17$) than the strength of the relationships found when examining disorganization (aggregated $r = -.29$) or reality distortion (aggregated $r = -.24$) separately.

Neurocognition and Social Cognition

Because there were too few published studies to include in this meta-analysis examining Attributional Bias as related to the domains of neurocognition, we did not report on those relationships. The relationships between the 6 MATRICS neurocognitive domains and the 3 domains of social cognition (for which there was sufficient published data) were mostly moderate in effect size and consistent across neurocognitive domains (table 3). We found a relatively small range of correlations for Emotion Perception and Processing (*r*'s ranged from .22 to .30), as compared with Social Perception and Knowledge (*r*'s ranged from .17 to .37), and ToM (*r*'s ranged from .18 to .34). However, only 3 of the correlations between

Table 1. Meta-Analysis Diagnostics

	r	Critical *N*	*Q* Statistic	*P*	Significant Study Characteristics	Number of Studies Dropped for Homogeneity	*r*	*P*
Emotion perception								
Verbal memory	.28	264	28.10	.01	Sample size	Gur et al[44]	.24	<.01
Visual memory	.30	143	2.61	.99				
Working memory	.22	90	12.20	.09				
Reasoning and problem solving	.30	266	17.38	.30				
Speed of processing	.29	279	18.28	.31				
Attention/vigilance	.26	216	20.00	.22				
Reality distortion	−.22	164	44.22	.00	—	Weniger et al[45]; Martin et al[46]	.21	<.01
Disorganization	−.32	324	42.33	.01	Proportion male	Höschel and Irle[47]	.34	<.01
Combined positive symptoms	−.17	128	6.99	.98				
Negative symptoms	−.26	701	109.70	.00	—	Streit et al[48]; Suslow et al[49]; Herbener et al[50]; Bell and Mishara[51]; Nelson et al[52]; Turetsky et al[53]; Behere et al[54]; Eack et al[55]	.30	<.01
Social perception								
Verbal memory	.37	337	25.87	.01	Sample size	Bell et al[41]	.31	<.01
Visual memory	.30	54	1.68	.43				
Working memory	.17	53	16.15	.00	Education	Schwartz et al[56]	.22	<.01
Reasoning and problem solving	.33	181	4.98	.76				
Speed of processing	.24	116	3.83	.80				
Attention/vigilance	.17	70	12.18	.09				
Reality distortion	−.21	37	13.34	.04	—	Revheim and Medalia[57]	.37	<.01
Disorganization	−.22	48	10.55	.16				
Combined positive symptoms	−.11	73	33.49	.00	—	Zhu et al[58]	.16	<.01
Negative symptoms	−.20	191	37.64	.00	—	Bellack et al[59]	.22	<.01
Attributional bias								
Verbal memory	—	—						
Visual memory	—	—						
Working memory	—	—						
Reasoning and problem solving	—	—						
Speed of processing	—	—						
Attention/vigilance	—	—						
Reality distortion	−.07	14	25.87	.01	—	Martin and Penn[60]; Mortiz et al[61]	−.06	.41
Combined positive symptoms	—	—						
Disorganization	—	—						
Negative symptoms	—	—						
Theory of Mind								
Verbal memory	.31	179	15.46	.08				
Visual memory	.22	41	0.22	.97				
Working memory	.33	117	5.04	.54				
Reasoning and problem solving	.34	258	15.58	.48				
Speed of processing	.18	122	8.18	.70				
Attention/vigilance	.24	65	6.48	.26				
Reality distortion	−.08	46	15.50	.34				
Disorganization	−.32	223	19.14	.26				
Combined positive symptoms	−.22	125	13.28	.58				
Negative symptoms	−.25	470	43.43	.25				

Table 2. Correlations Between Symptoms and Social Cognition

	Emotion Perception	*N* (*k*)	Social Perception	*N* (*k*)	Attributional Bias	*N* (*k*)	Theory of Mind	*N* (*k*)
Reality distortion	−.22	757 (18)	−.21	182 (6)	−.07	250 (6)	−.08	624 (14)
Disorganization	−.32	987 (22)	−.22	228 (7)			−.32	684 (16)
Negative symptoms	−.26	2303 (53)	−.20	952 (18)			−.25	1869 (38)
Studies that combined reality distortion and disorganization …								
Combined positive symptoms	−.17	771 (17)	−.11	684 (11)			−.22	583 (15)

Note: *k*, number of studies; *N*, total number of subjects.

neurocognition and any domain of social cognition were below $r = .22$ and only 2 were above $r = .33$, ie, the majority were between $r = .22$ and $r = .33$. Relatively speaking, no one domain or group of neurocognitive domains was obviously more strongly related to any of the domains of social cognition than another neurocognitive domain. The relative strength of the relationship of the 6 MATRICS neurocognitive domains with social cognition was medium (aggregated *r*'s ranged from $r = .23$ to $r = .34$), which was similar to but perhaps on average somewhat higher than that of disorganization (aggregated $r = -.29$) or negative symptoms (aggregated $r = -.24$).

Discussion

Meta-analytic techniques were used to examine the magnitude of cross-sectional relationships between 3 symptom domains, 6 MATRICS domains of neurocognition, and 4 domains of social cognition. Consistent with previous investigations and theorizing, reality distortion showed a moderately strong relationship with 2 domains of social cognition, Emotion Perception and Processing and Social Perception and Knowledge. But that relationship was minimal in strength for Attributional Bias and ToM. Compared with reality distortion, the correlations between social cognition and both disorganization and negative symptoms, separately, were moderate and more consistent across the domains of social cognition. Similarly, the relationships between most of the 6 domains of neurocognition and the domains of social cognition were mostly moderate and, on average, relatively consistent. When considering either disorganization or negative symptoms separately, the relative magnitude of the relationships to social cognition was similar in magnitude to the relationship between social cognition and the 6 separate domains of neurocognition.

Our findings support the classic models of Liddle[62] and Bilder et al[63] indicating the existence of a 3-dimensional symptom model in schizophrenia of reality distortion, disorganization, and negative symptoms, each of which should be considered separately in understanding social cognitive deficits in schizophrenia.[22,23,64] Specifically, our findings provide support for believing that reality distortion and disorganization represent separate dimensions of positive symptoms in schizophrenia with differential links to social cognition. The relative strength of the link between social cognition and reality distortion varied from minimal to medium, whereas disorganization was more consistently and moderately correlated to social cognition. Meta-analytic studies such as Kohler et al[7] which did not separate these 2 forms of positive symptoms run the risk of obscuring important independent relationships to social cognition variables. These authors used the SAPS total score and the PANSS positive symptom subscale score, which could be problematic because those sets of ratings combine delusions, hallucinations, disorganization, and formal thought disorder. Research that used combined positive symptom ratings might have contributed to the impression that symptoms, as a whole, are not related to key domains in schizophrenia such as neurocognition and functional outcome. While true that

Table 3. Correlations Between Neurocognition and Social Cognition

	Emotion Perception	*N* (*k*)	Social Perception	*N* (*k*)	Theory of Mind	*N* (*k*)
Verbal memory	.28	915 (14)	.37	867 (12)	.31	572 (9)
Visual memory	.30	469 (10)	.30	184 (2)	.22	198 (3)
Working memory	.22	424 (7)	.17	323 (4)	.33	352 (6)
Reasoning and problem solving	.30	870 (15)	.33	540 (8)	.34	747 (16)
Speed of processing	.29	943 (16)	.24	478 (7)	.18	683 (11)
Attention/vigilance	.26	828 (16)	.17	424 (7)	.24	279 (5)

Note: *k*, number of studies; *N*, total number of subjects; We did not report on the relationships between Attributional Bias and the domains of neurocognition because there were too few published studies examining these relationships to include in this meta-analysis.

meta-analyses have shown that reality distortion, when considered separately from disorganization, showed a weak correlation to neurocognitive deficits and functional outcome in schizophrenia, disorganization, and negative symptoms were significantly related to neurocognition and to functional outcome.[22,23] Given that reality distortion was related to 2 social cognition domains in the current analysis rather than all 4 could prove useful in understanding the development of both symptoms and social cognitive processes.

Our findings support theorists such as Frith[29] and Sarfati et al[65] who proposed explanations for how positive symptoms might be associated with social cognitive processes.[17] In fact, most of the theories about how social cognition in schizophrenia patients might be associated with the development of symptoms involve associations with ToM.[8,29,30,66] Theorists who studied ToM have hypothesized that an inability to adequately represent another person's mental state or integrate contextual information was associated with symptoms of disorganization.[8,30] Although reality distortion was not strongly related with ToM in our analysis, there was a small effect for ToM, which supports the hypothesis of a mechanism for the involvement of reality distortion. Perhaps there is a reason reality distortion was not more strongly or consistently related to ToM. Some patients experiencing paranoia might have enough intact intellectual functioning to infer that others have mental states but are more impaired in emotion processing or social perception.[29] Our findings support Couture et al's[16] model that deficits in emotion perception might involve the misattribution of ambiguous negative emotions, ie, sad emotions can be misinterpreted as anger, or the misreading of another's intentions.[3,29,30,67] Those misattributions, if accompanied by ToM deficits, might be related to paranoid thinking. The magnitude of the relationship between negative symptoms and social cognition provides some indication of how all of these constructs might be associated. Behaviors such as alogia or withdrawal could be associated with ToM deficits such as accurately monitoring the intentions of others or misattributing the reasoning used by one person about another person. Our findings also support theorists who have linked negative symptoms with the development of social cognitive deficits such as a lack of ability to understand social contexts.[66] Finding these links between symptoms and social cognitive processes could help broaden our understanding of these important relationships and guide the development of social cognition training programs.

The question of whether specific MATRICS domains of neurocognition and the 3 analyzable domains of social cognition "map on" to each other in a specific pattern was addressed in these analyses. Considering that most of the 6 MATRICS domains of neurocognition were moderately correlated with 3 domains of social cognition, the results of this meta-analysis support previous assertions that social cognition and neurocognition are related but separate constructs.[14,36,37] The relationship between neurocognition and the domains of social cognition was generally consistent with mostly moderate effect sizes (most *r*'s ranged from .22 to .33), with no zero relationships, and few minimal effect sizes. No one neurocognitive domain clearly stands out as the "core" ability or skill that is associated with good social cognitive processing. These findings do not support the hypothesis that specific neurocognitive abilities are selectively associated with specific social cognitive processes, eg, executive functions are associated with ToM. In addition, there is an average of about 10% of shared variance between the 2 constructs, which leaves a large amount of variance in social cognitive processes that needs to be explained in other ways. Interestingly, there were an insufficient number of studies that addressed the relationship between Attributional Bias and neurocognition. Finding the overlap and magnitude of the effects between the 6 domains of neurocognition and the 4 social cognitive processes, especially in comparison to strength of the relationships to symptoms, could help explain the nature of these fundamental processes in schizophrenia.

There are study limitations, several of which are common to all meta-analytic investigations, and a number of which we have discussed in prior meta-analytic work on related topics.[22,23,68,69] The study sample was not randomly selected, the data are from a secondary source, and the data are cross-sectional so directional hypotheses were not testable. The choice regarding which variables are conceptualized as "cause" and which to consider an "effect" is essentially arbitrary. Based on theoretical considerations, symptoms and neurocognition might be viewed as underlying contributors to the severity of social cognitive deficits. However, the converse has equal merit that social cognition deficits can contribute to symptom development. Additionally, symptoms, neurocognition, and social cognition are not homogenous concepts and their definition in this meta-analysis is influenced by how commonly a particular set of tests in a domain appear in the published literature, eg, the Continuous Performance Test (CPT), predominated in the category of attention/vigilance. Studies that include measures of negative symptoms and emotion perception are comparatively common, while the relationship between attributional bias as related to symptoms and neurocognition is underrepresented. We note that variability in the type of assessment selected, eg, of negative symptoms, could account for some of the heterogeneity in our results. While these imbalances reflect the current state of the field, they also lead to vastly differing degrees of confidence in the results in specific variable pairings. These study limitations suggest that caution should be used in interpreting the results, yet our findings still provide some direction for future research on social cognition.

Our findings suggest that the 3 major symptom types might contribute in unique ways to the understanding of the 4 identified social cognitive processes. This understanding could help toward differentiating the various domains of social cognition from each other and might help explain social cognitive processing deficits from a different perspective than neurocognition. However, there is still a fair amount of variance in each of these social cognition constructs that seems to be unique to that construct. Additionally, the causal direction of these associations has not been established which awaits future empirical research directed at clarifying the nature of these links that is longitudinal in design.

Supplementary Material

Supplementary material is available at http://schizophreniabulletin.oxfordjournals.org.

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