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# Motivation and its Relationship to Neurocognition, Social Cognition, and Functional Outcome in Schizophrenia

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ABSTRACT

*Objective:* A burgeoning area of research has focused on motivational deficits in schizophrenia, producing hypotheses about the role that motivation plays in the well-known relationship between neurocognition and functional outcome. However, little work has examined the role of motivation in more complex models of outcome that include social cognition, despite our increased understanding of the critical role of social cognition in community functioning in schizophrenia, and despite new basic science findings on the association between social cognitive and reward processing in neural systems in humans. Using path analysis, we directly contrasted whether motivation 1) causally influences known social cognitive deficits in schizophrenia, leading to poor outcome or 2) mediates the relationship between social cognitive deficits and outcome in this illness.

*Method:* Ninety one patients with schizophrenia or schizoaffective disorder completed interview-based measures of motivation and functional outcome as well as standardized measures of neurocognition and social cognition in a cross-sectional design.

*Results:* In line with recent research, motivation appears to mediate the relationship between neurocognition, social cognition and functional outcome. A model with motivation as a causal factor resulted in poor fit indicating that motivation does not appear to precede neurocognition.

*Conclusions:* Findings in the present study indicate that motivation plays a significant and mediating role between neurocognition, social cognition, and functional outcome. Potential psychosocial treatment implications are discussed, especially those that emphasize social cognitive and motivational enhancement.

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## 1. Introduction

It is now well-established that neurocognition and functional outcome are associated in schizophrenia (Gottesman, 1994; Green, 1996; Green et al., 2000) and compelling evidence indicates that deficits in social cognition mediate this relationship (Brekke et al., 2005; Couture et al., 2006; Yager and Ehmann, 2006). For example, using path analysis, Brekke and colleagues (2005) confirmed that social cognition and social support mediate the relationship between neurocognition and functional outcome, both cross-sectionally and prospectively. Although the model fit was excellent, the authors suggested further investigation of additional contributing factors unspecified by the model in order to identify other critical variables that play a role in community outcome.

One important factor that has received increasing attention in schizophrenia is motivation. As several researchers have noted, motivation likely plays an important role in the cognitive dysfunction of schizophrenia, with important associations to functioning (Barch, 2005; Barch et al., 2008; Docherty et al., 2001; Gold et al., 2008; Heerey and Gold, 2007). However, it is less clear what the causal relationships

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might be between motivation, neurocognition and social cognition, and it is also unclear how these variables interact to alter outcome. Historically, theorists and clinicians have considered emotion and motivation impairments to be a central feature of schizophrenia, and even one of the predisposing or causal factors in the development of the disorder. Bleuler (1911/1950) and Kraepelin (1919/1913) regarded "indifference" in patients as one of the fundamental symptoms of the illness. Later theorists (e.g., Meehl, 1962; Rado, 1953) expanded on this, positing that anhedonia is a congenital trait, primary among the factors in the development of schizophrenia. They argued that the result of this pleasure deficiency was two-fold: 1) pleasures resulting from goal attainment are not present and therefore do not reinforce positive or social behaviors, and 2) without the ability to experience pleasure, there is no 'softening' of negative emotional states, both of which lead to poor outcomes (e.g., Meehl, 1975). From this view, impaired motivation can be thought of as a causal factor influencing disease expression, cognitive performance, social interactions, and ultimately, outcome.

In contrast, recent work has framed motivation as a mediator between cognition and outcome. Drawing on behavioral neuroscience research which shows a distinction between in-the-moment pleasure and anticipated pleasure (Berridge and Robinson, 1998; Schultz, 2007; Wise, 2002), researchers have shown that schizophrenia patients appear to have a deficit in the more cognitively complex aspects of motivation – or anticipating that things will bring them pleasure (Gard et al., 2007). Others have linked anhedonia and other motivationally relevant deficits to problems with working memory (e.g., Burbridge and Barch, 2007). These findings suggest that the inability to maintain enduring representations of a pleasurable outcome, such as a positive social interaction, impedes patients from successfully engaging with goal-directed everyday activities, and thus ultimately leads patients to poorer functional status. Recently, structural equation modeling was used to test whether motivation acts as a mediator or a moderator between neurocognition and outcome (Nakagami et al., 2008). In other words, motivation was assessed on whether it accounted for the relationship between neurocognition and outcome (i.e., motivation as a mediator) or whether the relationship between neurocognition and outcome was explained by individuals who were particularly high or low in motivation (i.e., motivation as a moderator). In this analysis, motivation appeared to best fit as a mediator – explaining most of the relationship between neurocognition and functional outcome. However, in this study, there was no direct test of whether motivation would fit better as a factor preceding the relationship between neurocognition and outcome (e.g., using path analyses or other methods). In addition, social cognition was not included as a variable in the model.

Our position is that, in order to more fully understand the unique role of motivation in functional outcome in schizophrenia, it is important to consider its relationship to social cognition as well as to general neurocognition. Accurate processing of socio-emotive stimuli- which are among the most highly salient stimuli for the human brain- is intimately integrated with neural systems related to reward, learning, and motivation (Grace et al., 2007; Murray et al., 2008a,b; Waltz and Gold, 2007). Thus, it is possible that deficits in processing social stimuli have an adverse effect on motivational state in schizophrenia, and through this mechanism show a negative influence on functional status. This would be an important relationship to delineate, since it has potentially useful treatment implications, suggesting that focused treatment of social cognitive deficits could have a direct impact on motivational features of schizophrenia and thus on community functioning.

Therefore, in the present study we sought to: 1) replicate earlier findings that neurocognition is related to functional outcome and mediated by social cognition, 2) test motivation as a causal (or exogenous) factor (preceding neurocognition and social cognition) or 3) as a mediating variable (between neurocognition, social cognition and outcome) (see Fig. 1a-c for examples of these models). Using path analysis, and model fit indices, the present study tested these alternative models of functional outcome in a cross-sectional design. Path analysis is a useful technique in testing the relationships among variables, and though it cannot directly confirm causality with correlated variables, model fit statistics can illuminate whether variables are better construed as exogenous or endogenous.

## 2. Method

### 2.1. Participants

Ninety one schizophrenia outpatients were recruited from psychiatric outpatient services and community mental health clinics in the San Francisco Bay area. A diagnosis of schizophrenia was confirmed with the Structured Clinical Interview for Diagnosis for the DSM-IV-Clinician Version (SCID: First et al., 1997), the Positive and Negative Syndrome Scale-Extended (PANSS: Kay and Sevy, 1990; Poole et al., 2000) and a medical chart review. Additional exclusion criteria included history of head trauma, substance dependence during the past 6 months, neurological disorders, and English as a second language. All subjects were clinically stable at the time of testing (no hospitalization in the last three months, and no change in medication or dosage in the last 30 days). Symptom severity varied broadly; with average PANSS-E ratings in the mild range (see Table 1).

### 2.2. Procedures

The measures described below were administered within the context of a larger series of interviews and neuropsychological assessments over a 6 week period. All subjects provided written informed consent after experimental procedures were fully explained, and received nominal payment for their participation in this study.

### 2.3. Measures

#### 2.3.1.Neurocognition

A composite score of neurocognition was calculated for each participant as the average age-adjusted z-score across the following measures: WAIS-R Digit Symbol, Trail Making Test Part A, Category Fluency, WAIS-R Digit Span, WMS-R Visual Memory Span, California Verbal Learning Test (immediate and