Nonsocial and social cognitive function in psychosis: interrelationships, specificity and innovative approaches

In this issue of the journal, Green et al[1] present an excellent overview of impairments in nonsocial and social cognition in schizophrenia. They raise several key questions that are in need of further theoretical and methodological work.

One such question is the nature of the relationship between nonsocial and social cognition in general, and in schizophrenia more specifically. Green et al focus on these as separable constructs with differing psychological and neurological correlates. There is certainly ample evidence for meaningful distinctions between nonsocial and social cognition, with robust data about the engagement of different neural systems by tasks that focus more on one versus the other. Further, there is evidence that deficits in nonsocial and social cognition account for at least some independent variance in functional outcome in schizophrenia. However, there are also moderate to strong correlations between nonsocial and social cognition in schizophrenia[2]. Moreover, the intriguing data showing that social cognition mediates, at least in part, the relationship between nonsocial cognition and functional outcome suggest that at least some of the deficits in nonsocial cognition serve as building blocks (or barriers) to social cognitive function, and that there may be more synergy in attempts to treat both deficits simultaneously than previously emphasized.

This thinking about the ways in which different impairments interrelate and may mediate each other extends to the growing work on motivation discussed by Green et al. They note evidence that impairments in motivation or beliefs about one's inability to successfully carry out certain cognitive functions may partially mediate the relationship between nonsocial cognition and life function. Such results raise issues such as: To what extent living with cognitive impairment reduces motivation and creates negative beliefs? What components of motivational impairments might be independent of deficits in cognitive function? Would more integrated treatment approaches that tackle multiple levels of impairment simultaneously show more evidence for efficacy?

A second question is the status of cognition in schizophrenia versus psychopathology more broadly. Green et al describe cognitive deficits as a "core feature" of schizophrenia, which is central to understanding many aspects of risk and life function in that condition. However, these deficits are not a core feature in the sense of being selective to schizophrenia. It is becoming increasingly clear that many forms of psychopathology involve impairments in cognition. Green et al note this, but focus somewhat more on the differences across disorders than on the similarities. One could argue that the most robust evidence indicates similar profiles of cognitive impairment across disorders that involve psychosis, including schizophrenia, schizoaffective disorder, bipolar disorder with psychosis, and even psychotic depression[3-5]. The severity of these deficits often vary across illnesses, with the most severe in schizophrenia, but the general pattern is often remarkably similar[3,4]. Moreover, there is also evidence for impairment in at least some cognitive domains in a host of other forms of psychopathology, including non-psychotic major depression[6] and attention-deficit/hyperactivity disorder[7].

In fact, it has been argued that impairments in cognitive domains such as executive function, working memory, or cognitive control might form a more general risk factor for mental illness, perhaps being part of the "p factor" of shared risk for psychopathology[8]. If cognitive impairments, especially in domains thought to be critical for behavioral and emotional regulation, are part of a more general risk factor for psychopathology, we need to rethink their role in the development of psychotic disorders. This would not make cognitive deficits any less important for understanding the etiology, course or outcome of schizophrenia, but it would suggest a change in our thinking about causal factors and treatment interventions that may be much more widely applicable across forms of psychopathology.

A third question is how best to ameliorate deficits in either nonsocial or social cognition in schizophrenia. Green et al provide a nice review of the relevant literature, highlighting areas of both promise and concern. They note that remediation approaches have shown moderate effect sizes for improvement of both social and nonsocial domains, with the latter seeming to be most benefited when cognitive training is coupled with psychiatric rehabilitation. However, one can also read this literature in a much less positive light. Recent meta-analyses of cognitive remediation for nonsocial cognition suggest very modest effect sizes[9], and even effect sizes of a Cohen's d of .60 or .70 are likely too modest to make a meaningful and long-lasting impact on the lives of individuals with schizophrenia.

Green et al note features of cognitive impairment in schizophrenia that should lead us to question our focus on treating individuals who already have diagnosable illnesses or even prodromal symptoms. Specifically, cognitive impairment likely precedes the onset of psychosis by many years, and may be present even early in childhood. It seems highly unlikely that we can make significant inroads on improving cognitive function among individuals whose developmental trajectories have been disrupted by long-lasting and early occurring cognitive dysfunction. Instead, we may need to think about intervention approaches that can be applied much earlier in life, starting potentially in childhood, so as to help individuals shift back to a more typical developmental trajectory that may prevent the type of functional impairment often associated with schizophrenia.

The concern with such an approach has always been that we do not have any sufficiently predictive way to identify children who are likely to be at risk for psychosis. However, this is where the suggestion that at least some types of cognitive impairment

may be much broader risk factors for psychopathology comes into play. We need not be as concerned about identifying children who are *specifically* on a risk trajectory for psychosis if we think that impairments in domains such as cognitive control, executive function, or working memory serve as more general risk factors for psychopathology.

It is still absolutely critical to consider risk-benefit tradeoffs with even general risk factors. However, should we be able to develop non-invasive approaches that enhance these domains of cognition earlier in childhood or adolescence, we would be less concerned about whether such interventions have a protective effect against psychosis specifically, and more satisfied with either an overall reduction in risk for psychopathology, regardless of its manifestation, or an overall improvement in function even amongst those who still develop psychopathology.

While some might regard this suggestion as naive or unrealistic, I would argue that we need to consider fundamentally innovative approaches to treating or preventing cognitive impairment associated with all forms of mental illness, as years of research and countless treatment studies have yet to provide pathways that are sufficiently helpful once individuals develop severe psychiatric symptoms. It is time for us to think in ways that are much more "out of the box" and to use what the data are telling us about the developmental origins of cognitive deficits to identify the timing for intervention that is most likely to yield long-lasting and meaningful benefits.

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