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From the psychosis prodrome to the first-episode of psychosis: No evidence of a cognitive decline

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ABSTRACT

Cognitive deficits have an important role in the neurodevelopment of schizophrenia and other psychotic disorders. However, there is a continuing debate as to whether cognitive impairments in the psychosis prodrome are stable predictors of eventual psychosis or undergo a decline due to the onset of psychosis. In the present study, to determine how cognition changes as illness emerges, we examined baseline neurocognitive performance in a large sample of helping-seeking youth ranging in clinical state from low-risk for psychosis through individuals at clinical high-risk (CHR) for illness to early first-episode patients (EFEP). At baseline, the MATRICS Cognitive Consensus battery was administered to 322 individuals (205 CHRs, 28 EFEPs, and 89 help-seeking controls, HSC) that were part of the larger Early Detection, Intervention and Prevention of Psychosis Program study. CHR individuals were further divided into those who did (CHR-T; n = 12, 6.8%) and did not (CHR-NT, n = 163) convert to psychosis over follow-up (Mean = 99.20 weeks, SD = 21.54). ANCOVAs revealed that there were significant overall group differences (CHR, EFEP, HSC) in processing speed, verbal learning, and overall neurocognition, relative to healthy controls (CNTL). In addition, the CHR-NTs performed similarly to the HSC group, with mild to moderate cognitive deficits relative to the CTRL group. The CHR-Ts mirrored the EFEP group, with large deficits in processing speed, working memory, attention/vigilance, and verbal learning (> 1 SD below CNTLs). Interestingly, only verbal learning impairments predicted transition to psychosis, when adjusting for age, education, symptoms, antipsychotic medication, and neurocognitive performance in the other domains. Our findings suggest that large neurocognitive deficits are present prior to illness onset and represent vulnerability markers for psychosis. The results of this study further reinforce that verbal learning should be specifically targeted for preventive intervention for psychosis.

Impaired neurocognition has long been recognized to be a core feature of schizophrenia (Green, 2006; Nuechterlein et al., 2004). Cognitive deficits in attention, processing speed, working memory, verbal declarative memory, and executive functioning (Gold, 2004;

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Heinrichs and Zakzanis, 1998), for example, are not only readily apparent in the established illness (Harvey et al., 2010), but also prior to the onset of the disorder (Cannon et al., 2000). In fact, a pattern of cognitive dysfunction generally holds across a range of ages and clinical states, including very early in the pre-psychosis illness state, as extensively documented in individuals at clinical high-risk (CHR) (Brewer et al., 2006; Carrión et al., 2015; Cornblatt et al., 2015; Hawkins et al., 2004; Niendam et al., 2006; Seidman et al., 2010; Woodberry et al., 2010) for developing psychosis. Of particular interest, the early deficit pattern is typically less severe but qualitatively matches the cognitive impairment established for fully affected patients across all phases of psychosis (Aylward et al., 1984; Reichenberg et al., 2006). Decades of research have focused on the role of cognitive deficits in the processes leading to psychosis and possible prevention via cognitive remediation because of this developmental pattern. Nevertheless, there are a number of unresolved issues limiting progress in the field. Chief among these is whether cognitive impairment acts as a stable risk factor in a largely neurodevelopmental process or follows a neurodegenerative course through the progression of the illness (Harvey, 2009; Pino et al., 2014). A second, and related issue, is whether cognition as a whole declines after the onset of psychosis or whether deterioration is found only in specific domains. These distinctions have important implications for progress in prevention research. For example, treatment might best be directed to early and specific deficits while these are still moderate in intensity, thereby reducing the disease vulnerability (Cornblatt et al., 2003; Pukrop et al., 2007), possibly limiting the profound disability that is associated with the illness or improving the neurocognitive functioning itself (Green and Harvey, 2014).

Recent efforts aimed at reconciling the neurodevelopmental and neurodegenerative perspectives have increasingly focused on the extent of neurocognitive deficits prior to psychosis onset in clinical high-risk (CHR) adolescents and young adults (2017) (also referred to as ultra-high-risk, UHR) who display clinical features (e.g., symptoms, behaviors) that place them at heightened risk for developing psychosis. To date, numerous cross-sectional studies have reported small-to-medium impairments across various cognitive domains prior to illness onset (approximately 0.3–0.6SDs below healthy controls) in CHR individuals (Fusar-Poli et al., 2012; Giuliano et al., 2012; Woodberry et al., 2010). However, as noted above, these impairments are not as large as those seen at the first-episode of psychosis (Corigliano et al., 2014; Jahshan et al., 2010; Woodberry et al., 2013; Zhang et al., 2015), typically 1.0–1.5SDs below healthy controls (Corigliano et al., 2014; Zhang et al., 2015).

The aforementioned pattern of impairments suggests that while deficits precede acute psychosis manifestation, the period from CHR to psychosis onset may involve a progressive decline (Kim et al., 2011). In this case, rather than serving as vulnerability markers, neurocognition would serve as an illness (state) indicator of a worsening clinical state in the context of a neurodegenerative process. Accordingly, most functions would be deteriorating at around the same time as the illness progressed (Knoll et al., 1998; Seidman et al., 2006).

Alternatively, and consistent with neurodevelopmental models (Cornblatt et al., 2003, Lewis and Levitt, 2002; Murray et al., 1992; Walker and Bollini, 2002; Weinberger, 1987; Zubin and Spring 1977), there is evidence from CHR individuals that neurocognitive impairments are risk factors for psychosis that reflect underlying vulnerabilities of the emerging illness (Carrión et al., 2015, Cornblatt et al., 2015, Hawkins et al., 2008; Keefe et al., 2006; Lencz et al., 2006; Seidman et al., 2010) and do not decline post-onset (Carrión et al., 2015). For example, a recent report from the North American Prodrome Longitudinal Study (NAPLS), a large-scale, prospective study of high-risk youth, found that CHR subjects who transition to psychosis (also referred to as CHR converters) had moderate deficits in attention and working memory and declarative memory (approximately −0.75 SDs below controls) and performed significantly worse on these dimensions than non-converters (Cohen d effect size of 0.28 and 0.48, respectively). Transition to psychosis was best predicted by baseline measures of verbal learning and declarative memory (Seidman et al., 2016). In a recent report from our group (Carrión et al., 2015), CHR converters showed large domain-specific impairments at baseline in processing speed, verbal memory, sustained attention, and executive function, compared to CHR non-converters. These impairments were stable and persistent, but showed no further deterioration when retested soon after psychosis onset (Carrión et al., 2015).

These findings suggest that comparisons between CHR individuals and first-episode patients on neurocognitive performance are confounded, since, as a group and over a short-term (6–30 months), only approximately 20–35% of at-risk individuals are found to have an acute episode (Fusar-Poli et al., 2013). As a result, CHRs are expected as a group to be much less severely impaired (Bang et al., 2015; Liu et al., 2015). The true comparison, therefore, must be with individuals who are tested when they meet CHR criteria and develop psychosis over the course of the study. To date, however, very few studies have directly compared baseline performance of CHR converters to first-episode patients using the same neurocognitive battery.

The current study aimed to examine the baseline neurocognitive performance of three clinical subgroups of adolescents and young adults seeking treatment for psychosis-related symptoms. As part of the Early Detection, Intervention and Prevention of Psychosis Program (EDIPPP, McFarlane et al., 2012)), baseline performance on the Measurement and Treatment Research to Improve Cognition in Schizophrenia (MATRICS) (Green and Nuechterlein, 2004; Green et al., 2004b; Kern et al., 2004; Nuechterlein et al., 2008) consensus cognitive battery was collected from help-seeking controls (HSC), CHR individuals, and early first-episode psychosis patients (EFEPs). Help-seeking controls were included as an ecologically valid clinical control group, as they were referred to the prodromal clinic for risk assessment though did not meet strict CHR criteria (McGlashan et al., 2010). In addition, healthy comparison subjects (CNTL) were included to examine deviation from general population norms.

In the present study we aimed to: (1) Compare the three diagnostic subgroups (HSCs, CHRs, EFEPs) across six MATRICS neurocognitive domains, relative to healthy comparison (CNTL) subjects; (2) Examine differences between CHRs who transitioned to psychosis (CHR-T) to CHRs who did not (CHR-NT) and the EFEPs group; and (3) Determine whether specific neurocognitive impairments predict psychosis conversion among CHR youth. Our hypotheses were three-fold. First, we expected differences in baseline neurocognition across groups, with the largest global impairment in the EFEP group. Second, based on previous findings (Addington et al., 2017; Cornblatt et al., 2015; Hauser et al., 2017), we expected CHRs who transitioned to psychosis to perform worse than CHRs who did not transition to psychosis, specifically in verbal learning and processing speed. Finally, we expected baseline neurocognition to predict transition status beyond symptoms and other potential confounders, further supporting the role of neurocognition as a vulnerability marker for psychosis onset.

1. Material and methods

The data reported here were collected as part of EDIPPP, a large multi-site clinical trial for reducing risk for psychosis among young people funded by the Robert Wood Johnson Foundation (2007–2011) (Lynch et al., 2016; McFarlane et al., 2015). EDIPPP consisted of six participating sites: Portland, ME; Glen Oaks, NY; Ann Arbor, MI; Salem, OR; Sacramento, CA; Albuquerque, NM. Details of the study design, study implementation, assessments, psychosocial and pharmacological treatments, methods, and sample characteristics have been reported elsewhere (Carrión et al., 2016; McFarlane et al., 2015). Following standard CHR research, attenuated positive symptom levels were measured using the Scale of Prodromal Symptoms (SOPS) from the Structured Interview for Prodromal Syndromes (SIPS, Miller et al., 2003, 2002, 1999). Allocation to treatment was determined by a

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