In This Issue

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A brief summary of the articles appearing in this issue of *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*.

Disrupted Brain Development and Cognition in Early Psychosis

Subclinical psychotic symptoms peak during adolescence, a period during which brain connectivity and cognition dynamically mature. Using diffusion tractography imaging in typically developing youths and youths with psychosis spectrum symptoms, **Hegarty *et al.*** (pages 423–433) provide evidence of disrupted white matter development in the superior longitudinal fasciculus and retrolenticular internal capsule in youths on the psychosis spectrum. Further, white matter development in the superior longitudinal fasciculus mediated the relationship between age and cognition but only in typically developing youths, suggesting that disruptions in white matter development may drive cognitive deficits in psychosis.

Evidence indicates a link between abnormal early neurodevelopmental processes and risk of psychosis, but direct metrics are lacking. **Damme *et al.*** (pages 434–443) compared gyrification in clinical high-risk youths during the prodromal period to typically developing control subjects over two time points across three metrics. In the high-risk group, local gyrification index showed hypogyrification in the lateral orbitofrontal, superior temporal sulcus, anterior cingulate, and temporal poles; mean curvature index indicated sharper gyral and flatter/wider sulcal peaks in the cingulate, postcentral, and lingual gyrus; and sulcal depth identified shallow features in parietal, superior temporal sulcus, and cingulate regions. These findings suggest that gyrification may reflect early developmental insult and provide support for neurodevelopmental hypotheses of psychosis.

Schizophrenia is associated with both altered brain structure and impaired cognition, but whether antipsychotic exposure affects these characteristics is not clear. **Jessen *et al.*** (pages 444–453) report that antipsychotic-naïve first-episode schizophrenia patients display higher mean curvatures than healthy control subjects, suggesting that patients have a "sharper" folding of cortex. Moreover, analyses revealed a differential pattern of association between cortical thickness and cognitive functions in patients relative to controls. These results suggest that the link between cortical structures and cognitive deficits is present at the earliest, never-medicated stage of schizophrenia.

An extended duration of untreated psychosis is linked with poorer clinical outcomes in schizophrenia spectrum disorders, but the neural mechanisms underlying this phenomenon remain largely unknown. Using functional magnetic resonance imaging, **Manivannan *et al.*** (pages 454–461) demonstrate that reduced frontostriatal connectivity during working memory engagement is related to longer duration without treatment in individuals with first-episode psychosis. These data provide insight into the neural circuitry associated with untreated psychosis.

Antipsychotic Effects on White Matter in Schizophrenia

Progressive alterations in white matter have been identified in individuals with schizophrenia, but the underlying causes remain unclear, including whether treatment with antipsychotic medication plays a role. **Kraguljac *et al.*** (pages 462–471) used diffusion tensor imaging to investigate white matter integrity in never-treated and currently unmedicated patients with schizophrenia, before and after 6 weeks of treatment with risperidone. The authors found that microstructural white matter integrity at baseline was abnormal in patients compared to healthy control subjects, but they identified no changes in any white matter indices following treatment. These data suggest that antipsychotic treatment may not have short-term effects on white matter in schizophrenia.

Network Correlates of ADHD

Attention-deficit/hyperactivity disorder (ADHD) is a heterogeneous neurodevelopmental disorder. In this proof-of-concept study, **Pruim *et al.*** (pages 472–483) used data from a large cohort to investigate functional connectivity across multiple brain networks using a novel modeling framework. They then functionally mapped these neural correlates to neurocognitive measures. Abnormalities in the default mode network were primarily categorical, whereas those in the visual and sensorimotor networks were mainly related to inattentive and hyperactive/impulsive behavior. Findings within the salience network and cerebellum showed both categorical and dimensional mechanisms. This study presents a strategy to disentangle categorical diagnosis-related effects from dimensional behavior-related effects and provides insight into the neurobiology of ADHD.

Exercise, Depression, and the Hippocampus

Higher levels of exercise have been associated with less depression in children. In addition, exercise may modify hippocampal volume, a brain region that shows reduced volume in depression. Here, **Gorham *et al.*** (pages 484–492) examined sports involvement, depression, and hippocampal volume in children 9 to 11 years of age. The authors found that involvement in sports predicted fewer depressive symptoms, but only in boys. Additionally, involvement in sports showed association with larger hippocampal volume in both boys and girls. These findings help identify a potential mechanism for the impact of exercise on the developing brain.

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Thalamic Responses to Alcohol Expectancy and Cues

Alcohol expectancy contributes to excessive alcohol consumption. Drinkers also show enhanced reactivity to alcohol-related cues in association with problem alcohol use. In this study of nondependent adult drinkers, **Zhornitsky *et al.*** (pages 493–504) show that thalamus activation and its connectivity with the superior frontal gyrus is altered in response to alcohol cues, versus neutral cues, in correlation with alcohol expectancy. Mediation analyses demonstrated that alcohol expectancy mediated the contribution of cue-elicited thalamic response and connectivity to at-risk alcohol use.