In This Issue

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A brief summary of the articles appearing in this issue of *Biological Psychiatry*.

Transdiagnostic Network Alterations

The mesolimbic dopamine system and the salience network overlap in function and have been implicated in numerous psychiatric disorders, including schizophrenia and substance use disorders. **McCutcheon *et al.*** (pages 368–378) used a graph theory approach in combination with positron emission tomography and resting-state functional magnetic resonance imaging (fMRI) to probe the relationship between dopamine function and salience network connectivity. They show that increased dopamine synthesis capacity is related to greater salience network connectivity, particularly in brain regions identified as playing a central role in information processing.

Psychiatric disorders are commonly associated with cognitive dysfunction, but the underlying neural correlates are not well understood. **Sha *et al.*** (pages 379–388) conducted structural and functional imaging meta-analyses that revealed shared connectivity alterations across eight psychiatric disorders between three large-scale neurocognitive networks—the default mode, frontoparietal, and salience networks. This pattern of network alterations was associated with reduced gray matter in patients and was localized in regions linked to general cognitive performance. These findings suggest that shared network dysfunctions may underlie the generalized cognitive deficits observed across psychopathology.

Neurodevelopment and Psychopathology

Deviations in brain development across childhood and adolescence are relevant for several psychiatric disorders. Here, **Norbom *et al.*** (pages 389–398) used MRI to investigate age-related differences in gray/white matter contrast and its associations with psychopathology and cognition in a population-based sample of youths. Gray and white matter intensities were more similar for older youths. Regional gray/white matter contrast deviations were associated with higher symptom levels of psychosis and anxiety and also lower cognitive functioning, which may be related to differences in myelination.

Callous traits are risk markers for antisocial behavior and are associated with structural and functional brain alterations, but most research has been conducted in high-risk boys. Using structural MRI and diffusion tensor imaging in a general population sample of children, **Bolhuis *et al.*** (pages 399–407) found that callous traits are associated with widespread macro- and microstructural brain alterations, including lower global brain volume, decreased cortical surface area in frontal and temporal regions, and increased global white matter microstructure. The white matter associations were present only in girls. These findings provide insight into the neural correlates of callous traits in the general pediatric population.

ADHD and Traumatic Brain Injury

Attention-deficit/hyperactivity disorder (ADHD) is commonly diagnosed after traumatic brain injury (TBI) in youths. Here, **Stojanovski *et al.*** (pages 408–416) examined whether ADHD symptoms are differentially associated with genetic risk and brain structure in youths with and without a history of mild TBI. They report that genetic predisposition to ADHD does not increase the risk for ADHD symptoms associated with mild TBI. However, they did detect a differential relationship between ADHD symptoms and fractional anisotropy in the genu of the corpus callosum in youths with and without a TBI history, potentially suggestive of different underlying cellular-level disruption.

LINC01268 and Violent Suicide

Suicide by violent means was recently linked to the expression of *LINC01268*, a long intergenic noncoding RNA, in prefrontal cortex. **Punzi *et al.*** (pages 417–424) used RNA sequencing in prefrontal postmortem tissue to replicate this finding, where they confirmed that *LINC01268* expression was higher in individuals who committed suicide by violent means, compared with both individuals who committed suicide by nonviolent means and those who did not commit suicide. Then, in a cohort of living healthy subjects, the authors found an association between a single nucleotide polymorphism linked to increased *LINC01268* expression and aggressive behavior and emotional regulation. These data provide further evidence implicating *LINC01268* in suicidal behavior and aggression.

Decision Making Across Psychiatric Constructs

The process of making decisions occurs through habit (model-free control) and planning (model-based control). Psychiatric dimensions have been associated with disruptions in model-based control, yet it is unknown if symptom severity impairs dynamic changes in the model-based process. Using a computational framework, **Patzelt *et al.*** (pages 425–433) show that individuals boost model-based control in response to incentives across a range of severity on several psychiatric constructs. These boosts were more pronounced for the sensation-seeking trait and an anxious-depression factor. These data highlight the potential for incentive-based interventions to increase model-based control.

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