In This Issue

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

The Kappa Theory of Schizophrenia Reviewed

The kappa opioid receptor may play a role in the biology and treatment of multiple psychiatric disorders. Here, **Clark and Abi-Dargham** (pages 502–511) provide a comprehensive review of the preclinical, clinical, and histological evidence for a potential role of the dynorphin/kappa opioid receptor in the biology of both the positive and negative symptoms of schizophrenia.

Review: Multiscale Neuroscience of Psychiatric Disorders

In this review, **van den Heuvel *et al.*** (pages 512–522) discuss the emerging trend of multiscale neuroscience of psychiatric disorders, a multidisciplinary field that brings together data from all different levels of brain organization, from genes to neurons to macroscale levels of biological complexity and behavior. The overarching goal is to obtain an integrative understanding of how different layers of brain structure, function, and behavior work together in both health and disease. The authors conclude by discussing the opportunities, challenges, and future of this new field.

Genetic Risk for Schizophrenia: Targeted Treatment and Cortical Structure

A goal in psychiatry is to identify rare structural variants linked to disease biology that can then be targeted via therapeutic intervention. Here, **Bodkin *et al.*** (pages 523–535) identified a mutation (*GLDC* triplication) in two individuals with a diagnosis of a psychotic disorder that would be expected to result in *N*-methyl-D-aspartate receptor hypofunction. In two double-blind, placebo-controlled trials, glycine and D-cycloserine augmentation of psychotropic drug treatment each separately improved psychotic and mood symptoms. These results provide proof-of-principle demonstrations of symptom relief by targeting a specific mutation and explicitly linking it to a beneficial treatment response.

Schizophrenia is highly heritable and has been linked to both reduced cortical thickness and lower birth weight. However, the relationship between these factors is unclear. In this population-based study, **Neilson *et al.*** (pages 536–544) found that individuals with a higher genetic risk for schizophrenia have lower cortical thickness. No interactions with birth weight were found. These findings suggest that lower cortical thickness may be partly influenced by genetic variants rather than by downstream effects of the disorder.

Brain Structure in Bipolar and Schizophrenia Relatives

Schizophrenia and bipolar disorder are both highly heritable and genetically correlated. In this family cohort study, **de Zwarte *et al.*** (pages 545–556) investigated structural brain abnormalities of first-degree relatives of patients with schizophrenia and bipolar disorder. The results revealed distinct disorder-specific patterns of brain abnormalities in relatives compared with control subjects, with increased intracranial volume in bipolar disorder relatives and smaller thalamic volume in schizophrenia relatives. These data suggest that brain abnormalities associated with familial risk may be disease specific.

Visual Processing Deficits in Schizophrenia and Autism

An important component of social functioning is the ability to rapidly and accurately perceive and respond to facial expressions, which is impaired in individuals with schizophrenia and autism spectrum disorder. In both populations, these deficits have been linked to atypical activity within the visual cortex. Using multimodal imaging to investigate visual processing patterns in these disorders, **Martínez *et al.*** (pages 557–567) found impaired face emotion recognition in both groups, but distinctly different underlying patterns of visual processing dysfunction. Patients with schizophrenia showed reduced sensory activation, whereas individuals with autism spectrum disorder showed increased sensory-driven responses, suggesting that these deficits are driven by differential underlying pathophysiological mechanisms.

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