In This Issue

Volume 4, Number 11, November 2019

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

Brain Development: Effects of Maternal Depression and Fluoxetine

Prenatal maternal depression has long-term impacts on brain development and other outcomes. Using structural magnetic resonance imaging (MRI) in a longitudinal birth cohort, **Lee *et al.*** (pages 940–947) discovered that girls, but not boys, exposed prenatally to high maternal depressive symptoms show disrupted covariance between amygdala volume and cortical thickness at birth and at 4.5 years of age. These findings suggest that amygdala-cortical circuitry may be vulnerable to environmental factors in a sex-dependent manner.

Selective serotonin reuptake inhibitors are increasingly prescribed to children, but long-term effects on brain development and brain function are not known. **Golub *et al.*** (pages 948–955) assessed serotonin transporter binding in young male nonhuman primates using positron emission tomography 1 year after discontinuation of a 2-year course of fluoxetine or vehicle treatment. The authors found a fluoxetine × *MAOA* genotype interaction, whereby binding potentials were lower in monkeys with the low transcription polymorphism. These results suggest that *MAOA* genotype may be an important consideration when treating children with fluoxetine.

Striatal Impairments in Psychotic and Mood Disorders

Striatal abnormalities have been implicated in schizophrenia, but connectivity studies have reported mixed results. Here, **Karcher *et al.*** (pages 956–965) aimed to clarify the pattern of connectivity between the cortex and striatum in schizophrenia and whether such impairments extend transdiagnostically to psychotic bipolar disorder. Analyses revealed that both patient groups, relative to control subjects, showed reduced connectivity of the salience network with the striatum, and also reduced connectivity between the putamen and medial prefrontal cortex. These data suggest that corticostriatal dysconnectivity may represent a common neurobiology underlying psychosis, and also reveal that salience network connectivity may play an important role in psychotic disorders.

Stress promotes recurrence of mood episodes in bipolar disorder, but the neurobiological mechanisms through which this occurs are not clear. **van Leeuwen *et al.*** (pages 966–974) found reduced activity in the striatum to a functional MRI (fMRI) reward task after acute stress in patients with bipolar disorder, compared with healthy control subjects. These data suggest that altered reward processing in the striatum during stress recovery may contribute to stress-induced mood episodes in individuals with bipolar disorder.

Emotion Regulation: Stress Responding and Conduct Disorder

Impaired emotion control, impaired prefrontal cortex functioning, and dysregulated reactivity of the body's major stress systems are all implicated in stress-related disorders, but direct evidence that links these findings is lacking. In this study of healthy subjects who completed an fMRI task followed by a stress-induction procedure, **Kaldewaij *et al.*** (pages 975–983) show that the ability to recruit prefrontal brain regions during emotion regulation predicts cortisol responses to acute social stress, with reduced prefrontal cortex activation associated with greater cortisol increases. These findings provide insight into the link between emotion regulation and biological stress responsiveness.

Conduct disorder is linked to deficits in emotion processing and regulation, but the underlying neural correlates remain unclear. Using fMRI, **Raschle *et al.*** (pages 984–994) demonstrate that female adolescents with conduct disorder show reduced prefrontal brain activity and weaker functional connectivity during effortful emotion regulation compared with female control adolescents. These results provide insight into the behavioral deficits associated with conduct disorder and suggest that targeted interventions to improve emotion regulation in conduct disorder should be investigated.

Loss Insensitivity in Anorexia Nervosa

Patients with anorexia nervosa show aberrations in choice behavior, including impairments in laboratory measures of value-based decision making. In this work, **Verharen *et al.*** (pages 995–1003) used computational modeling to demonstrate a mechanism that may underlie this impairment. Specifically, the authors found that compared with control participants, patients with anorexia nervosa show a reduced sensitivity to monetary losses in a gambling task. They then replicated this finding in a second independent cohort. These findings indicate that insensitivity to losses may be a driving factor of impaired decision-making behavior in anorexia nervosa, which could potentially be targeted therapeutically.

Habituation in Early Psychosis

Memory impairment in patients with chronic schizophrenia has been associated with failed hippocampal habituation, which is the natural reduction in neural activity over time to a repeatedly encountered stimulus. Using fMRI, **Avery *et al.*** (pages 1004–1012) found an association between faster habituation in the anterior hippocampus and better relational memory ability in healthy control participants, but this brain-behavior relationship was not present in patients with early psychosis. These results suggest that impaired habituation may be present early in psychosis and may be a mechanism underlying relational memory deficits.

ISSN: 2451-9022