# In This Issue

# Volume 4, Number 3, March 2019

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

## Meta-analysis: Negative Affect in Psychopathology

Negative affect is prominent in borderline personality disorder, major depressive disorder, and posttraumatic stress disorder, which may reflect shared neural abnormalities in affective processing. This meta-analysis by **Schulze et al.** (pages 220–232) investigated functional brain abnormalities in patients with affective disturbances across these diagnostic categories. They found limbic hyperactivation in patients with borderline personality disorder and posttraumatic stress disorder, compared to control subjects. Patients with major depressive disorder showed blunted amygdala activation compared with control subjects. They also observed enhanced activations of the median cingulate gyrus across all disorders, in addition to the abnormalities that were unique to each disorder. These results highlight the importance of both transdiagnostic and disorder-specific approaches to advance our understanding of affective disturbances in psychopathology.

## White Matter Pathways: ADHD and Cognition

Findings from diffusion tensor imaging studies of attention-deficit/hyperactivity disorder (ADHD) have been inconsistent. In this study, **Bessette and Stevens** (pages 233–242) sought to assess whether ADHD white matter abnormalities are more clearly related to one of three distinct impulsivity-related factors (motor inhibition, impulsive choice, and delay aversion) rather than the ADHD diagnostic phenotype. They found that adolescents with ADHD, compared with non-ADHD adolescents, show a disrupted relationship between delay aversion and widespread white matter tracts. These results provide further evidence that the various etiological pathways of ADHD may have distinct neurobiological features.

Structural connectivity of the brain is an important determinant of human behavior, including symptoms of psychiatric disorders and cognitive functioning, but whether integrity of white matter bundles is linked to a genetic predisposition for neuropsychiatric traits remains unclear. In this study of 10-year-old children, **Jansen et al.** (pages 243–250) observed a positive association between global fractional anisotropy and genetic predisposition to intelligence and educational attainment. There was no association, however, between white matter microstructure and polygenic scores for any of the five psychiatric disorders. These data suggest that there may be a shared genetic etiology among white matter integrity, cognitive functioning, and later educational achievement.

## Dysconnectivity in Autism

Cognitive impairment is common in autism spectrum disorder (ASD), but most research into resting-state functional network connectivity in children with ASD has been conducted in those with average IQ. Here, **Reiter et al.** (pages 251–259) compared functional connectivity in lower-IQ children with ASD, higher-IQ children with ASD, and typically developing children. The lower-functioning ASD group showed reduced connectivity within the default mode network and the ventral visual stream. This pattern of atypical connectivity was distinct from the higher-functioning ASD group, which suggests that future connectivity studies in ASD should stratify by general level of functioning.

Resting-state functional magnetic resonance imaging (fMRI)-based research in ASD has primarily focused on altered functional connectivity within networks. Here, **Oldehinkel et al.** (pages 260–270) used resting-state fMRI data to comprehensively investigate functional connectivity within and between 20 resting-state networks. They demonstrate that connectivity between multiple visual, motor, and cerebellar networks is altered in individuals with ASD compared with control subjects. These alterations may underlie the impairments in multisensory and visual-motor integration that are frequently observed in ASD.

## Unconscious Processing of Loss

The loss processing that occurs during grief helps bereaved individuals adapt to the loss, but evidence suggests that the effectiveness of this processing may be affected by whether it occurs consciously or unconsciously. Using fMRI and machine learning in bereaved subjects, **Schneck et al.** (pages 271–279) identified self-generated unconscious loss processing through a neural pattern for deceased-related attention that occurred in the absence of conscious thoughts of loss. Subjects with a greater tendency to process their loss in this naturalistic, unconscious manner showed reduced grief severity, suggesting that unconscious loss processing may represent a coping strategy that promotes recovery from the loss.

## Altered Learning in Schizophrenia and Addiction

People with schizophrenia often display motivational deficits. Using computational modeling and a reinforcement learning task, **Hernaus et al.** (pages 280–290) show that compared with healthy volunteers, people with schizophrenia were driven by an increased reliance on how often a reward is presented (probability), at the cost of learning about the size of the reward (magnitude). Their computational model suggests that such reward integration deficits are linked to an inability to precisely learn about value.

Both behavioral responses and dopamine signaling adaptations are sensitive to drug state (use vs. deprivation) in individuals with substance dependence. Using a computational framework in cocaine-dependent participants, **Wang et al.** (pages 291–299)

© 2019 Society of Biological Psychiatry.   **211**

ISSN: 2451-9022          Biological Psychiatry: Cognitive Neuroscience and Neuroimaging March 2019; 4:211–212 www.sobp.org/BPCNNI

show that cocaine deprivation heightens behavioral and neural learning signals during successful avoidance of negative outcomes. This deprivation-enhanced loss avoidance learning signal was associated with longer history of drug use and greater desire to use cocaine. These data suggest that drug state may also play a role in reinforcement learning, whereby increased learning during drug deprivation reinforces drug seeking, leading to continued substance use in addiction.

### The Hippocampus and Electroconvulsive Therapy Response

The hippocampus is widely implicated in depression, and hippocampal volume increases are commonly observed in patients treated with electroconvulsive therapy (ECT). In this study, **Kubicki et al.** (pages 300–309) used diffusion MRI and probabilistic tractography to establish whether changes in hippocampal structural connectivity also occur in association with ECT and relate to clinical response. Their findings revealed ECT effects in right hippocampal structural connectivity that also differentiated treatment responders and nonresponders. These data suggest that changes in right hippocampus structural connectivity may serve as a biomarker of antidepressant response to ECT.

### Systemic and Cognitive Cortisol Sensitivity in Depression

Cortisol insensitivity is a biomarker of depression. In this study of premenopausal women with varying levels of depression, **Gaffey et al.** (pages 310–320) report that glucocorticoid (GC) insensitivity is associated with more negatively biased memory. Further, they found that pharmacologically induced cortisol elevation alleviated negatively biased memory formation, particularly in women exhibiting systemic GC insensitivity. These results suggest a link between GC insensitivity and depression-related emotional cognition and suggest that treatments aimed at boosting the cortisol signal in depressed individuals with GC insensitivity may be beneficial.