# In This Issue

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

### Neural Correlates of Behavior and Emotion Problems

Regulatory problems (RPs) with crying, feeding, and/or sleeping during infancy are associated with childhood behavioral and emotional problems, but it is unclear whether these persist into adulthood. Using behavioral and resting-state functional magnetic resonance imaging (fMRI) data, **Bäuml *et al.*** (pages 333–342) provide evidence that infant RPs impact behavioral and brain functioning in adulthood. Specifically, individuals with a history of infant RPs, compared to those without, showed more total problems, more internalizing problems, and more avoidant personality traits in adulthood, which were mediated by functional connectivity (FC) decreases in the default mode network. These results suggest that the default mode network may be a target for intervention in individuals with RP-related behavioral and emotional problems.

Attention-deficit/hyperactivity disorder (ADHD) symptoms often co-occur in autism spectrum disorder (ASD) and are associated with worse daily functioning, but knowledge of the underlying neurobiology of ADHD symptoms in ASD is limited. **Yerys *et al.*** (pages 343–351) compared resting-state FC in executive brain networks and found that compared with typically developing children, children with ASD have weaker FC within and between the frontoparietal and salience/ventral attention subnetworks. Decreased FC correlated with ADHD symptoms. These findings provide insight into the heterogeneity of ASD and may help advance the identification of more homogeneous ASD subgroups to optimize treatments.

### Emotion Processing Across Psychopathology

Obsessive-compulsive disorder (OCD) is highly heritable, and diagnosed persons often show difficulty with emotion regulation, which may be an endophenotype for the disorder. **Thorsen *et al.*** (pages 352–360) investigated this hypothesis in patients with OCD, unaffected siblings, and healthy control participants, all of whom completed an emotion regulation task during fMRI. The authors found that distress levels were elevated in patients, compared with unaffected siblings and healthy participants. Patients had higher amygdala and dorsomedial prefrontal cortex activation during OCD-related emotion provocation and regulation. Unaffected siblings showed distinct temporo-occipital activation compared to patients and healthy participants, which may represent a compensatory mechanism. These data suggest that emotion regulation is not a strong endophenotype for OCD.

Emotion-elicited amygdala reactivity has been identified as a neural marker that can be used to differentiate bipolar disorder (BD) from major depressive disorder (MDD), but it is unclear whether this distinction remains present in the absence of an acutely depressed mood. Using emotion processing fMRI tasks, **Korgaonkar *et al.*** (pages 361–370) found that relative to remitted patients with MDD, remitted patients with BD showed left amygdala hypoactivation, lower connectivity to insula and hippocampus for threat emotions, and greater connectivity to insula for sad emotions. The pattern of amygdala activation and connectivity distinguished BD patients from MDD patients, providing evidence for a disorder-specific trait marker that is independent of illness state.

Early-life trauma has been associated with hyperactivation of the salience network (SN) during negative emotion processing. However, hypoactivation of the SN during reinforcement learning has been identified in trauma-exposed youths. Here, **Cisler *et al.*** (pages 371–380) directly compared the impact of early-life trauma exposure on SN engagement during threat processing versus reinforcement learning in adolescent girls with and without histories of assault. The authors found that SN responses to facial threat signals increased, whereas SN responses to negative prediction errors decreased, relative to the severity of trauma exposure. These data suggest that early-life trauma may confer heightened risk for posttraumatic stress disorder (PTSD) via differential dysfunction of the SN.

Some evidence suggests that increased neurocognitive processing during trauma (i.e., peritraumatic processing) may contribute to development of PTSD. Cumulative lifetime adversity has also been proposed to play a role. **Rattel *et al.*** (pages 381–389) played aversive or neutral film scenes for healthy women during fMRI. They found that increased peritraumatic processing in multiple SN regions (amygdala, anterior insula, dorsal and rostral anterior cingulate cortices) predicted later memory intrusions, but only in women who reported more than five lifetime adversities. This interaction model to predict PTSD-like symptoms may help guide identification of at-risk individuals following traumatic incidents.

### Prefrontal and Insula FC: Subregional Distinctions

Prefrontal subregions are differentially implicated in the pathophysiology of PTSD. In this study, **Olson *et al.*** (pages 390–398) report that relative to healthy control and trauma-exposed control subjects, individuals with PTSD show increased negative resting-state FC between the dorsolateral prefrontal cortex and the precuneus. This increased connectivity was associated with overall symptom severity. These data provide insight into the subregional prefrontal differences present in PTSD and also implicate the central executive network in PTSD.

The insula consistently shows aberrant connectivity patterns in psychotic disorders, but most research has analyzed it as a single region. Here, **Tian *et al.*** (pages 399–408) investigated resting-state FC diversity of the insular cortex in individuals with schizophrenia and healthy comparison subjects by using voxel-level fMRI data to parcellate the insula into two distinct subregions. The anterior and posterior subregions of the insula showed marked differentiation in their respective regional network interactions. This anterior-posterior differentiation was reduced in schizophrenia and explained interindividual variation in symptom severity. These findings indicate that connectional diversity across the insula is reduced in schizophrenia.

 

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