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

Neuroimaging Investigations of Cognitive Impairment, Anxiety, and Aggression

The adaptive use of negative self-beliefs is a key feature of psychological flexibility, but this capacity is impaired in individuals with social anxiety disorder. **Goldin *et al.*** (pages 1091–1099) found that, when using cognitive reappraisal, adults diagnosed with social anxiety disorder reported greater negative emotion experience and had delayed onset of cognitive regulation-related brain systems, as compared to healthy controls.

The cerebellum may be subject to experience-dependent changes in structure. Using imaging to examine the role of early experience on cerebellar development, **Bauer *et al.*** (pages 1100–1106) tested previously neglected and typically developing children. They found that the neglected children had smaller volume of the superior-posterior lobe and that this brain region appears related to patterns of cognitive problems observed in these children.

Using functional magnetic resonance imaging (fMRI), **Milad *et al.*** (pages 1075–1082) studied fear conditioning and extinction in posttraumatic stress disorder (PTSD) patients and a matched comparison group. During extinction, PTSD patients showed higher levels of fear that was associated with reduced activation of the ventromedial prefrontal cortex and the hippocampus, brain regions involved in fear extinction learning. These data support the long-standing hypothesis that exaggerated fear responses commonly observed in PTSD patients may be due to failure to activate these critical brain regions.

PTSD may be associated with dysfunctional reward processing. **Elman *et al.*** (pages 1083–1090) administered a monetary reward task during fMRI in individuals with chronic PTSD and healthy controls. The PTSD subjects showed smaller bilateral striatal activations and less striatal activation to gains versus losses, which was associated with more self-reported motivational and social deficits. The findings suggest that reward dysfunction may be an important treatment target for PTSD.

Borderline personality disorder is often associated with impulsive aggression. Using positron emission tomography, **New *et al.*** (pages 1107–1114) found that borderline personality disorder patients with prominent aggressive traits responded more aggressively than controls, both when provoked and when not provoked. When not provoked, patients showed lower brain metabolism in orbital cortex and amygdala. However, with provocation, patients showed heightened metabolic response in orbital cortex and amygdala, but not in dorsal brain regions associated with aggression modulation. In contrast, controls showed increased brain metabolism in dorsal regions of prefrontal cortex during provocation.

Zitterl *et al.* (pages 1115–1122) investigated whether a pre-treatment measurement of central serotonin transporter (SERT) availability was related to treatment response to sertraline in patients with obsessive-compulsive disorder (OCD). Using single photon emission computed tomography to measure thalamic-hypothalamic SERT availability in OCD patients, they found that higher pretreatment SERT availability significantly predicted higher occupancy rates as well as better treatment response after 14 weeks. Furthermore, a positive association was found between transporter occupancy and treatment response directly.

Genetic Analysis of Bipolar Disorder and Borderline Personality Disorder

De Mooij-van Malsen *et al.* (pages 1123–1130) performed a genetic mapping study for mouse avoidance behavior using automated home cage behavioral observations. They identified a genetic locus on mouse chromosome 15 that is homologous to a human genome region (8q24) linked to bipolar disorder. Integration of the mouse locus with genotypes of bipolar disorder cases versus controls revealed two associated genes, *ADCY8* and *KCNQ3*, with additional analyses suggesting that *Adcy8* may encode a translational behavioral endophenotype of bipolar disorder.

Using data of twins and their siblings, **Distel *et al.*** (pages 1131–1138) investigated the clinical and genetic association between the five factor model of personality traits and borderline personality disorder. A combination of high neuroticism and low agreeableness best predicted the borderline personality score. Multivariate genetic analysis showed that borderline personality shares genetic variation with neuroticism, agreeableness, conscientiousness and extraversion.

Role of Calcineurin Activity in Mood Disorders

Cyclosporin-A is a suppressor of the immune system that is commonly prescribed to patients to prevent their rejection of transplanted organs. However, this compound may also suppress calcineurin, a neural protein, resulting in psychiatric complications. **Bahi *et al.*** (pages 1139–1146) provide new evidence that calcineurin inhibition either by cyclosporin-A treatment or by local knockdown of the protein in the basolateral amygdala induces depression- and anxiety-like behaviors in mice. These data demonstrate that calcineurin activity in the amygdala plays an important role in the signaling events that trigger mood abnormalities and suggest that transplant patients would benefit from screening for mood disorders.

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