In This Issue

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

Vasopressin Boosts Placebo Effects in Women

Colloca *et al.* (pages 794–802) conducted a randomized, double-blind trial in healthy participants to study the placebo effects of vasopressin, compared with no treatment, oxytocin, and saline, in a model of placebo analgesia. They found that a nonselective vasopressin 1A/1B receptor agonist boosts placebo effects, but only in women. Women with lower anxiety and cortisol levels showed the largest vasopressin-induced modulation of placebo effects. These findings have implications for the therapeutic potential of vasopressin and shed new light on the neurobiological aspects of the placebo phenomenon.

Mechanisms of Stress Exposure: Neuroinflammation and Serotonergic Activity

Chronic psychological stress alters immune system functioning and is associated with poor mental health outcomes. Recent data have shown that mice exposed to repeated social defeat (RSD) exhibit monocyte trafficking and anxiety-like behavior when later exposed to subthreshold stress. Here, **McKim *et al.*** (pages 803–813) report that splenectomy before RSD prevented the immunological and behavioral responses following later subthreshold stress but had no effect on monocyte accumulation or anxiety immediately after RSD. Collectively, these data demonstrate that the long-term storage of primed monocytes in the spleen may play a role in the maintenance of stress sensitization and recurring anxiety disorders.

Prior exposure to stress is a risk factor for developing posttraumatic stress disorder in response to trauma. Investigating the mechanisms by which this occurs, **Baratta *et al.*** (pages 814–822) found that fear memory consolidation in mice exposed to repeated stress requires serotonergic activity in both the amygdala and dorsal raphe nucleus. These processes did not affect fear memory in unstressed mice. These data indicate that prior stress experiences augment later fear learning by engaging distinct serotonergic pathways and suggest that amygdala serotonin 2C receptors may be a promising therapeutic target for psychiatric disorders characterized by excessive fear responses.

Brain Stimulation Reduces Threat Vigilance

Noninvasive forms of brain stimulation, such as transcranial direct current stimulation (tDCS), have shown promise as treatments for mood disorders. **Ironside *et al.*** (pages 823–830) investigated the effects of dorsolateral prefrontal cortex-targeted tDCS in healthy volunteers who then completed emotional processing tasks. Compared to those who received sham stimulation, volunteers who received tDCS showed reduced fear vigilance, similar to what has been observed with anxiolytic treatments in the same cognitive paradigm. This tDCS-induced alteration in the cognitive processing of threatening information may have therapeutic effects in clinical populations.

Effects of Trauma Transmitted During Pregnancy

The effects of trauma exposure are transmitted across generations, but the timing and mechanisms remain unclear. **Moog *et al.*** (pages 831–839) investigated intergenerational transmission by assessing the impact of a woman's exposure to trauma as a child on fetal development during a woman's pregnancy. They found that maternal childhood trauma exposure is associated with increased placental corticotropin-releasing hormone, the primary placental-fetal stress hormone, compared with nonexposed women. These data indicate that intergenerational transmission of trauma-related effects may occur as early as intrauterine life.

Brain Structure and Stress Resilience

Anacker *et al.* (pages 840–849) investigated changes in brain anatomy of mice that were either susceptible or resilient to the effects of chronic social stress. They found structural differences and corresponding volume changes between brain regions, including the ventral tegmental area, cingulate cortex, hippocampus, and hypothalamus. Their results suggest that neuroanatomical differences in brain regions that integrate stressful information contribute to individual differences in stress susceptibility and resilience.

Affective Disorders and Genetic Effects

The *FMR1* premutation is associated with elevated rates of mood and anxiety disorders, but longitudinal data regarding this vulnerability has been lacking. **Roberts *et al.*** (pages 850–857) followed a sample of women with the *FMR1* premutation to characterize the stability and predictors of mood and anxiety disorders across a 3-year period. Their findings revealed an increased prevalence of major depressive disorder and anxiety disorders over time. CGG repeat length, child problem behaviors, and divorced marital status, but not primary ovarian insufficiency, were associated with elevated risk for psychiatric diagnoses.

The endocannabinoid system is an important modulator of affective behaviors and stress responses in animals and humans. Here, **Jenniches *et al.*** (pages 858–868) report that the genetic deletion of diacylglycerol lipase α, an endocannabinoid-producing enzyme, in mice results in enhanced stress-related behaviors and fear responses. These findings are consistent with the idea that disrupted endocannabinoid signaling contributes to the development of affective disorders.

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