In This Issue-May 1st

Can we become addicted to tasty food? **Teegarden and Bale** (pages 1021–1029) show that mice withdrawn from their preferred high fat diet and placed on a healthier, but less desirable diet start to exhibit more stress-related behaviors. Further, they show neurobiological changes in limbic reward and emotion centers that have been reported previously in animal models of addiction, including increases in the levels of the phosphoproteins, pCREB and deltaFosB. They also were willing to expose themselves to risky environments in order to gain access to the fatty food. These data highlight the addictive quality of high fat diets, raising important questions about the way we think about obesity and its treatment.

Endogenous opiate systems are activated by stress, but their involvement in resilience and post-traumatic stress disorder (PTSD) is not well understood. In this article, **Liberzon *et al.*** (pages 1030–1038) report reductions in the density of μ- opiate receptor binding in limbic and cortical brain regions and increases in binding in the orbital frontal cortex (OFC) in PTSD patients and combat-exposed veterans without PTSD compared to healthy comparison subjects, as assessed using [^{11}C]carfentanil and positron emission tomography (PET). The pattern of reduced μ-opiate receptor density in the amygdala and increased receptor density in the OFC was exaggerated in the veterans without PTSD compared to the PTSD veterans and healthy controls. The finding that endogenous systems are altered following extreme stress response, even in resilient individuals, raises the possibility that these systems play an important persisting role in coping.

McElroy *et al.* (pages 1039–1048) report that topiramate, an anticonvulsant and antimigraine medication that can produce weight loss, was significantly superior to placebo in reducing binge eating, weight, obsessive and compulsive symptoms, and disability among 394 patients with binge eating disorder and obesity. In this double-blind study, approximately 30% of the patients in each group discontinued study medications prior to the end of the study.

Paul *et al.* (pages 1049–1061) identify an important molecular "switch" in the transition from short-term to long-term forms of learned fear responses. The tyrosine phosphatase STEP is a brain-specific protein that regulates the activity of key signaling proteins implicated in neural mechanisms underlying learning. Here, the authors inactivated STEP by infusing a dominant-negative STEP protein into the lateral amygdala, a brain region implicated in fear conditioning, i.e., the learning of danger cues. Inactivation of STEP allowed rats to acquire short-term fear conditioning, but blocked the consolidation of this learning into long-term fear conditioning.

De Geus *et al.* (pages 1062–1071) report the results of a creative effort to understand the interaction of genetic and environmental factors that influence brain development upon the development of temperament. They used structural magnetic resonance imaging (MRI) to study 64 monozygotic twins selected from over 3300 Dutch twin pairs to be highly concordant or discordant for anxiety and depression. In these twins, reduced temporal lobe volume was observed in high-risk twins, most notably in the left posterior hippocampal region. The volume deficits were entirely attributable to environmental risk factors. In contrast to a recent report in post-traumatic stress disorder, genetic risk factors were not related to temporal lobe volume.

Symptoms of obsessive-compulsive disorder (OCD) are exaggerations of behaviors that are commonly observed. **Lawrence *et al.*** (pages 1072–1080) present new functional MRI data linking an exaggerated activation of ventrolateral prefrontal cortex in response to viewing pictures with facial expressions of disgust, but not to those of fear, in OCD patients with contamination-related obsessions and compulsions.

The serotonin-1A ($5HT_{1A}$) receptor is a target for the treatment of anxiety disorders and may be abnormally regulated in anxiety disorders. Using positron emission tomography and the selective radioligand for this receptor, **Lanzenberger *et al.*** (pages 1081–1089) found reduced $5HT_{1A}$ receptor density in patients with social phobia in brain regions implicated in anxiety, including the amygdala and cingulate gyrus.

In contrast to reports on panic disorder and social phobia (see Lanzenberger *et al.* in this issue), anorexia nervosa may be associated with increased density of the serotonin-1A receptor. **Bailer *et al.*** (pages 1090–1099) report this interesting finding based on their study employing positron emission tomography. Future research will be needed to determine whether this finding is related to the extent to which these patients respond to antidepressant treatments.

Versions of the monoamine oxidase-A (MAOA) gene have been associated with aggression. In their report, **Eisenberger *et al.*** (pages 1100–1108) now report that the genotype associated with aggression is also associated with increased subjective distress and greater activation of the anterior cingulate cortex, a region of the limbic system, in response to social exclusion. These data suggest that heightened reactivity to negative social events might link the risk genotype to aggressive behavior.

Early childhood adversity can have long-lasting effects on social function and stress reactivity. **Meinlschmidt and Heim** (pages 1109–1111) report blunted cortisol responses to one hormone implicated in both processes, oxytocin, in men who underwent parental separation as children. This finding raises interesting questions about the origin and functional implications of altered oxytocin response.

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