IN THIS ISSUE

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

Examining the Biology of Suicide

Ernst *et al.* (pages 312–319) report reduced expression of two proteins that enable astrocytes to establish gap junctions, the connexins Cx30 and Cx43, in dorsal lateral prefrontal cortex of suicide completers compared to sudden death controls. Using complementary techniques, they also show that the transcription factor Sox9 affects expression of Cx30. These results implicate connexin genes in the neurobiology of suicide and further implicate astrocytes in psychopathology.

Corpus callosum (CC) size has been associated with cognitive and emotional deficits in a range of neuropsychiatric and mood disorders. **Cyprien *et al.*** (pages 320–326) used magnetic resonance imaging (MRI) in a cohort of community-dwelling elderly persons to examine whether there is an association between CC atrophy and suicidal behavior. They found that the posterior third of the CC was smaller in suicide attempters, suggesting a diminished inter-hemispheric connectivity and a possible role of CC in the pathophysiology of suicidal behavior.

Neuroimaging in Depression: Cognition, Emotion, and Risk

Ruminations are important symptoms of major depressive disorder (MDD). They consist of both passive/maladaptive and active/adaptive components. Using functional MRI (fMRI), **Hamilton *et al.*** (pages 327–333) found that, in MDD patients, increasing levels of activity in the default-mode network (involved in passive, self-relational processing) relative to the task-positive network (involved in active cognitive processing) are associated with higher levels of maladaptive, depressive rumination and lower levels of adaptive, reflective rumination. These findings suggest that relative activity levels in these networks represent neural substrates of rumination in MDD.

Very little is known about topological patterns of whole-brain functional networks in MDD. Using resting-state fMRI and graph theory-based approaches, **Zhang *et al.*** (pages 334–342) report abnormal small-world efficiency in whole-brain functional networks in MDD, with the most pronounced changes in the caudate nucleus and default-mode regions. The results suggest that depression is a disorder associated with disrupted neuronal network organization that likely underlies deficient cognitive and mood processing.

Whalley *et al.* (pages 343–349) compared performance on a language task during fMRI in young unaffected relatives of bipolar disorder (BD) patients versus healthy controls. The high-risk group showed increased activation in the left amygdala, an important region for emotion regulation. Across groups, they also found associations between depression and measures of cyclothymia. These results provide evidence for the neural basis of increased genetic risk for BD as well as for subclinical features.

BD is associated with reductions in white matter integrity and carries high genetic risk. Here, **Sprooten *et al.*** (pages 350–356) report that unaffected relatives also show widespread white matter integrity reductions, and further, that cyclothymic temperament, an affective trait thought to underlie the bipolar spectrum, is associated with more localized reductions in both relatives and healthy controls. These findings suggest that white matter integrity is an endophenotype for BD with important behavioral associations previously linked to the etiology of the condition.

Individuals with BD exhibit difficulties in decision-making, but it is unknown if these deficits are related to illness phase. Using a laboratory test, **Adida *et al.*** (pages 357–365) found similar decision-making impairments in three groups of BD individuals, manic, depressed and euthymic, compared to healthy controls. Decision-making impairment was significantly predicted by low education level, high depressive scores, family history of BD, use of benzodiazepines, and nonuse of serotonin and norepinephrine reuptake inhibitors.

Patients remitted from depression remain at high risk for relapse. **Farb *et al.*** (pages 366–372) used fMRI to compare neural reactivity to sadness in remitted patients who relapsed and those who sustained remission over an 18-month follow-up period. Frontal cortical reactivity predicted future relapse, while visual cortical reactivity predicted sustained remission. These finding highlight the role of the executive control of emotion in depressive relapse.

Reduced brain activity to pleasure and rewards may be an important mechanism of postpartum depression. **Moses-Kolko *et al.*** (pages 395–399) report that, relative to healthy mothers, depressed mothers display a briefer period of brain activity to winning money, adding to our understanding of altered brain activity during emotion processing in postpartum depression.

Brain Volume: Associations with Depression, Cortisol, and Obesity

Gerritsen *et al.* (pages 373–380) examined whether hypothalamic-pituitary-adrenal (HPA) axis dysfunction may be involved in the structural brain changes often found in MDD. In individuals with a history of atherosclerotic disease, they found that having past or current MDD was associated with smaller hippocampal volumes, but not entorhinal cortex volumes. In addition, age of depression onset was differentially associated with hippocampal and entorhinal cortex volumes. None of the findings were explained by HPA axis regulation, as measured by cortisol levels.

Here, **Bond *et al.*** (pages 381–387) report unique brain changes associated with elevated body mass index (BMI) in first-episode mania patients with BD. Patients with increased BMI had reduced volume of white matter and temporal lobes, areas of known vulnerability in early BD. Healthy subjects with elevated BMI had reduced total brain and gray matter volumes, consistent with previous general population studies. These findings suggest a mechanism underlying the association of obesity with a more severe illness course in BD.

Electroencephalographic Alpha: Predicting Antidepressant Response

Previous evidence has suggested that noninvasive and cost-effective electroencephalographic measures may be of value for predicting clinical response to antidepressants. Using high-resolution methods to classify the strength and pattern of brain electrical activity, **Tenke *et al.*** (pages 388–394) report that depressed patients with a prominent alpha rhythm can be confidently predicted to be responsive to serotonin reuptake inhibitors. These results provide additional support for the potential use of simple, objective measures as a predictor of antidepressant treatment response.

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