Neumeister et al (p 765) have studied unmedicated recovered patients with depression and healthy controls using tryptophan depletion and positron emission tomography. They show that tryptophan depletion, which induces a transient decrease in brain serotonin function, unmasks abnormal regional glucose metabolism in a circuit including the orbitofrontal cortex, medial thalamus, anterior and posterior cingulated cortices, and the ventral striatum. The results suggest a trait abnormality in depression.

Brown et al (p 774) examined whether serologically documented prenatal exposure to influenza was associated with an increased risk of schizophrenia and other schizophrenia spectrum disorders in a large birth cohort with archived maternal serum samples. The risk of schizophrenia and spectrum disorders was increased 3-fold for influenza exposure in early to mid-pregnancy and was increased 7-fold for influenza in the first trimester. These findings represent the first serologic evidence that prenatal influenza is associated with schizophrenia.

Chang et al (p 781) used functional magnetic resonance imaging to determine whole brain activation patterns in children with euthymia with familial bipolar disorder compared with controls when performing both a visuospatial working memory task and a task involving watching valenced emotional stimuli. Both tasks revealed predominant overactivation in the prefrontal and subcortical limbic areas. These findings support a model of prefrontal-subcortical dysregulation, whether elicited by cognitive or emotional tasks, as a trait finding in pediatric bipolar disorder.

Dougherty et al (p 795) used an emotion-induction paradigm in conjunction with positron emission tomography to measure regional cerebral blood flow changes during anger induction in patients with major depression with or without anger attacks and healthy volunteers. Patients with major depression with anger attacks exhibited dysfunction in the left ventromedial prefrontal cortex and left amygdala during anger induction, which differentiated them from patients with major depression without anger attacks and healthy volunteers.

Grant et al (p 807) examined the current prevalence and comorbidity of DSM-IV substance use disorders and independent mood and anxiety disorders. Independent mood and anxiety disorders included only those that were not substance-induced or due to a general medical condition. Associations between substance use disorders and mood and anxiety disorders were positive and significant, suggesting that treatment for a comorbid mood or anxiety disorder should not be withheld from individuals with substance use disorders.

Data from animal studies indicate the adrenal steroid dehydroepiandrosterone sulfate (DHEA-S) may diminish the negative impact of stress. Morgan et al (p 819) assessed plasma DHEA-S and cortisol levels, psychological symptoms of dissociation, and military performance in soldiers exposed to acute, uncontrollable stress. Soldiers with greater levels of DHEA-S exhibited fewer stress-induced symptoms of dissociation and superior military performance. These data provide evidence that DHEA-S may have buffered the negative effect of acute stress in healthy subjects.

Miranda et al (p 827) evaluated the effect of evidence-based depression care comparing ethnic minority and nonminority participants who were treated in their primary health care setting. They used instrumental variable techniques to account for selection bias, which occurs because those who have worse depression receive treatment. The depression care was equally effective for lowering rates of depression in minority and nonminority participants and resulted in continued employment for nonminority participants but not for the minority participants.

Prenatal smoking is associated with antisocial behavior in offspring, but commentators have questioned whether this reflects a causal link. Using a twin study, Maughan et al (p 836) found that associations between parental smoking and early childhood behavior problems were heavily confounded with other known risks for child antisocial outcomes, both genetic and environmental. This high level of confounding casts doubt on the causal status of prenatal smoking and argues that conclusive answers must await findings from experimental studies.