In a double-blind, 104-week study, Lieberman et al (page 361) examined brain volume changes in patients with first-episode psychosis treated with olanzapine and haloperidol. These changes were then analyzed for any association between changes in psychopathology and neurocognition. Patients exhibited a significant between-treatment difference in brain volume changes. Haloperidol was associated with significant reductions in gray matter volume, whereas olanzapine was not. Post hoc analyses suggested that antipsychotic treatment effects on brain volume and the behavioral pathology of the illness may be associated.

Hirvonen et al (page 371) studied the neurobiological correlates of increased genetic risk for schizophrenia. Monozygotic and dizygotic healthy co-twins of patients with schizophrenia and matched, healthy twin pairs were scanned with positron emission tomography and carbon 11 (11C)-labeled raclopride. Monozygotic co-twins had significantly higher D2 receptor binding in the caudate nucleus compared with healthy control twins. High caudate D2 receptor binding was associated with low cognitive performance in this sample. This finding suggests that the caudate dopamine D2 receptor up-regulation is related to genetic risk for schizophrenia.

Eyer-Lindenberg et al (page 379) used neuroimaging to study the functional connections of the hippocampus in patients with schizophrenia and normal controls during a working memory and a control condition. They found that patients with schizophrenia had a regional specific abnormality in coupling to the dorsolateral-prefrontal cortex that became apparent only during the working memory challenge, providing in vivo evidence that interactions between these 2 brain structures are specifically abnormal in schizophrenia.

Ofzinger et al (page 387) measured cerebral glucose metabolic changes from waking to non-rapid eye movement (NREM) sleep between patients with depression and healthy controls. Patients showed a small decrease in relative metabolism from waking to NREM sleep in a dorsal emotional neural network including prefrontal cortex. They also showed waking hypermetabolism in a ventral emotional neural network that may mediate emotional arousal including the amygdala and anterior cingulate. Elevated activity in this network persisted into sleep and may be responsible for preventing restorative sleep in depression.

Imaging studies relate major depression to many brain regions. Major depression comprises distinct symptom clusters. Milak et al (page 397) used positron emission tomography to examine symptom clusters and resting cerebral metabolic activity. They show that severity of discrete symptom clusters correlates with distinct brain regions, suggesting that specific brain regions are responsible for specific symptom components.

DeRubeis et al (page 409) compared antidepressant medications (ADMs) with cognitive therapy (CT) in a 2-site, randomized study of 240 patients with moderate to severe depression. After the first 8 weeks, during which time the ADM vs CT comparisons were placebo controlled, no difference was observed in response rates between ADM and CT, and both conditions yielded response rates superior to those observed in the placebo condition. After 16 weeks, the response rate was the same in ADM and CT.

In a companion article, Hollon et al (page 417) tested whether cognitive therapy (CT) has an enduring effect. Patients who responded to CT were withdrawn from treatment and followed up for 2 years; patients who responded to medications either continued taking medications or began taking a pill placebo. Results showed that patients previously treated with CT were no more likely to relapse than patients who continued taking medications, and they were less likely to relapse than patients who stopped taking medications.

Qin et al (page 427) used data from Danish longitudinal population registers and found that the elevated risk associated with hospitalized patients with psychiatric disorders varied significantly according to clinical phases, length of hospital treatment, and the diagnosis. The risk peaked in periods immediately after hospital admission and discharge and was particularly high in persons with affective disorders and in those undergoing short-term hospital treatment.

Uskamp et al (page 435) assessed the effect of copayment increases associated with the adoption of a 3-tier formulary by 1 large employer’s health plan on use and spending patterns for medications used to treat attention-deficit/hyperactivity disorder (ADHD) in children enrolled as dependents. The copayment increases resulted in lower total ADHD medication spending, sizeable increases in out-of-pocket expenditures for families of children with ADHD, and a significant decrease in the probability of using these medications.

Ordahl et al (page 444) measured N-acetylaspartate–creatine and phosphocreatine (NAA/Cr), choline–creatine and phosphocreatine (Cho/Cr), and choline–N-acetylaspartate (Cho/NAA) metabolite levels in the frontostriatal regions of methamphetamine abusers at different points in abstinence. All of the methamphetamine abusers had abnormally low NAA/Cr levels within the anterior cingulum but not in the primary visual cortex regardless of time abstinente. In contrast, Cho/NAA values for the anterior cingulum were abnormally high in the methamphetamine abusers who initiated abstinence more than 1 year prior to study. The relative choline normalization across periods of abstinence suggests that following cessation of methamphetamine abuse, adaptive changes occur that may contribute to some degree of normalization of neuronal structure and function in the anterior cingulum.