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A brief summary of the articles appearing in this issue of *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*.

### Neural Effects of Schizophrenia Cognitive Training

Evidence indicates that behaviorally based training programs can be effective in improving cognition in schizophrenia, but the underlying neural mechanisms remain unclear. In this systematic review, **Mothersill and Donohoe** (pages 688–696) present data on functional magnetic resonance imaging (fMRI) studies that examined brain activation changes in individuals with schizophrenia following cognitive training, with the most frequently reported finding being increased activation of the left prefrontal cortex. The authors then conducted a meta-analysis, which revealed no brain areas that showed consistent effects across studies, perhaps owing to the variety of training programs involved, suggesting that training may have only broad effects on brain activation.

### Brain Connectivity and Sleep Spindle Biomarkers in Schizophrenia

Sensory-motor deficits are common in schizophrenia, but the underlying brain impairments are unclear. Using fMRI, **Zhang et al.** (pages 697–705) found disrupted functional connectivity in the sensory-motor network of first-episode, drug-naïve patients with schizophrenia, compared with control subjects. Further, the authors identified a link between degree of functional connectivity impairment and both duration of untreated psychosis and motor-related symptoms. Functional network connectivity at baseline also predicted improvement of positive symptoms following 2 months of antipsychotic treatment. These data highlight the importance of the sensory-motor network in schizophrenia and suggest that it should be evaluated as a potential neuroimaging biomarker.

Thalamocortical abnormalities have been implicated in schizophrenia, including increased connectivity between the thalamus and the cortex during wakeful rest, and reduced sleep spindle activity during non-REM sleep. Using polysomnography and resting-state fMRI, **Baran et al.** (pages 706–714) replicated these prior findings in patients with schizophrenia. Further, they found that reduced spindle density is correlated with increased thalamocortical connectivity in both patients with schizophrenia and healthy subjects. These data support the hypothesis that these two schizophrenia biomarkers represent a common pathophysiology, suggestive of abnormal thalamic reticular nucleus function.

### Early Neural Markers of Mood Disorder Risk

Depression and other mood disorders often emerge in adolescence, but objective neural markers that can predict risk are lacking. In this study of adolescents, **Kaiser et al.** (pages 715–725) report that dysfunctional frontoinsula network connectivity to an emotional memory task was associated with greater baseline depression, increased depression at a 2-week follow-up, and more intense negative mood in daily life. These data suggest that frontoinsula network functioning in adolescence may predict current and prospective mood health and may therefore hold promise as a neurobiological marker for risk prediction.

Impulsive sensation-seeking behavior and emerging mania symptoms are associated with risk for bipolar spectrum disorder. Using a machine learning approach with fMRI data in a transdiagnostic sample of young adults, **de Oliveira et al.** (pages 726–733) found that a specific manic symptom subdomain, heightened energy, was predicted from patterns of brain activation to reward expectancy. The ventrolateral prefrontal cortex was the region with highest contribution for prediction. These findings may help provide neural biomarkers to aid the early identification of bipolar disorder risk in young adults.

### Childhood Trauma and Brain Morphology

Childhood trauma is a significant risk factor for adult psychopathology and is associated with multiple structural brain changes. In a transdiagnostic community sample, **Clausen et al.** (pages 734–742) used machine learning to identify relationships between regional gray matter volume and childhood trauma severity, independently of adult psychopathology. The model identified numerous gray matter regions that predicted childhood trauma severity, including regions implicated in emotional processing (prefrontal cortex) as well as regions involved in reward, interoceptive, attentional, and sensory processing (e.g., striatal, insular, and parietal/occipital cortices).

### Methylphenidate's Effects on Neural Noise in ADHD

Attention-deficit/hyperactivity disorder (ADHD) is associated with high intraindividual variability, which may suggest increased neural noise. Using electroencephalography to investigate this hypothesis, **Pertermann et al.** (pages 743–750) report that medication-naïve children with ADHD showed increased neural noise compared with healthy control children during an inhibitory task, particularly within the theta band. Following initiation of methylphenidate treatment in the ADHD group, neural noise was reduced and comparable to that of the healthy control group. Improved performance correlated with decreased neural noise. These data provide a mechanistic link for the therapeutic effects of methylphenidate in ADHD.

### Striatal Regions Mediate the Transition to Addiction

Cannabis use does not always lead to addiction, and the neuroadaptations underlying the transition from use to dependence are not clearly understood. In this fMRI study, **Zhou et al.** (pages 751–762) examined drug-cue elicited neural responses in dependent and nondependent cannabis users relative to nonusing control subjects. Whereas both groups demonstrated exaggerated neural cue reactivity in the ventral striatal reward system, dependent users additionally exhibited exaggerated reactivity in the dorsal striatum, a region involved in habit formation. These findings suggest that individual differences in habitual learning mechanisms may promote the transition to cannabis addiction.