Biological Psychiatry

A Journal of Psychiatric Neuroscience and Therapeutics

# In This Issue-June 15th

***Genetic Bases of ADHD***

**Neuman *et al.*** (pages 1320–1328) provide new evidence that in utero exposure to smoking is associated with attention deficit hyperactivity disorder (ADHD) problems in genetically susceptible children. The authors describe that among individuals with versions of the dopamine-4 and dopamine transporter genes that are associated with ADHD, in utero exposure to smoking may increase the risk for ADHD by as much as 9-fold.

**Jain *et al.*** (pages 1329–1339) report evidence that psychiatric disorders that commonly co-occur with ADHD share common genetic bases. The authors used genetic marker information in large families to demonstrate that ADHD is co-transmitted with oppositional defiant disorder, conduct disorder and substance abuse.

***Animal Models in ADHD***

**Blondeau and Dellu-Hagedorn** (pages 1340–1350) investigated new rodent models for subtypes of ADHD, based on behavioral differences between animals in their levels of attention, impulsiveness and motor activity. Four distinct subgroups were demonstrated that parallel those observed in humans: efficient, middle, inattentive and inattentive-impulsive/hyperactive. The study of these subgroups may help to better understand their biological bases and to explain therapeutic effects specific to ADHD subtypes.

Females with Turner Syndrome (TS), a developmental disorder in which one X chromosome is missing or aberrant, exhibit abnormalities in attention as a consequence of reduced X-linked gene dosage. **Davies *et al.*** (pages 1351–1360) show that these abnormalities can be recapitulated in a mouse model of TS, and that they may be alleviated by the addition of a small number of X-linked genes, including steroid sulfatase (*Sts*). These data raise important questions about the role of the X chromosome in attention.

***Neuroimaging in ADHD***

In a meta-analysis combining data from multiple magnetic resonance imaging (MRI) studies of brain structure, **Valera *et al.*** (pages 1361–1369) report reductions in regional brain volumes in children and adolescents with ADHD, including parts of the cerebellum, the corpus callosum, frontal cortex, and basal ganglia.

**Krauel *et al.*** (pages 1370–1379) provide new functional MRI evidence that adolescents with ADHD encode neutral events less efficiently than healthy controls, but have no such problems with emotionally salient events that capture their attention. When encoding neutral stimuli, individuals with ADHD showed reduced activation of the anterior cingulate and superior parietal lobe. However, emotional engagement reduced these group differences.

Using a positron emission tomography (PET) radiotracer with high selectivity for the dopamine transporter, **Spencer *et al.*** (pages 1380–1387) provide new evidence for increased density of this transporter in individuals with ADHD. These findings may be consistent with evidence that a version of the dopamine transporter gene is associated with the risk for ADHD.

***OCD & ADHD***

Obsessive compulsive disorder (OCD) and ADHD occur together in children and adolescents more often than expected by chance. **Geller *et al.*** (pages 1388–1394) used family study methods to assess first-degree relatives of children with OCD with and without ADHD and matched controls. The authors provide new support for a familial link between OCD and ADHD, particularly a pediatric-onset form of OCD with ADHD.

***Neural Circuitry of ADHD***

**Clark *et al.*** (pages 1395–1401) provide new evidence implicating the frontal cortex on the right side of the brain in ADHD. Their data shows that right frontal cortex injury produces deficits in behavioral inhibition and a form of memory that, to some extent, resemble the deficits associated with ADHD. This pattern of cognitive impairments was not observed in individuals who had damage of their left frontal cortex.

0006-3223/07/$32.00