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Volume 66, Number 10, November 15, 2009

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

Molecular Genetics of Neurodevelopmental Alterations in Autism and Tourette Syndrome

Abnormalities in synapse formation have been described in post-mortem brain tissue from individuals with autism. **Daoud *et al.*** (pages 906–910) provide new evidence that a gene that codes for a protein that plays an important role in synapse formation, *NLGN4X*, is involved in the genetics of autism. They identified a *de novo* variation in the promotor region of *NLGN4X* that may be associated with autism. This mutation increased expression levels of the *NLGN4X* transcripts in a patient with autism and nonsyndromic profound mental retardation. This result provides further insight about the syndromes that might be associated with *NLGN4X* mutations and it suggests that analyses of *NLGN4X* expression might detect new cases.

Previous human genetic analysis identified the homeobox gene, *ENGRAILED 2* (*EN2*), an important regulator of central nervous system development, as a possible susceptibility locus for autism. These studies demonstrated certain *EN2* genetic variants are inherited more often in individuals with autism than unaffected siblings. **Benayed *et al.*** (pages 911–917) now demonstrate that these genetic variants are functional, affecting gene expression levels. They also identified a common haplotype as a possible risk allele for autism.

Rare mutations of *SLITRK1* have been associated with Tourette syndrome and other disorders. In this report, **Kajiwara *et al.*** (pages 918–925) show that SLITRK1 undergoes cleavage to release a fragment inside and outside the cell, and that SLITRK1 is phosphorylated by a number of phosphoproteins. The phosphorylation of the intracellular domain of SLITRK1 regulates the relationship with 14-3-3 proteins, and in turn, the effects of SLITRK1 on neurite outgrowth.

Neuroanatomy of ADHD and Autism

Abnormal left-right brain asymmetries in attention-deficit/hyperactivity disorder (ADHD) patients may contribute to ADHD-related cognitive impairments. In a case-control association study, **Ribasés *et al.*** (pages 926–934) selected six functional candidate genes and identified and replicated an association between adult ADHD and the *BAIAP2* gene, which is differentially expressed between cerebral hemispheres.

Examining a key region for reward processes, **Carmona *et al.*** (pages 972–977) found that children with ADHD have a volumetric reduction in the ventral striatum compared to healthy children. Right ventral striatum volume was associated with hyperactive/impulsive symptoms. These results provide neuroanatomical evidence in support of recent models of ADHD, which stress the relevance of reward brain circuits in the pathogenesis of hyperactive/impulsive symptoms.

Focusing on structural brain abnormalities in autism, **Frazier and Hardan** (pages 935–941) quantitatively reviewed 10 previous studies examining the size of the corpus callosum, the largest bundle of fibers connecting the cerebral hemispheres. Results of this meta-analysis show that the corpus callosum is smaller in individuals with autism, indicating reduced connectivity of long-distance neural fibers in the brains of individuals with autism.

Seeking to characterize abnormal neuropathology in autism, **Schumann *et al.*** (pages 942–949) found that the amygdala was enlarged in toddlers who were later diagnosed with autism, compared to typically developing toddlers. The degree of enlargement was associated with the severity of clinical impairment at ~5 years of age. They found that the amygdala was enlarged to a greater extent in girls than in boys, but amygdala volume in boys was more strongly associated with clinical impairment.

Autistic Endophenotype: Immune Dysfunction and Facial Processing

Immunologic dysregulation may be an important feature of autism. To examine this hypothesis, **Saresella *et al.*** (pages 978–984) performed an analysis of immune parameters in children with autism and their healthy, non-autistic siblings. Results showed that a complex immune impairment is present both in autistic children and in their non-autistic siblings, supporting the hypothesis that immunologic dysregulation may be a heritable component of the genetic risk for autism.

Using event-related potentials, **McCleery *et al.*** (pages 950–957) investigated face/object processing in infants at increased risk for autism by virtue of having a sibling with autism. They found that infants with no family history of autism show faster responses to faces than objects, while high-risk infants show the opposite pattern, i.e., faster responses to objects than faces. The results also show that while low-risk infants exhibit asymmetries in the volume of the cortical hemispheres, high-risk infants do not.

Phthalates Exposure: Link to ADHD Symptoms

There is widespread human exposure to phthalates, synthetic chemicals commonly used in plastics and other consumer products. **Kim *et al.*** (pages 958–963) report a significant positive association between urinary measures of phthalate exposure and both teacher-reported ADHD symptoms and measures from a computerized test of attention and impulsivity. These data, while cross-sectional, suggest an association between phthalate exposure and ADHD symptoms in school-aged children.

Developmental Alterations During Transition to Adulthood

Pan *et al.* (pages 964–971) discovered that amygdala-based fear pathways in mice undergo dramatic developmental changes during transition from the juvenile state to adulthood. These changes involve partial loss of synaptic plasticity in the thalamic but not cortical input to the amygdala. This may suggest that, in juveniles, processing of emotional information depends more on subcortical pathways, whereas in adults, cortex assumes a greater role.

0006-3223/09/$36.00