

NEWS



**Press release for
EMBARGOED UNTIL FRIDAY, OCT. 26 AT 8:00 AM PT**

Bipolar Disorder and ADHD: Could there be a connection?

San Diego – October 26 --Between 1994 and 2003, the number of U.S. children and adolescents screened for bipolar affective disorders (BPAD) increased 40-fold, and has increased further in the intervening 4 years. Despite controversy about its diagnostic criteria, the magnitude of this disorder, marked by mood swings, is clear. A new study identifying polymorphisms in the SNAP25 gene on chromosome 20p12 are associated with early-onset bipolar affective disorder. Its etiology has been determined to be distinct from ADHD, which is also linked to the same gene. Apparently, mutations in distinct regions of the SNAP25 gene might be independently associated with each of the disorders, such that mutations for ADHD reside at the 3' end, while mutations for BPD reside at the 5' end.

A study done by Dr. Stéphane Jamain, from Inserm, Creteil, France, tested the following premise -- that the gene located on chromosome 20p12, in a region that has been recently reported to be more frequently shared in early-onset bipolar affective disorder families, is known to be a frequent ADHD comorbid condition. As an altered level of SNAP25 has been reported in bipolar patient's brains, it was assumed that ADHD and early-onset BPAD may share common susceptibility variants in SNAP25.

Bipolar patients included in this study were between 14 and 81 years old at the time of inclusion, with 42% males. This population reflects the distribution of the general population. In this analysis, the age-at-onset was used to define early and late onset patients. The age of onset was defined as the age at which the patients first met the DSM-IV criteria for either a major depressive episode or mania according to medical case notes and interviews. The threshold for early-onset BPAD (age-at-onset lower than 22) was chosen according to previous admixture analyses (Bellivier et al, *Arch. Gen. Psychiatry* 2001; Bellivier et al, *Am. J. Psychiatry* 2003). The distribution of early and late onset patients is approximately 50% for each subgroup.

To Jamain's knowledge, the functional variation, putatively responsible for ADHD, has not yet been identified in SNAP25. His results suggest that the allele associated with BPAD may be different than the one associated with ADHD. However, these results need to be confirmed and clearly may be demonstrated using populations of patients well characterized for BPAD and ADHD. He says that the next step will be to replicate his results on a bigger population and confirm these results using larger, clinically well-defined populations.

ADHD and bipolar disorder may occur together, but separation of the risk factors for each will be an important step in understanding the underlying causes and interventions for each of these debilitating disorders.

XX

Please contact Jane Nelson, Media Relations, the American Society of Human Genetics, 9650 Rockville Pike, Bethesda, MD 20814 Tele: 301-634-7308 (em: jnelson@ashg.org) During the 57th Annual Meeting she can be reached at the San Diego Convention Center (619-525-6413).