

References.

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RISK FACTORS BY GENDER FOR CHILDREN WITH ADHD

Investigators at University of Western Australia conducted a population-based, case-control study of maternal, pregnancy, and newborn risk factors by gender for children prescribed stimulant medication for treatment of ADHD. Mothers of children with ADHD were significantly more likely to be younger, be single, have smoked in pregnancy, have labor induced, and experience threatened preterm labor, preeclampsia, urinary tract infection in pregnancy, or early term delivery irrespective of the gender of the child, compared with the control group. A possible protective effect of oxytocin augmentation is noted in girls. Factors not identified as risk factors included low birth weight, postterm pregnancy, small for gestational age infant, fetal distress, and low Apgar scores. (Silva D, Colvin L, et al. Environmental risk factors by gender associated with attention-deficit/hyperactivity disorder. **Pediatrics** 2014 Jan;133(1):e14-22).

COMMENTARY. Evidence of environmental mediators in ADHD is demonstrated in twin studies, affected twins having greater exposure to certain risk factors [1]. A familial, genetic factor in an estimated 80% of cases may involve the dopamine receptor and transporter genes, but gene-environment interaction is increasingly recognized as a mechanism in the etiology of ADHD [2][3]. Environmental factors may occur prenatally, in the perinatal period, or postnatally [4]. Of all the environmental factors implicated, maternal smoking and nicotine exposure attract the most attention in the literature, but in practice, maternal cigarette smoking is almost invariably denied [4].

References.

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NEUROMUSCULAR DISORDERS

AXONAL NEUROPATHY, MICROCEPHALY AND VRK1 MUTATIONS

Investigators from Baylor College of Medicine, Texas Children's Hospital, Houston, TX; Kennedy Krieger Institute, Baltimore, MD; and University of Minnesota, MN, report 3 patients from 2 unrelated families with a complex neuropathy phenotype characterized by axonal sensorimotor neuropathy, severe nonprogressive microcephaly and cerebral dysgenesis. Compound heterozygous alleles responsible for the clinical phenotype were identified by whole-genome and whole-exome sequencing in 2 affected siblings from 1 family and a homozygous nonsense variant in the third unrelated patient