

Genes and Parenting: Risk Factors in the Development of Attention-Deficit-Hyperactivity Disorder

The delineation of developmental pathways to psychiatric disorders of childhood and adulthood is a major challenge in the field of developmental psychopathology. A powerful means of identifying these pathways and the organismic and contextual factors that contribute to the onset and severity of psychopathology is through the use of prospective longitudinal designs. A longitudinal design that incorporates a high risk research strategy is most appropriate for the investigations of psychiatric disorders in which there is evidence of familial transmission (Garmezy & Streitman, 1974). Using this



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strategy, sample selection is based on the presence of symptoms of the disorder in a family member thereby increasing the probability that a higher percentage of the research cohort will eventually develop the disorder. This strategy is the one my colleagues and I have used to identify infants at risk for attention deficit hyperactivity disorder

(ADHD) based on paternal symptoms of ADHD.

ADHD is one of the most common disorders of childhood with a prevalence of 3–5% (American Psychiatric Association, 1994). Surprisingly, possible early developmental precursors of this disorder have been relatively uninvestigated. ADHD is a developmental disorder with both genetic and environmental underpinnings making it an ideal candidate for a comprehensive longitudinal investigation of its developmental trajectory.

The evidence that ADHD is a familial disorder is compelling (Barkley, 1990). Children of parents or siblings with ADHD are at increased risk of receiving a childhood diagnosis of ADHD (Biederman et al., 1995a). Twin studies support a strong genetic contribution to ADHD with heritability estimates ranging from 75% to 91% (Levy, Hay, McStephen, Wood, & Waldman, 1997). Recent evidence from molecular genetics also attests to a genetic basis for the disorder with the focus primarily being on genes associated with the dopamine system, for example, the dopamine D4 receptor gene (DRD4) (Faraone et al., 2001).

Nonoptimal parent-child interaction seems to antedate the disorder with coercive, overstimulating, intrusive, and restrictive parenting in the first years of life being predictive of later hyperactivity and impulsivity (Campbell, 2002). Family adversity, in the form of parental psychopathology, marital stress, and other stressful life events, is also associated with ADHD, with the risk of ADHD being particularly high in families with three or more risk factors (Biederman et al., 1995b).

One of the first tasks in our research was to link the symptoms and behaviors characteristic of children with ADHD to behaviors characteristic of the first years of life. Two approaches to the conceptualization of ADHD were particularly relevant for this task. The first approach conceptualizes the behaviors defining ADHD as falling at the extreme edge on a continuum of temperament traits seen throughout the population (Taylor, 1999). Emotionality, activity, and attention/orienting are included under the rubric of temperament and individual differences in their expression can be seen early in life (Rothbart, 1989). The second approach conceptualizes ADHD symptomatology as reflecting neurodevelopmental immaturity. Kinsbourne (1973) suggests that children with ADHD suffer from a delay in neurological maturation resulting in behaviors considered deviant for their chronological age. Making use of these two approaches, our research protocol in the first years of life includes measures which tap the expression of temperament and neurodevelopmental maturity. The effects of the environment are assessed by observations of parent-infant interaction and parenting questionnaires.

The sample is composed of boys only because the ratio of boys to girls with ADHD ranges from 3:1 to 9:1 (American Psychiatric Association, 1994). Families were recruited into the study at the birth of their sons. Two groups were formed based on ADHD symptomatology in fathers. Infants in the ADHD risk group have fathers with 7 or more ADHD symptoms while those in the comparison (low risk) group have 3 or less symptoms. The boys were assessed at one month of age with a standard neonatal behavioral assessment scale and at seven months, they were observed in standard episodes tapping different domains of temperament, such as interest, fear, activity, anger, pleasure, and in interaction with their parents. The episodes are designed to be similar to those encountered by infants in everyday situations. At both assessment periods, parents

completed questionnaires on infant temperament and at one year, a questionnaire on parenting competence. This is an ongoing study and the boys and their families are seen at the following ages: 1, 12, 24, 36, and 54 months.

The figure above presents a summary of the association between ADHD risk and infant behavior at one and seven months and for ADHD risk and parenting behavior and competence at seven months and one year. The associations between the measures are also presented. All lines between boxes indicate a significant finding of $p < .05$. The arrows in the boxes indicate the direction (more or less) of the behavior. For full details of the

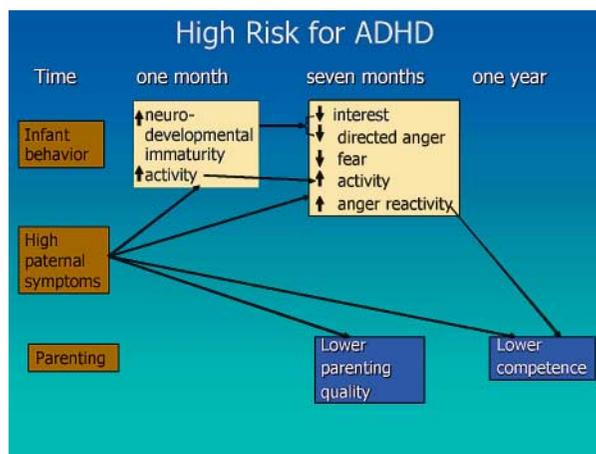


Figure 1. Summary of infant behavior and parenting indicative of high risk for ADHD.

infant behavior results, see Auerbach et al. (2004, 2005). With the exception of the one-month data and the seven-month infant-parent interaction data, all other data are based on partial data sets since not all the seven-month and one-year data have been coded.

The infant behavior results are compatible with ADHD literature conceptualizing ADHD as falling on a continuum of temperamental characteristics and also reflecting neurodevelopmental immaturity. In addition, the importance of including measures of parenting behavior is demonstrated by the less optimal parenting seen in the parents of the risk infants at seven-months of age.

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Note

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