The Biology of Attention Deficit Hyper-activity Disorder: Findings in Search of Conclusive Answers

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INTRODUCTION

ADHD is an early onset, highly prevalent neurobehavioral disorder, with genetic, environmental and biological etiologies that persist into adolescence and adulthood in a sizeable majority of afflicted children and adolescents of both sexes (Nijmeijer, et al. 2008).

It is characterized by behavioral symptoms of inattention, hyperactivity and impulsivity across the life cycle and is associated with considerable morbidity and disability (Spencer, et al. 2007).

ADHD has had a checkered history where a great deal of myth and misconception has guided both the diagnosis and the management of the disorder. The disorder has been known earlier by other names such as "brain damaged syndrome", "minimal brain dysfunction", "hyper kinetic impulsive disorder" and "attention deficit disorder". The changes in terminology have generally reflected an increase in the understanding of etiology, identification and management in more recent years (Rickel and Brown, 2007).

The prevalence of ADHD has been reported with great variations among different studies. Population variables that definitely affect the prevalence rates include gender, age, type of informants, source of information and population sample, i.e., community or clinic cohorts. Methodology features that can affect the findings include one-or two-setting design, DSM-III-R or DSM-IV criteria and clinical impairment evaluation. As a result of this complex influence, a definite prevalence of ADHD seems impossible to be achieved, unless studies of very similar design are employed (Skounti, et al. 2007). Recent systematic reviews report ADHD prevalence estimates as wide as 2%-18% (Sayal, 2007).

The Biology of ADHD:

Considerable research has accumulated on various etiologies for ADHD. Although multiple etiologies may lead to ADHD, evidence points to neurological and genetic factors as the greatest contributors to the disorder. In the past decade, no credible social-environmental theory even hypothesis concerning causation in ADHD has been developed. Yet, evidence suggests only some role for the social environment in the onset, course, and severity of those comorbid conditions (Barkley, 2005).

I- Genetic Factors:

Current research is being conducted to identify specific genetic contributions to ADHD. Molecular genetic studies and research investigating twins and adopted children suggest that genes predispose individuals to ADHD. Faraone, (2004) has hypothesized that it is the combined effects of multiple genes, yet to be identified.

A- Family Aggregation Studies

Research shows that between 10% and 35% of the immediate family members of children with ADHD are also likely to have the disorder, with the risk to siblings of these children being approximately 32% (Lévy and Hay, 2001).

Higher-than-expected rates of family aggregation of the disorder have been found in African American families, similar to rates reported in families of European American children (Samuel, et al. 1997). And these higher rates are as evident in the families of girls as of boys with ADHD (Faraone and Doyle, 2001). Even more striking is the finding that if a parent has ADHD, the risk to the offspring is 57% (Biederman, et al. 1995). Further evidence for the familial clustering of ADHD in families of affected children came from a study by Smalley, et al. (2000), in which they identified families having at least two affected children with ADHD (n= 132). They then assessed the 256 parents in these families for various psychiatric disorders and found that 55% of the families had at least one parent with a lifetime diagnosis of ADHD.

Genetic relatives of individuals with ADHD are more likely to have the disorder when compared to nongenetic relatives. In addition, genetic relatives of individuals affected by ADHD are more likely to have symptoms of the disorder than individuals from Psychiatric samples or the general population (Cook, 2000).