Common and specific genes and peripheral biomarkers in children and adults with Attention-Deficit/Hyperactivity Disorder.

Bonvicini C, Faraone SV, Scassellati C.


Abstract

OBJECTIVES:
Elucidating the biological mechanisms involved in Attention-deficit/hyperactivity disorder (ADHD) has been challenging. Relatively unexplored is the fact that these mechanisms can differ with age.

METHODS:
We present an overview of the major differences between children and adults with ADHD, describing several studies from genomics to metabolomics performed in ADHD children and in adults. A systematic search (up until February 2016) was conducted.

RESULTS:
From a PRISMA flowchart, a total of eligibility 350 studies from genomics and metabolomics were found for cADHD and 91 for aADHD. For children, associations were found for genes belonging to dopaminergic (SLC6A3, DRD4, MAOA) and neurodevelopmental (LPHN3, DIRAS2) systems and OPRM1 (Yates Corrected p = 0.016; OR = 2.27 95%CI:1.15-4.47). Studies of adults have implicated circadian rhythms genes, HTR2A, MAOB and a more generic neurodevelopmental/neurite outgrowth network (BCHE, SNAP25, BAIAP2, NOS1/NO, KCNIP4, SPOCK3; Yates Corrected p = 0.007; OR= 3.30 95%CI:1.33-8.29). In common among cADHD and aADHD, the most significant findings are for oxidative stress proteins (MAD, SOD, PON1, ARES, TOS, TAS, OSI), and, in the second level, DISC1, DBH, DDC, microRNA and adiponectin.

CONCLUSIONS:
Through a convergent functional genomics, this review contributes to clarify which genetic/biological mechanisms differ with age. The effects of some genes do not change throughout a lifetime, whereas others are linked to age-specific stages. Additional research and further studies are needed to generate firmer conclusions that might someday be useful for predicting the remission and persistence of the disorder. Although the limitations, some of these genes/proteins could be potential useful biomarkers to discriminate cADHD versus aADHD.