SLC6A1 gene involvement in susceptibility to attention-deficit/hyperactivity disorder: A case-control study and gene-environment interaction.

Yuan FF, Gu X, Huang X, Zhong Y, Wu J.


Abstract

Attention-deficit/hyperactivity disorder (ADHD) is an early-onset childhood neurodevelopmental disorder with an estimated heritability of approximately 76%. We conducted a case-control study to explore the role of the SLC6A1 gene in ADHD. The genotypes of eight variants were determined using Sequenom MassARRAY technology. The participants in the study were 302 children with ADHD and 411 controls. ADHD symptoms were assessed using the Conners Parent Symptom Questionnaire. In our study, rs2944366 was consistently shown to be associated with the ADHD risk in the dominant model (odds ratio [OR]=0.554, 95% confidence interval [CI]=0.404-0.760), and nominally associated with Hyperactive index score (P=0.027). In addition, rs1170695 has been found to be associated with the ADHD risk in the additive model (OR=1.457, 95%CI=1.173-1.809), while rs9990174 was associated with the Hyperactive index score (P=0.010). Intriguingly, gene-environmental interactions analysis consistently revealed the potential interactions of rs1170695 with blood lead (Pmul=0.044) to modify the ADHD risk. Expression quantitative trait loci analysis suggested that these positive single nucleotide polymorphisms (SNPs) may mediate SLC6A1 gene expression. Therefore, our results suggest that selected SLC6A1 gene variants may have a significant effect on the ADHD risk.