No genetic association between attention-deficit/hyperactivity disorder (ADHD) and Parkinson's disease in nine ADHD candidate SNPs.


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Abstract

Attention-deficit/hyperactivity disorder (ADHD) and Parkinson's disease (PD) involve pathological changes in brain structures such as the basal ganglia, which are essential for the control of the motor and cognitive behaviour and impulsivity. The cause of ADHD and PD remains unknown, but there is increasing evidence that both seem to result from a complicated interplay of genetic and environmental factors affecting numerous cellular processes and brain regions. To explore the possibility of common genetic pathways within the respective pathophysiologies, nine ADHD candidate single nucleotide polymorphisms (SNPs) in seven genes were tested for association with PD in 5333 cases and 12,019 healthy controls: one variant, respectively, in the genes coding for synaptosomal-associated protein 25 k (SNAP25), the dopamine (DA) transporter (SLC6A3; DAT1), DA receptor D4 (DRD4), serotonin receptor 1B (HTR1B), tryptophan hydroxylase 2 (TPH2), the norepinephrine transporter SLC6A2 and three SNPs in cadherin 13 (CDH13). Information was extracted from a recent meta-analysis of five genome-wide association studies, in which 7,689,524 SNPs in European samples were successfully imputed. No significant association was observed after correction for multiple testing. Therefore, it is reasonable to conclude that candidate variants implicated in the pathogenesis of ADHD do not play a substantial role in PD.