Relationship between the SNAP-25 gene and the effects of methylphenidate on the anterior cingulate cortex of patients with adult attention deficit hyperactivity disorder: a magnetic resonance spectroscopy study

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European Review for Medical and Pharmacological Sciences 2016; 20: 2443-2449

Abstract.

OBJECTIVE:
The effects of certain genetic alterations in the brain function of patients with attention deficit hyperactivity disorder (ADHD) remain unclear and, in fact, there is a limited amount of data in this field. For example, the relationship between the SNAP-25 polymorphism and brain metabolites in response to methylphenidate (MPH) has yet to be investigated. Thus, the present study aimed to determine the relationship between changes in creatine (Cr), choline (Cho), and N-acetyl aspartate (NAA) levels in the prefrontal cortex (PFC) and anterior cingulate cortex (ACC) of adults with ADHD and the SNAP-25 gene polymorphism following the use of MPH.

PATIENTS AND METHODS:
The present study assessed 60 patients between 18 and 60 years of age who were diagnosed with ADHD according to criteria from the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (DSM-IV). Genetic analyses were carried out using blood samples obtained from the ADHD patients and included a detailed clinical evaluation for the SNAP-25 gene polymorphism. The NAA, Cr, and Cho levels in the ACC and PFC were measured using magnetic resonance spectroscopy (MRS). Following the evaluation, 10 mg of oral MPH was given to the patients, and the same metabolite levels were measured after 30 minutes.

RESULTS:
The levels of NAA, Cr, and Cho in the PFC and ACC of patients with the SNAP-25 Ddel and Mnll polymorphism genotypes did not significantly differ before and after the administration of MPH. However, in patients with the SNAP-25 Ddel polymorphism T/T genotype and the Mnll polymorphism G/G genotype, there was a significant increase in NAA levels in the ACC after MPH treatment compared with before MPH treatment.

CONCLUSION:
The present results suggest that the SNAP-25 Ddel and Mnll polymorphisms might be associated with MPH-related changes in NAA levels in the ACC.