

Migraine and Autism Spectrum Disorder: A Shared Hereditary Basis [Letter]

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Dear editor

I have read the review article entitled “Exploring the Hereditary Nature of Migraines” by Bron et al published in *Neuropsychiatric Disease and Treatment*.¹ I congratulate the authors on the successful publication of this informative review article and would like to make some contributions.

The article discussed the pathophysiology of migraine in association with certain allelic variations in the genome. The review article gave a detailed account of Monogenic Forms of Migraine including the common gene variants that are associated with each form.

Although the article provided multiple hereditary aspects of migraine, I think the association of migraine with Autism Spectrum Disorder (ASD) should have been included. Several studies have been conducted that study migraine in relation to other pathological conditions highlighting a common genetic susceptibility. The meta-analysis conducted by Malik et al indicates shared genetic susceptibility to migraine and ischemic stroke (IS), with particularly strong overlap between Migraine without aura (MO) and both Large Artery Stroke (LAS) and Cardioembolic Stroke (CE) pointing towards shared mechanisms.² The findings in another meta-analysis conducted by Adewuyi et al confirm the comorbidity of endometriosis and migraine and indicate a non-causal relationship between the two traits, with shared genetically controlled biological mechanisms underlying the co-occurrence of the two disorders.³ These studies show that several conditions occur in association with migraine more often than it is believed. The association of migraine and ASD has the potential to be studied because both conditions share some common pathophysiological and genetic basis including calcium channel mutations and polymorphisms.⁴ The review article by Vetri also concluded that Autism and migraine share common pathophysiological changes: neurotransmission dysregulation, especially of the serotonergic system; altered immune response causing neurogenic neuroinflammation; abnormal findings especially in the cortical minicolumn organization and in the dysfunctional gut–brain axis; shared susceptibility genes.⁵

Therefore, it has become evident that the association of migraine with ASD warrants a thorough investigation to highlight their comorbidity correctly and completely. This will allow new preventive measures and treatment methods to be developed for both conditions and can lead to a proper understanding of their genetic basis.

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Disclosure

The author reports no conflicts of interest in this communication.

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