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Disturbed Cellular Iron Metabolism Genes in Neurodevelopmental Disorders is Different from Neurodegenerative Disorders

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Abstract : Background: Iron had been a focus of interest recently as a main exaggerating factor for oxidative stresses in the central nervous system and a link to various neurological disorders is suspected. Many studies with various techniques showed evidence of disturbed iron-related proteins in the cell in human and animal models of neurodegenerative disorders. Also, linkage to significant pathological changes had been evidenced e.g. apoptosis and cell signaling. On the other hand, the role of iron in neurodevelopmental disorders is still unclear. With increasing prevalence of autism worldwide, some changes in iron parameters and its stores were documented in many studies. This study includes Haemochromatosis HFE gene polymorphisms (p.H63D and p.C282Y) and ferroportin gene (SLC40A1) Q248H polymorphism in autism and control children. Materials and Methods: Whole genome DNA was extracted; p.H63D and p.C282Y genotyping was studied using specific sequence amplification followed by restriction enzyme digestion on a sample of autism patients (25 cases) and twenty controls. Results: The p.H63D is seen more than the C282Y among both autism and control samples, with no significant association of p.H63D or p.C282Y polymorphism and autism was revealed. Also, no association with Q248H polymorphism was evidenced. Conclusion: The study results do not prove the role of cellular iron genes polymorphisms as risk factors for neurodevelopmental disorders, and in turn highlights the specificity of cellular iron related pathways in neurodegeneration. These results demand further gene expression studies to elucidate the main pathophysiological pathways that are disturbed in autism and other neurodevelopmental disorders.

Keywords : iron, neurodevelopmental, oxidative stress, haemohromatosis, ferroportin, genes
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