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Genetic Analysis of the Clinical Manifestations of Alzheimer's Disease

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ABSTRACT

Introduction: Alzheimer's disease (ad) is characterized by being a progressive neurological disorder that involves the accumulation of the protein Amyloid Beta (Aβ) and Tau in neurons, leading to degeneration and death of these. Causing changes in memory, brain and behavior, is a slow and installation syndrome that often goes unnoticed in the early stages. Objectives: Analyze the main gene involved in the and identify a genetic interaction network, correlating to the changes in the functions of this gene. Methodology: We used the ALZGene database to know the main gene involved in the. After identifying the gene APOE, bioinformatics, GeneMANIA, was accessed to establish relations of APOE with other genes involved in the. Results and Discussion: It was found that the APOE gene is more predominant in the mutations, and that genes that exhibit physical interaction with APOE are: APP, LCK, APOC2, VLDLR, LDLR, LRP1, PLTP, APOA2, LIPC, CNTF, APOB, SCARB1. The results showed that one of the isoforms of APOE ε4 is causing increased risk of triggering, stimulating the Aβ deposition, affecting cognitive and functional decline in patients with mild cognitive decline and triggering inflammatory Cascades that cause neurovascular dysfunction allowing the entry of toxic proteins derived from blood in the brain. High education, leisure activities and exercise can reduce the risk of and cognitive decline. The diagnosis is made based on the signs of cognitive impairment being important to the exclusion of other potential causes, such as dementia and cerebal vascular disease. Conclusion: The presence of APOE ε4 does not require the development of the disease, however, the effects of this on the brain network connectivity, memory and cognitive decline are present in the patients and cognitively normal individuals.

Keywords: APOE; Alzheimer's disease; Genes

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