APOE ϵ 2 is Associated with Milder Clinical and Pathological Alzheimer's Disease

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Background

Alzheimer's disease (AD) is one of the forms of dementia, which affects >25 million people around the world. AD is not restricted to developed or developing nations; rather, it has become one of the major concerns among age-related health issues. Both etiological and genetic factors do not shell out enough information about the disease onset [1, 2]. Molecular biology plays a crucial role in understanding the pathophysiology of such diseases, which has become a major concern for people from all different continents. APOE ϵ 2 is associated with rescue or delaying the onset of AD in aged people contrary to APOE ϵ 4, which is associated with increased risk of AD. In this study, the authors test the potential of existing literature for APOE2 and APOE4 by comparing the effects on the large population data set for United States for well-characterized AD patients.

Methods

Study is a multicentric study. Subjects were selected from the National Alzheimer's Coordinating Center. All the patients were the part of any of 34 studies funded by National Institute of Aging (NIA). Inclusion criteria contained the following points: (1) no

primary neuropathological diagnosis other than AD neuropathological changes was found at autopsy, (2) final clinical evaluation within 2 years of death, (3) age of death 50 years or older and (4) APOE genotype available. Demographic characters were the part of the study that included sex, years of education, age of death, age of onset, symptom duration (age of death – age of onset), APOE genotype, last clinical dementia rating sum of boxes score and last Mini Mental State Examination score. Association of APOE alleles was done using multivariable models.

Interpretation

The authors analyzed the relation between APOE alleles and AD in a larger cohort. Parameters were set for various demographic factors as well as disease duration. Results interpreted the milder association of APOE ϵ 2 as compared to APOE ϵ 4. Variant 2 had been associated more with that of rescuing the AD pathology. However, the interesting fact that was observed was that both the alleles act differently when studied for their coactivity. It was observed that both did not exert direct effect on cognition of patient; rather, both had indirect effect on AD pathology mediated by their individual opposite effect against neuropathological lesions.

References

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