An overview of Alzheimer’s disease genetics

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With advances in medicine, public health, and nutrition, people are living longer than ever before. Longevity comes at a price, however, as more and more of the elderly develop dementia. Alzheimer’s disease (AD) is the most common form of dementia, affecting up to half of individuals by the time they are 80 years old. Much of what we know about AD arises from the study of mutations in one or more genes associated with familial disease. The identification of mutations that cause AD has completely changed the way both clinicians and scientists view the disorder. Instead of being an unfortunate ‘mental condition’, AD is now recognized as a progressive neurodegenerative disorder targeting the neural substrates of memory. Here, I will briefly discuss the growing epidemiologic concerns of AD in an aging population, then focus upon the biology of AD and how genetics has continually informed and directed ongoing investigations. I will pay particular attention to recent, unanswered questions in AD, including an intriguing link between AD and epilepsy, new evidence suggesting an infectious component to neurodegeneration in AD, and growing efforts to prevent the disease by co-opting the human immune system. I will also summarize our efforts to determine the means by which AD spreads in the nervous system, and potential opportunities for therapy in this regard. Questions such as these are essential for truly understanding why neurons die in AD, and are fueling the next generation of treatments aimed at stopping the growing epidemic that is AD.

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