Alzheimer's disease (AD) is a common neurodegenerative disease that affects cognitive function in the elderly. Alzheimer's disease is pathologically defined by amyloid-beta (Ab) senile plaques (SP) and neurofibrillary tangles (NFT) composed of tau. From the time of their original description, nearly a century ago, a major focus has been to understand the role that these lesions play in the pathogenesis of the disease. Despite an enormous production of data, Alzheimer's disease remains an enigma. According to the amyloid cascade hypothesis, amyloid precursor protein (APP) proteolytic process leading to the formation of beta-amyloid fibrils is considered to be the primary culprit of neurodegeneration. Other theories advance the notion that neuronal death is triggered by intracellular events, such as the hyperphosphorylation of tau proteins, which results in the composition of neurofibrillary tangles. But defining which one is still an ambiguous matter, as both theories have been thoroughly examined and supported by a great amount of data. Lately, less specific mechanisms, such as oxidative-stress, apoptotic degeneration, genetic defects, neurotransmitter dysfunction and inflammation as an overall procedure, have come to the front of scientific research. The forenamed processes altogether create one of the most bewildering puzzles of modern medicine. In the current paper, we try to point out the highlights of the current knowledge on Alzheimer's disease and to make a simplifying approach to the potential pathogenetic mechanisms. Deciphering the underlying mechanisms of the disease will generate the efforts for a rational therapeutic approach for this major public health problem.

Key words: Alzheimer's disease, beta-amyloid, protein tau, pathogenesis.

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