Aim & Scope:

Alzheimer’s disease (AD) is one of the most common neurodegenerative disorders of human beings. There are two forms of AD, sporadic (SAD) with non-Mendelian genetic pattern, and autosomal dominantly inherited familial AD (FAD). Mutations of the amyloid precursor protein (APP), the presenilin 1 (PS1) and the presenilin 2 (PS2) coding genes have been linked to FAD. Approximately 95% of AD cases occur as a SAD form, the rest (5%) have autosomal dominant inheritance (FAD). Although the discovery of AD took more than 100 years, its cause is not fully understood. It is known that in the course of the disease there is a loss of neurons in the most vulnerable areas, in the hippocampus as well as temporal, frontal and entorhinal cortex. It is also known that neuropathological hallmarks of AD are neuronal loss, extracellular senile plaques (SPs), containing β-amyloid (Aβ) and intracellular neurofibrillary tangles (NFTs) composed of hyperphosphorylated tau protein. The pathogenesis of AD is intensively studied throughout the world, however up till now, the cause of the damage to the neurons in the course of this incurable neurological disease is not clearly explained.

Moreover, despite the technological advances we are still not able to confidently identify AD intravitally. Additionally, there are currently no drugs that might inhibit the progression of this disease. Due to the aging of the population, the number of patients with AD increases and will constitute a growing social and economic issue for many countries. We hope that the proposed publications will inspire researchers to study the genetic and environmental triggers of the disease and will facilitate finding the effective treatment for patients with AD.

Keywords: Alzheimer’s disease, neurodegenerative disorders, tau protein, neurons.

Schedule:

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