Abstract

Alzheimer’s disease (AD) is a neurodegenerative disorder of CNS and very serious type of dementia. AD affected 46.8 million people worldwide in 2015, and it is estimated that the number of patients will double every 20 years, reaching over 130 million people in 2050 according to Alzheimer’s Disease International. There are two forms of the AD: familial (FAD) and sporadic (SAD) form. FAD is an early-onset disease caused by genetic mutations. SAD is more common, a late-onset disease with the age and ε allele of apolipoprotein E as major risk factors. The most crucial symptom is memory disorder, followed by disorientation, confusion, depression and later on, serious psychical and motor-skill problems. These symptoms are the result of a neuronal loss due to formation of β-amyloid oligomers and neurofibrillary tangles in the central nervous system (CNS).

As for now, there are neither efficient diagnostic approaches, nor therapeutic ways to stop the degeneration of the brain. There are some drugs available, such as inhibitors of acetylcholinesterase, that have proven to slow down the progression of the AD. Other cholinergic approaches have been developed, but they have shown a lot of side effects, as they are targeting a large scale of receptors. Additional approaches are focusing on clearance of β-amyloid deposition and neurofibrillary tangles.

Key words: acetylcholine, Alzheimer’s disease, β-amyloid, cholinergic system, inhibitors of cholinesterase, muscarinic receptors, nicotinic receptors, pharmacology