

Abstract

Alzheimer's disease (AD) is a progressive neurodegenerative disorder with complex pathophysiology affecting the central nervous system (CNS). In progress of the disease, various pathological changes occur in the brain, leading to neurodegeneration and subsequent impairment of physiological and cognitive functions. Although it is the most common cause of dementia in elderly, currently, there is no effective treatment for AD that targets its underlying mechanisms. There are different theories as to which process is the key trigger for the development of AD. The generally accepted theory considers increased production of amyloid β ($A\beta$), its accumulation in the ECS and the formation of amyloid plaques as the main cause of the disease. However, recent studies show that the primary cause of amyloid plaque formation is not increased $A\beta$ production, but rather its impaired clearance through the glymphatic system, the main component of which are aquaporin water channels, specifically aquaporin-4 (AQP4). The goal of this thesis is to provide an overview of the available knowledge on the involvement of aquaporins in AD pathophysiology, with a particular focus on AQP4 and its role in the glymphatic system.

Key words: Alzheimer's disease, neurodegeneration, central nervous system, astrocytes, aquaporins, glymphatic system