ABSTRACT

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Alzheimer’s disease (AD) is the most common cause of dementia of late life. AD manifests as progressive memory impairment followed by a gradual decline in other cognitive abilities. For AD is characteristic the abnormal accumulation or aggregation of proteins.

Advanced glycation endproducts (AGEs) are a heterogenous group of compounds that are formed when carbonyl group of reducing sugars reacts by a non-enzymatic reaction with amino acids in proteins, lipids and nucleic acids. The formation of AGEs promotes the deposition of proteins due to the protease resistant crosslinking, therefore is difficult to eliminace them from organism. AGEs can be detected in pathological deposits such as amyloid plaques and neurofibrillary tangles, which are prominent pathological feature sof AD. The formation of AGEs activate inflammatory signalling pathways, which are mediated by their receptor for advanced glycation endproducts (RAGE). RAGE is the signal transduction receptor which distinguishes a variety of signalling molecules including AGEs, β-amyloid, HMGB1 and etc. Therefore AGEs and and their receptor RAGE may play a crucial role in pathogenesis of AD.

This thesis is a literature review, which aimed to assemble findings targered to AGEs and receptor RAGE nad their involvement in the pathogenesis of AD. Since AGEs and their harmful effects to human health can cause undesirable diseases, it is necessary to investigate these potential therapeutical compounds that show inhibition effects to AGEs and RAGE. Therefore I have focused on compounds both synthetic and natural products, which might bet the effective inhibitors of AGEs and their receptor RAGE. In this review , I tried to summarize recent advances in the development of novel strategies which slow down the progression and treatment of neurodegenerative diseases especially of AD.

Keywords: Advanced glycation endproducts (AGEs), Alzheimer’s disease, RAGE, AGE inhibitors, RAGE antagonists