**Abstract:** One of the hallmarks of Alzheimer Disease (AD) is the presence of senile plaques consisting primarily of amyloid β peptide (Aβ), a 40–42(3) amino acid containing polypeptide. Like acetylcholinesterase (AChE), butyrylcholinesterase (BChE) inactivates the neurotransmitter acetylcholine (ACh) and is hence could be of considerable importance as a potential therapeutic target in Alzheimer’s disease, which is a disease characterized by a cholinergic deficit and is treated by AChE inhibitors. In this study we compared the effects AChE inhibitors, fasciculine, propidium and galantamine on amyloid aggregation induced by amyloid-β peptide (Aβ1–40) in the presence and absence of BChE and/or AChE. Our results showed that BChE like AChE could promote Amyloid-β peptide aggregation. Also the presence of inhibitors, which interacted differently with BChE, in the reaction mixtures containing BChE reduced Aβ40 aggregation, initially promoted by BChE. Our results shows that BChE can promote Aβ1–40 aggregation (primarily non-fibrillar aggregates which can be inhibited by AChE inhibitors although to different degrees. These kinds of studies is of potentially great importance in the design of new anti-cholinergic drugs with dual functions.

**Keywords:** beta amyloid (Aβ), fibril formation, acetylcholinesterase (AChE), butyrylcholinesterase (BChE), inhibitors.