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Molecular Docking of Compounds Modulating Amyloid Peptide Aggregation Schemes

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Alzheimer's disease is characterized by the formation of plaques in the brain, which are commonly composed of amyloid peptides as a result of aspartyl protease β- secretase expression. This work is dedicated to *in silico* studies of the interaction of artemisinin, dihydroartemisinin, dihydroartemisinin dimer with amyloid 12Aβ$_{9-40}$ peptide and β-secretase. The comparison was made with curcumin, which is in phase II of clinical trials. It has been shown that all ligands, similarly to curcumin, bind to the specific amino acids of the peptide that are responsible for the formation and the growth of the fibril with high affinity. Moreover, dihydroartemisinin and dihydroartemisinin dimer bind to amino acids that are responsible for the stabilization of formed fibril. All studied ligands interact with the critical amino acids of the catalytic center of β- secretase, while dihydroartemisinin dimer can also bind to Arg235, which is characteristic of peptide inhibitors of β-secretase. Dihydroartemisinin dimer has a higher binding affinity compared to other ligands. Thus, the selected compounds can be considered as possible candidates for the treatment of Alzheimer's disease.