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(57) Abstract :  
 [05] The elderly is disproportionately affected by Alzheimer's disease (AD), a form of dementia. Cognitive abilities and memory in Alzheimer's sufferers deteriorate with time. Recent studies have shown that people with Alzheimer's disease have elevated inflammatory markers, suggesting that inflammation plays a significant role in the onset and progression of the disease. Microglial receptors CD14, CD36, and CD47, and TLRs, may all be able to detect Aβ oligomers and fibrils. The neurodegenerative process begins when Aβ binds to either CD36 or TLR4, setting off an inflammatory cascade of chemokines and cytokines. TLR4 has been implicated in type 2 diabetes and Alzheimer's disease as of late. Several diabetes-related clinical problems, as well as changes in the body's internal environment and the brain's microenvironment, have been connected to TLR4 activation. Clinical trials have demonstrated that TLR4 inhibitors not only decrease the likelihood of getting sick but also increase life expectancy. Molecular docking and molecular dynamics modelling were used to examine the effectiveness of antidiabetic drugs against the TLR4 receptor. Parlodol's primary interactions were anticipated with the help of molecular docking investigations. With a binding affinity of -9.6 kcal/mol, it was the most promising of the candidates. The interaction pattern between Parlodol and the TLR4 receptor was verified by running a molecular dynamic simulation at a time scale of 50 nanoseconds. By making substantial contact with the active site, Parlodol ensured the complex's structural integrity was maintained throughout its rapid expansion. In light of these findings, further research into Parlodol's potential as a lead drug for TLR4 receptors is warranted. Accompanied Drawing [FIG. 1] [FIG. 2] [FIG. 3]

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