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Inhibition and disaggregation of human lysozyme amyloid aggregates by levodopa

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Amyloid related neurodegenerative diseases and non-neuropathic systemic amyloidosis have attracted much research efforts; however no curative drugs have been known till date other than the symptomatic cure. Therapeutic compounds that can either inhibit or disaggregate fibrillar species have been explored and many more are yet to be discovered. The current research describes an inclusive biophysical, microscopic and computational study establishing that L-3, 4-dihydroxyphenylalanine (Levodopa) to be promising against the inhibition and disaggregation of thermally induced amyloid fibrillation of human lysozyme (HL). The IC50 value of Levodopa was estimated to be 63.0±0.09 μM. Levodopa interferes amyloid fibrillation by forming hydrophobic interaction and hydrogen bond formation with the amino acid residues present in the amyloid fibril forming prone region of HL as explained by molecular simulation results. Levodopa was also found to disaggregate mature amyloid fibrils into unordered species and the DC50 value was calculated to be 19.95±0.06 μM. Thus, Levodopa and compounds with similar structure could be operative as a strong inhibitor towards the therapeutic development against systemic amyloidosis.

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