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The Inhibitory Effect of Synthesized Porphyrins on Lysozyme Protein Aggregation

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Abstract

Amyloid aggregation is recognized as a key pathological feature in many neurodegenerative diseases, including Alzheimer's disease, Parkinson's disease, and Huntington's disease. These conditions are characterized by the gradual loss of neurons and glial cells in the brain and spinal cord, leading to significant neurological impairments. The aggregation of specific proteins in these diseases results in neuronal toxicity and causes serious damage to the functionality of nerve cells. Amyloid beta (Aβ) protein is one of the most important proteins associated with Alzheimer's disease, where the aggregation of amyloid beta peptides leads to neuronal toxicity and serious damage to the functionality of nerve cells in the brain due to an imbalance, disrupting neuronal function. Additionally, the tau protein, which aggregates inside neurons, appears in the form of neurofibrillary tangles and plays a significant role in

disease progression. Targeting the aggregation of proteins and peptides presents a promising avenue for therapeutic intervention in neurodegenerative diseases. Small molecules and compounds, such as tetrapyrroles, have been identified as potential inhibitors of amyloid formation. These compounds interact with the aromatic residues of proteins and peptides, thereby stabilizing non-toxic oligomeric forms and preventing the formation of harmful aggregates. The aggregation of lysozyme in the presence and absence of MCTPP and TCPP was initially investigated using thioflavin T fluorescence assays. The results indicated that various concentrations of these compounds could inhibit lysozyme fibrillation by approximately 89% through an increase in the lag phase and a decrease in emission intensity. These findings demonstrate that MCTPP and TCPP effectively influence lysozyme fibrillation, showing a dose-dependent effect of the porphyrin compounds. To confirm the results obtained from thioflavin T fluorescence, Congo red assays, and atomic force microscopy imaging were also conducted, which verified the presence of fibrils in both the presence and absence of porphyrin compounds. The results obtained from UV spectroscopy confirmed the structure of the porphyrin compounds and lysozyme protein, as well as potential interactions between MCTPP, TCPP, and lysozyme protein.

Keywords: **Degenerative diseases of the nervous system, Lysozyme, Porphyrin, Porphyrin derivatives, Amyloid aggregation**

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