

## Abstract

Neurodegenerative diseases such as Alzheimer's, Parkinson's, and Huntington's are characterized by the accumulation of misfolded proteins in the brain, leading to neuronal dysfunction and eventual cell death. Understanding the molecular mechanisms of protein misfolding and aggregation is crucial for developing therapeutic interventions. Various molecular mechanisms implicated in protein misfolding, including mechanical stress, chaperone activity, and genetic mutations, can predispose certain proteins to misfolding by altering their primary structure or stability, as observed in familial forms of neurodegenerative diseases such as AD and HD [4,5].

**Role of misfolded protein in neurodegenerative disease**

## Molecular chaperone and protein folding

Molecular chaperones are proteins that assist in the folding of other proteins. They are essential for maintaining the proper structure and function of proteins. Chaperones can prevent the aggregation of misfolded proteins and facilitate the refolding of denatured proteins. They are involved in various cellular processes, including protein synthesis, maturation, and degradation.

### Application of chaperone in protein folding

Chaperones have been shown to have therapeutic potential in neurodegenerative diseases. For example, chaperone treatment has been shown to reduce the levels of misfolded proteins and improve neuronal function in animal models of Alzheimer's and Parkinson's disease. Chaperones are also being investigated as potential targets for drug development.

Exploring the Mechanisms of Protein Folding and Misfolding in Neurodegenerative Diseases

The process of protein folding is a complex and highly regulated biological event. It involves the transition of a polypeptide chain from a random coil to a specific, functional three-dimensional structure. This process is driven by the hydrophobic effect, hydrogen bonding, and electrostatic interactions. Misfolding, on the other hand, occurs when a protein fails to adopt its native structure, leading to the formation of aggregates that are often associated with neurodegenerative diseases. The mechanisms of protein folding and misfolding are still being actively researched, and understanding them is crucial for developing therapeutic strategies to prevent and treat these diseases.

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