The role of α-Synuclein misfolding in Parkinson's disease

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Abstract: Parkinson disease (PD) is a progressive neurodegenerative disorder in the central nervous system.Misfolding and aggregation of alpha-synoclain is one of the important factors in the incidence and progression of Parkinson's disease [1]. α-Synuclein is a 140-amino acid protein which has been proposed that it may act like a prion: pathological forms of the protein may be capable of changing the conformation of normal alpha-synuclein to aggregation form [2].

In this study, the protein domains were obtained from of Alzheimer's beta-amyloid proteins, human and sheep prion and also alpha-synuclein Parkinson's protein. We then obtained the motifs from domains, and after review, the motifs of human and sheep prion and Alzheimer's beta amyloid, which had the smallest and most similarity to the alpha-synuclin protein motifs, were selected. The interactions of these motifs with Parkinson's Protein were investigated using Autodokc vina. Molecular dynamics simulating of docked molecules detect change in behavior of alpha-synuclein protein, which it may be attribute to changes in conformation of alpha-synuclein.

Keywords: α-Synuclein; Parkinson; Autodock

References

[1] L. Breydo, J.W. Wu, "A-synuclein misfolding and Parkinson's disease**",** Biochim. Biophys. Acta, 1822 (2012), 261-285
[2] Y. Chu, J.H. Kordower, 'The prion hypothesis of Parkinson's disease" *Curr. Neurol. Neurosci. Rep, 15 (2015) doi: 10.1007*/s11910-015-0549