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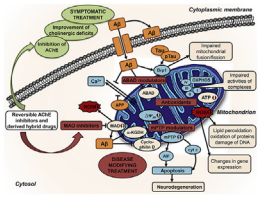
Jana Hroudová, Namrata Singh, Zdeněk Fišar and Kallol K. Ghosh*



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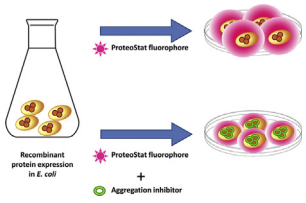
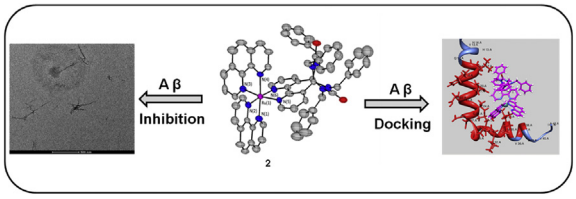
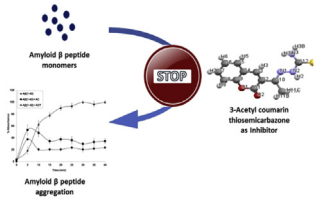
Susanna Navarro**, Anita Carija, Diego Muñoz-Torrero and Salvador Ventura*



Ruthenium(II) polypyridyl complexes with hydrophobic ancillary ligand as Aβ aggregation inhibitors pp. 793–802
Nilima A. Vyas, Shefali N. Ramteke, Avinash S. Kumbhar*, Prasad P. Kulkarni, Vinod Jani, Uddhavesh B. Sonawane, Rajendra R. Joshi, Bimba Joshi and Andrea Erxleben



Thiosemicarbazone modification of 3-acetyl coumarin inhibits Aβ peptide aggregation and protect against Aβ-induced cytotoxicity **pp. 803–809**
Dnyanesh S. Ranade, Archika M. Bapat**, Shefali N. Ramteke, Bimba N. Joshi, Pascal Roussel, Alain Tomas, Patrick Deschamps and Prasad P. Kulkarni*



Aggregation of amyloid-β peptide (Aβ) into amyloid plaques is essential event in Alzheimer's disease (AD) pathogenesis. Inhibition of Aβ aggregation is a promising avenue to explore as therapeutic treatment for AD. Our results indicate that 3-acetyl coumarin thiosemicarbazone inhibits Aβ(1-42) peptide aggregation up to 80% compared to the parent 3-acetyl coumarin which inhibits 49%. Further, 3-acetyl coumarin thiosemicarbazone provides neuroprotection against Aβ-induced cytotoxicity in SH-SY5Y cell line. These findings indicate that thiosemicarbazone modification renders 3-acetyl coumarin significant neuroprotective properties.

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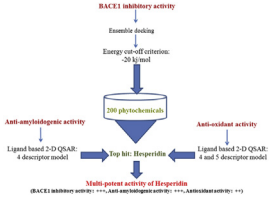
Sandipan Chakraborty**, Jaya Bandyopadhyay, Sourav Chakraborty and Soumalee Basu*



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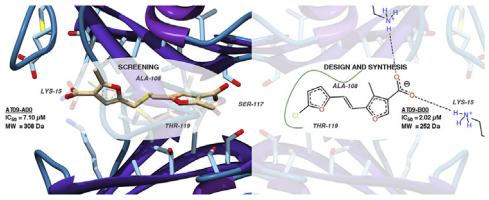

Coupling of the non-amyloid-component (NAC) domain and the KTK(E/Q)GV repeats stabilize the α-synuclein fibrils **pp. 841–850**
Liang Xu, Ruth Nussinov and Buyong Ma*