INTERACTION OF Zn AND Cu WITH THE AMYLOID-β PEPTIDE AND PROSPECTIVE FOR A METAL-BASED THERAPY OF ALZHEIMER'S DISEASE

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In the last decades different neurodegenerative diseases have been linked to a common characteristic: the misfolding of specific proteins or peptides, which causes the deposition of amyloid fibrils and plaques in different tissues. Furthermore, metal ion dishomeostasis has been linked to, among others, Alzheimer's and Parkinson's. These metal ions, especially Cu and Zn, are thought to influence the aggregation of the peptides and proteins, and modulate their toxicity. Particularly relevant would be the role of Cu, since it could be implied in the oxidative stress found in Alzheimer's disease through the formation of Reactive Oxygen Species. To address these questions, the latest studies relevant to the formation of ROS by Cu-Aβ complexes will be presented. Another important question is the mutual influence Cu and Zn could have on the metal-induced aggregation, the ROS production and the therapy based on Cu-chelation. In this last context, we have also developed some requirements which the designed chelators should fulfil, e.g. a good selectivity for Cu over Zn, their capability for chelating both Cu(I) and Cu(II) or a multifunctional role. Some of these concepts will be presented.

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