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CSB-PO-22 Copper(II) and zinc(II) interaction with Aβ42: effects of metal binding on peptide's aggregation rate and morphology of the aggregates.

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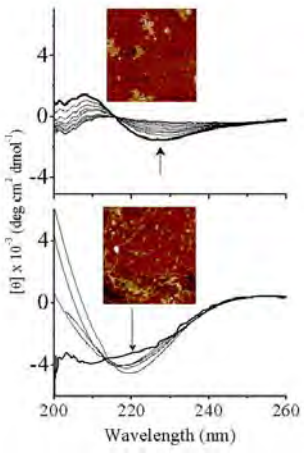
Altered levels of zinc(II) and copper(II) in different brain districts have been implicated in various aspects of Alzheimer's diseases.[1] In particular, it is well established that metal ions play a major role in the self-assembling of Aβ, but their effects on fibrillogenesis and morphology of peptide aggregates are not fully elucidated yet and conflicting results are reported in the literature.[2] In the attempt to shed light on these debated issues, two slightly different monomerization protocols were developed to mimic "seeded" and "unseeded" Aβ(1-42) assembling. Then, metal effects on the peptide aggregation and morphology were comparatively investigated by CD, ThT fluorescence and SFM techniques. Our results indicate that unlike copper(II) which promotes the formation of amorphous aggregates, zinc(II) is quite able to convert soluble Aβ peptides into amyloid-like structures. The obtained results might contribute to set up a hypothesis that correlates metals' coordination modes and different aggregate morphologies as well as in vitro toxicities towards neuronal cell cultures.



[1] Bush A.I., Tanzi R.E., Proc. Natl. Acad. Sci., USA, **2002**, 99, 7317-7319.
[2]. Pappalardo G., Milardi D., Rizzarelli E., Sovago I., in "Neurodegeneration: Metallostasis and Proteostasis" Milardi E., Rizzarelli E. Eds., Chapter 6 pp 112-131, RSC UK London **2011**. In press