

fac-{Ru(CO)₃}²⁺ selectively targets the histidine residues of the β-amyloid peptide 1-28. Implications for new Alzheimer's disease treatments based on ruthenium complexes.

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Streszczenie

The reaction of the ruthenium(II) complex *fac*-[Ru(CO)₃Cl₂(N(1)-thz)] (I hereafter; thz = 1,3-thiazole) with human beta-amyloid peptide 1-28 (Abeta(28)) and the resulting {Ru(CO)₃}²⁺ peptide adduct was investigated by a variety of biophysical methods. (1)H NMR titrations highlighted a selective interaction of {Ru(CO)₃}²⁺ with Abeta(28) histidine residues; circular dichroism revealed the occurrence of a substantial conformational rearrangement of Abeta(28); electrospray ionization mass spectrometry (ESI-MS) suggested a prevalent 1:1 metal/peptide stoichiometry and disclosed the nature of peptide-bound metallic fragments. Notably, very similar ESI-MS results were obtained when I was reacted with Abeta(42). The implications of the above findings for a possible use of ruthenium compounds in Alzheimer's disease are discussed.

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