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Copper(II) and amylin analogues: a complicated relationship

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Protein aggregation has attracted substantial interest because of its role in causing many serious illnesses, such as neurodegenerative diseases and type II diabetes. Recent studies have shown that protein aggregation can be prevented by forming metal ion complexes with a target protein, which affects their conformation in solution and their physical properties, such as aggregation. Thus, understanding the interactions between aggregating molecules and bioactive metal ions such as Cu^{2+} is beneficial for new drug discovery. Pramlintide, a synthetic peptide drug, and its natural counterpart rat amylin are known to be resistant to aggregation because of the presence of proline residues, which are usually β-sheet “breakers” within their amino acid sequence. Here, we investigate the Cu^{2+} coordination properties of pramlintide and rat amylin using nuclear magnetic resonance, circular dichroism, electron paramagnetic resonance, ultraviolet–visible spectroscopy, potentiometry, and mass spectrometry. We test the influence of Cu^{2+} on the aggregation properties of these amylin analogues with thioflavin T assays. We find that both peptides form stable complexes with Cu^{2+} with similar affinities at a 1:1 ratio. The N-termini of both peptides are involved in Cu^{2+} binding; His18 imidazole is an equally attractive binding site in the case of pramlintide. Our results show that Cu^{2+} ions influence the aggregation of pramlintide, but not that of rat amylin.

Słowa kluczowe

Peptides and proteins, Ligands, Monomers, Ions Rodent models

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