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Molecular mechanisms in nickel carcinogenesis: modeling Ni(II) binding site in histone H4.

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Streszczenie

Ni(II) compounds are well known as human carcinogens, though the molecular events which are responsible for this are not yet fully understood. It has been proposed that the binding of N(II) ions within the cell nucleus is a crucial element in the mechanism of carcinogenesis. The most abundant proteins in the cell nucleus are histones, and this makes them the prime candidates for this role. This article is a review of our recent studies of histone H4 models of Ni(II) binding. We analyzed the sequence of the N-terminal tail of the histone H4, Ac-SGRGKGGKGLGKGG AKRH18RKVL-Am, for Ni(II) binding. This site has been proposed mainly because of the potent inhibitory effect of Ni(II) on the acetylation of lysine residues near the histidine H18, and also because of the accessibility of the H4 tail in the histone octamer. Combined potentiometric and spectroscopic studies showed that the histidine 18 acted as an anchoring binding site for metal ions in the peptide investigated. Comparison with the results for Cu(II) binding are also reported. The results allowed us to propose that the binding of Ni(II) is able to promote a secondary structure with organized side-chain orientation on the N-terminal tail of histone H4.

Słowa kluczowe

nickel, histone H4, carcinogenesis

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