Molecular models in nickel carcinogenesis.

Autorzy

Wojciech Bal

Henryk Kozłowski

K. S. Kasprzak

Rok wydania

2000

Czasopismo

Journal of Inorganic Biochemistry

Numer woluminu

79

Strony

213-218

DOI

10.1016/S0162-0134(99)00169-5

Kolekcja

Naukowa

Język

Angielski

Typ publikacji

Artykuł

Streszczenie

Nickel compounds are known human carcinogens, but the exact molecular mechanisms of nickel carcinogenesis are not known. Due to their abundance, histones are likely targets for Ni(II) ions among nuclear macromolecules. This paper reviews our recent studies of peptide and protein models of Ni(II) binding to histones. The results allowed us to propose several mechanisms of Ni(II)-inflicted damage, including nucleobase oxidation and sequence-specific histone hydrolysis. Quantitative estimations of Ni(II) speciation, based on these studies, support the likelihood of Ni(II) binding to histones in vivo, and the protective role of high levels of glutathione. These calculations indicate the importance of histidine in the intracellular Ni(II) speciation.

Słowa kluczowe

Nickel(II), Nickel(II)–peptide interactions, Histone H1, Histone H2A, Histone H3, Histone H4, Core histone tetramer, Peptide bond hydrolysis, 2′-Deoxyguanosine oxidation, Speciation

Adres publiczny

https://doi.org/10.1016/S0162-0134(99)00169-5

Strona internetowa wydawcy

http://www.elsevier.com