



Specific poly-histidyl and poly-cysteil protein sites involved in Ni^{2+} homeostasis in *Helicobacter pylori*. Impact of Bi^{3+} ions on Ni^{2+} binding to proteins. Structural and thermodynamic aspects.

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Rok wydania

2012

Czasopismo

Coordination Chemistry

Reviews

Numer woluminu

256

Strony

133-148

DOI

10.1016/j.ccr.2011.06.014

Kolekcja

Naukowa

Język

Angielski

Typ publikacji

Artykuł

Cysteine and histidine residues are tempting donors for Ni^{2+} which coordinates to the sulfur of Cys and amide nitrogen atoms, or, in the absence of available thiol groups, to His imidazoles and amides. Bi^{3+} , on the other hand, has a very strong affinity towards Cys thiol groups, and can also coordinate an additional His imidazole.

In this review, the complicated pathway of nickel uptake, delivery and regulation in microorganisms is summarized. We show potential binding sites, binding geometries, protein structures and discuss the predicted thermodynamic and kinetic aspects. We focus on the numerous recent observations on the homeostasis of nickel in *Helicobacter pylori* (*H. pylori*), a Gram-negative bacterium that colonizes the gastric mucosa in humans, and is the causative agent of acute and chronic gastritis, peptic ulcer disease, gastric carcinoma, and gastric lymphoma.

The homeostasis of Ni^{2+} is crucial for the survival of *H. pylori* in the extremely acidic environment of the stomach. The metal is delivered to urease (which catalyzes the hydrolysis of urea into carbon dioxide and ammonia and therefore neutralizes the low gastric pH) and to hydrogenase (which permits respiratory based energy production for the bacteria in the mucosa) by a set of accessory proteins. Most of the bacterium's metal metabolism is centered upon their expression and maturation.

Below, a detailed description of the structural and thermodynamic aspects of the binding of nickel ions to poly-histidyl and poly-cysteil sites of urease and hydrogenase accessory proteins is given. Because bismuth compounds are one of the treatments for peptic ulcer disease, the inhibitory effect of Bi^{3+} ions is described; the affinity of bismuth towards Cys side groups is much stronger than the affinity of nickel towards the same sites, therefore bismuth is able to displace nickel from its binding site, causing the inhibition of nickel chaperones.

Słowa kluczowe

Nickel homeostasis, *Helicobacter pylori*, Inhibitory bismuth, Metal binding sites

Adres publiczny

<https://doi.org/10.1016/j.ccr.2011.06.014>

Strona internetowa wydawcy

<http://www.elsevier.com>

Plik został wygenerowany dnia 2026-08-23 14:46:07

Adres w repozytorium <https://old.chem.uni.wroc.pl/pl/repozytorium/vnAkASs>.