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Coordination chemistry of virulence: Zn(II), Cu(II) and Ni(II) in bacterial metal control

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Streszczenie

Antimicrobial resistance is accelerating worldwide, forcing the search for therapeutic strategies that undermine bacterial survival pathways beyond the canonical antibiotic targets. Transition-metal control is one such vulnerability: Zn(II) and Ni(II) are required cofactors for numerous bacterial proteins, whereas copper is simultaneously a micronutrient and a potent antimicrobial stressor. During infection, pathogens must operate inside a chemically dynamic battlefield shaped by host nutritional immunity and metal intoxication, where scarcity, toxicity and mismetallation risks coexist.

Here we review bacterial Zn(II), Cu(I/II) and Ni(II) homeostasis through the lens of coordination chemistry and show how donor preferences, geometry and redox speciation are encoded into transport, sensing and trafficking circuits. For zinc, we focus on high-affinity uptake modules (e.g., ZnuABC/AdcABC supported by ZinT-like proteins, outer-membrane receptors such as ZnuD/AztC, and metallophore-linked Cnt systems), complemented by low-affinity routes (e.g., ZupT) and detoxification/export via P-type ATPases, CDF transporters and RND pumps. For copper, we outline the redox-aware network that keeps intracellular copper largely as Cu(I) and routes it through dedicated chaperones (CopZ, CusF, CueP, CupA/CopC) toward either cuproenzyme assembly or removal by CopA-type ATPases and the CusABCF efflux machine; we also discuss outer-membrane and inner-membrane handling (OprC-like proteins, CcoA/CalT family import) and regulatory control by CueR, CsoR and CopY as well as Cu-responsive two-component systems. For nickel—largely irrelevant to mammalian physiology—we highlight dedicated acquisition (NikABCDE, NixA), delivery and storage pathways that sustain Ni-enzymes central to colonization and stress tolerance (urease, [NiFe]-hydrogenases, NiSOD, glyoxalase I).

We then extract a compact set of physicochemical “rules” (HSAB complementarity, Irving–Williams bias,

geometry/nuclearity constraints and copper redox/speciation) that rationalize why bacterial metal machineries diverge from mammalian ZIP/ZnT-centered handling and therefore offer selectivity windows. Finally, we discuss actionable antimicrobial angles: blocking metal uptake/export nodes and metalloregulators absent or highly divergent in humans (Zur/AdcR/ZntR/CzrA), disrupting chaperone–transporter hand-off steps, and exploiting metallophore recognition for “Trojan-horse” delivery or toxic-metal hijacking to sensitize multidrug-resistant pathogens.

Słowa kluczowe

Transition metal homeostasis, Nutritional immunity, Host-pathogen metal competition, Copper redox stress, Nickel-dependent enzymes, Metal trafficking, Mismetallation, Antimicrobial target discovery

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