DNA-binding property of *Helicobacter pylori* NikR

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H. pylori is a Gram-negative gastric pathogen that is responsible for the majority of peptic ulcer diseases in human and significantly increases the risk of gastric cancers. Nickel is a cofactor of the *H. pylori* urease and hydrogenase enzymes. Nickel uptake in *H. pylori* is mediated by the NixA protein and the expression of both NixA and urease are regulated by *H. pylori* NikR (*Hp*NikR) (1). *Hp*NikR also serves as a major regulator of the protective response against acid, and controls many important cellular processes and virulence factors either directly or indirectly (2). As *Hp*NikR regulates gene expression by binding to specific DNA sequences, the investigation of the sequence-specific DNA-binding properties of *Hp*NikR is important for understanding the mechanism by which *Hp*NikR regulates *H. pylori* physiological functions. In the present work, the binding properties of *Hp*NikR with DNA were studied by electrophoresis mobility shift assays (EMSA) and DNaseI footprinting assays. We found that *Hp*NikR can bind to different DNA sequences in the presence of Ni^{2+}, and the affinities are dependent on both the DNA sequence and the Ni^{2+} concentration. Our results indicate that *Hp*NikR may regulate *H. pylori* transcription in a different manner than the *E. coli* NikR metalloregulator.

1. Contreras M, Thiberge JM, Mandrand-Berthelot MA, Labigne A. *Mol.Microbiol.* 49: 947 (2003)
2. van Vliet AH, Ernst FD, Kusters JG. *Trends Microbiol.* 12: 489 (2004)