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DNA Probe-Mediated Detection and Nucleotide Sequence of Plasmid-encoded Nickel Resistance Determinant from *Hafnia alvei* 5-5

Jeong Eun Park*, Kyung Seon Choi, H. G. Schlegel¹ and Ho Sa Lee
Department of Biology, Kyunghee University, Seoul, Korea, 130-701
¹*Institut für Mikrobiologie und Genetik, Universität Göttingen, Germany*

Hafnia alvei is a new highly nickel-resistant bacterium. It was isolated after enrichment culture selective for *Escherichia coli* type bacteria from a soil-litter mixture underneath the canopy of the nickel-hyperaccumulating tree *Sebertia acuminata* in New Caledonia. Two plasmids were identified, one of the size of 70kb and another is 3kb. *Hafnia alvei* 5-5 DNA fragments encoding resistance to Ni²⁺, Co²⁺, Zn²⁺ were cloned by DNA-DNA hybridization. The biotinylated DNA probes were derived from *Alcaligenes eutrophus* CH34, *Alcaligenes xylooxidans* 31A, and *Klebsiella oxytoca* CCUG 15788. The nickel resistance fragment isolated from *H. alvei* 5-5 was studied in some detail. This 7.9kb *EcoRI*-*Bam*HI fragment conferred resistance to 6 mM nickel to *Escherichia coli*. It showed strong homologies to both the ncc operon and the nre operon. The determinant of which has been cloned and sequenced.

F801 Interaction of TEL/AML1 and AML1 in the human *CRI* gene promoter

Sun Young Park and Soo Young Choe
Department of Biology, Chungbuk National University

Complement receptor type1(CR1, CD35) plays an essential role in immune complex processing and regulation of complement system. The human *CRI* promoter contains an AML1 binding site as well as an adjacent site to which the GGAA ETS family protein binds. In this study we use the *CRI* proximal promoter to characterize TEL, AML1 and TEL/AML1 in regulation of the human *CRI* promoter. Reporter luciferase genes downstream of *CRI* proximal promoters are activated in HEL cells when cotransfected with AML1 expressing vector. By contrast, expression of a mutant AML1, which the AML1 are fused with TEL, had no effect. Moreover, TEL/AML1 fusion protein could interfere efficiently AML1-dependent transactivation of human *CRI* gene promoter. These results suggest that the interaction of TEL/AML1 and AML1 protein is an important regulatory system in *CRI* gene expression and possibly play a key role in hematopoietic-specific gene expression.