F334 Distribution of Genes Coding for Aminoglycoside Acetyltransferases among Gentamicin Resistance Bacteria Isolated from Aquatic Environments

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Most resistance to aminoglycosides result from the production of plasmid-encoded aminoglycoside modifying enzymes namely, acetyltransferases, nucleotidyltransferases and phosphotransferases. Studies on the distribution of antibiotic resistance genes will supply data not only of clinical and epidemiological implications but also information about gene transfer processes in natural environments. DNA probes are useful tools studying epidemiology of antimicrobial resistance gene. Recently, PCR methods are used for the identification of aminoglycoside modifying enzymes. In this study we report the distribution of genes for aminoglycoside acetyltransferase among the gentamicin resistance bacteria isolated from aquatic environments.

F801 The *YDR1/BUR6* heterodimeric complex is a Transcriptional Repressor in Yeast

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The human heterodimeric Drl/DRAP1 is a general transcription repressor extensively studied *in vitro*. The two proteins repress transcription by binding TATA-binding protein (TBP), inhibiting the formation of transcription preinitiation complex. Recently the counterparts of the human Drl and DRAP1 were identified and named *YDR1* and *BUR6*, respectively, in the yeast *Saccharomyces cerevisiae*. The *YDR1* gene is essential for cell viability. *BUR6* is also essential for cell viability. The human Drl gene can replace the *YDR1* gene *in vivo*. *YDR1* overexpression *in vivo* confers slow growth phenotype. The *YDR1* and *BUR6* can be copurified in a gel filtration column, using affinity-purified polyclonal antibody against both yDrl and Bur6 polypeptides. The gel mobility shift assay showed that Ydrl and Bur6 repress *in vitro* transcription and this repression of basal transcription can be overcome by TBP, a global regulator. These results indicate that *YDR1/BUR6* functions as a global repressor of transcription *in vivo* and directly targets TBP. We are currently mutagenizing *YDR1* and *BUR6* to study sturcuture-function of the two genes.