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Heat shock response plays a central role in cellular adaptation to stress and hostile environments. Of several heat shock proteins (hsps), DnaK (hsp70 family) and GroEL (hsp60 family) function as molecular chaperones in the folding of nascent protein chains and in the refolding of proteins after thermal damage. Expression of dnaK operon and groEL operon in S. pneumoniae and other gram positive organisms is negatively controlled by a repressor encoded by hrcA, the first gene of dnaK operon. HrcA repressor bound to CIRCE elements at the promoter site of dnaK and groEL operon. Since HrcA depends on GroEL to acquire on active conformation, GroEL seems to be specific modulator of the CIRCE regulon. Previously we demonstrated that the dnaK operon has three promoter sites and binding of HrcA to CIRCE element was stimulated by supplement of calcium. In this study, to elucidate the pathological role and expression control of dnaK operon in S. pneumoniae, nucleotide sequence of the dnaK operon was completely determined and effect of hrcA mutation on dnaK expression was examined. In the hrcA mutant, the basal and induced levels of DnaK protein and dnaK transcript were much higher than that of the wild type. When the effect of calcium on hsp expression was determined by Western blot analysis, both GroEL and HrcA proteins were decreased to the basal level by 1mM calcium but DnaK was not affected by calcium. These results suggest that calcium may play a differential roles in either promoter 1, 5' to the hrcA or promoter 3, 5' to the dnak.

[PC2-8] [10/20/2000 (Fri) 15:30 - 16:30 / [Hall B]]

ermAM and ermK are also inducibly expressed by 16 -membered-ring macrolide antibiotics.

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The *erm* family is one of the major groups of genes responsible for inducible macrolide-lincosamide-streptogramin B (MLS_B) antibiotics resistance. The 16-membered-ring macrolides have been generally considered noninducers except in the case of *Streptomyces* and the selected mutant strains. In this study, the induction specificity of *ermAM*, *ermK*, and *ermC* for MLS_B resistance was studied by *lac* reporter gene assay. The unexpected MLS_B resistance phenotypes were observed. *ermAM* was induced by the 16-membered-ring macrolides tylosin, kitasamycin, josamycin, and rokitamycin more strongly than the lincosamide clindamycin known as inducer of *ermAM*. *ermK* was only induced by the tylosin of the 16-membered-ring macrolides tested. However *ermC* was not induced by any 16-membered-ring macrolides.

[PC2-9] [10/20/2000 (Fri) 15:30 - 16:30 / [Hall B]]

Ulcerative colitis induced GAGs degrading enzymes of intestinal bacteria

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Ulcerative colitis(UC) is a non-infectious chronic intestinal inflammatory disease in humans. The mechanisms underlying their pathogenesis are not well known, although it has been long discussed.
We produced acute experimental UC models in mice by several methods (5% dextran sulfate