Tn10dTc insertion mutant. Surprisingly, it showed an increased level of ATR in comparision with wild type UK1 in log phase ATR test while no such effect was detected in stationary phase ATR test. To complement this phenomenon of the mutant, several plasmids were constructed and introduced into the mutant. As the result, it was proposed that the region was critical in log phase ATR. Finally, its transcriptional start site was examined by primer extension assay and sequencing analysis. Taken all together, it is suggested that the unidentified ORF is acid-inducible and may be important in survival of Salmonella enterica serovar Typhimurium inside macrophage.

**F334**

## The Expression, Regulation and Promoter Analysis of cspH, One of the Cold Shock Genes in *Salmonella typhimurium*

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cspH is one of the genes encoding cold shock proteins (CSPs) in *Salmonella typhimurium*. Previously, we showed that its promoter was active not only upon cold shock condition (approximately 15℃) but also at 37℃ and proved that its 5'-untranslated region (UTR) was unusually short unlike the long 5' -UTRs in the other cold-shock inducible genes. In this study, we showed that mRNA of cspH was more stable than that of other csp genes at 37℃ using analysis of mRNA stability. It was shown that the 14 base downstream box (DB) locating 12 base downstream of the initiation codon of cspH mRNA and complementary to a region near the decoding region of 16S rRNA was essential for the mRNA translation during the growth acclimation phase immediately after cold shock. The cspH-lacZ fusion plasmid revealed that a minimal promoter sequence consisting of 55 bp was sufficient to generate its growth phase-dependent expression and cold-shock induction pattern. Furthermore, we found that a putative Fis binding site was present upstream cspH promoter. Using the fis mutant strain contaning wild type cspH-lacZ translational fusion and the wild type strain containing Fis site deleted cspH-lacZ translational fusion plasmid, we revealed that Fis regulated the expression of cspH at 37℃.

**F335**

## The Regulation of *rfaYZ* against Oxidative Stress in *Escherichia coli*

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Promoters inducible by paraquat, a superoxide-generating agent, were isolated from *Escherichia coli*, using a promoter-probing plasmid pRS415 with promoterless *lacZ* gene. Twenty two promoters induced by paraquat, were selected and further characterized. One of SoxRS-dependent promoters, *rfaY*p, was characterized. The *rfaY* gene is found in the middle of LPS core biosynthetic gene cluster in *E. coli*. *rfaY*p-*lacZ* fusion was induced 10 fold by paraquat and other superoxide generators (menadione, plumbagin, and lawsone) in single copy state, while no induction was observed by $H_2O_2$, ethanol, and heat shock. Induction of *rfaY* disappeared by introducing a *soxRS* mutation into the fusion strain, indicating that *rfaY* is a member of the *soxRS* regulon. The transcriptional start site was determined by primer extension analysis. The -10 and -35 boxes of *rfaY*p were predicted. The Northern

analysis revealed that the downstream *rfaZ* gene consists an operon with the *rfaY*. SoxS bound upstream of the -35 box and directly activated *rfaY* as judged by gel shift and in vitro transcription assay. To elucidate the function of *rfaYZ* genes in oxidative stress response, we disrupted *rfaYZ* operon. The *DrfaYZ* mutant was more sensitive to the oxidative stress. In addition, SoxR became more sensitized in the mutant to the quinone-type redox cycling agents, such as menadione and plumbagin. When *DrfaYZ* mutation was introduced into *rpoH*p3-*lacZ* single copy fusion strain, expression from *rpoH*p3 promoter increased 5.4 fold, implying that *rfaYZ* mutation cause some extracytoplasmic stress. The phenotype of SoxR-sensitization in the mutant was complemented by overexpression of the *rfaYZ* genes. However, increase in *rpoH* expression was not rescued even with the overexpression of *rfaYZK* genes.

**F801**

## (GT)n Repeats Polymorphism of the Third Intron of T Cell Receptor Delta (*TCRD*) among Koreans

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The gamma/delta T cells recognize unprocessed or native antigens independently of the MHC, rapidly enhancing the immunity defense mechanism. The gamma/delta T cells have cytotoxic effects and produce large amounts of Th1 cytokines. The *TCRD* is located on the chromosome 14q11.2 and consists of three variables, three diversities, and three joining and constant genes. We analyzed the (GT)n repeats polymorphism of the third intron of *TCRD* using 6% denaturing PAGE in two hundred thirty-one unrelated Korean subjects. The number of GT repeat was estimated for repeat polymorphism by auto-sequencing. Six alleles ranged in size from *TCRD*13* (116 bp) to *TCRD*19* (128 bp). The allele frequencies, *TCRD*13*, *TCRD*15*, *TCRD*16*, *TCRD*17*, *TCRD*18*, and *TCRD*19* were 0.002, 0.424, 0.273, 0.026 0.253, and, 0.022 respectively. No deviation from the expectations according to the Hardy-Weinberg equilibrium was found. The heterozygosity and polymorphism information content were 0.68 and 0.62. The highest allele frequency among Koreans was *TCRD*15* (0.424), while *TCRD*18* (0.308) among Caucasians.

**F802**

## Single Nucleotide Polymorphisms in the Non-coding region of *CYP2E1* among Koreans

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Cytochrome P4502E1 (CYP2E1) is a major component of the microsomal ethanol-oxidizing system involved in the metabolism of a variety of foreign compounds including carcinogens. CYP2E1 has been implicated in alcohol-related liver diseases and several forms of cancer because of its contribution to oxidative stress. From the genomic DNA of two hundred forty-eight Koreans who are not relatives, single nucleotide polymorphisms at -1293 and -1053 nucleotide of the 5′ flanking region and at 7623 nucleotide of intron 6 of *CYP2E1*, which are contribute to regulation of CYP2E1 expression, were screened using *Pst*I, *Rsa*I and *Dra*I RFLP. The genotype frequencies of *CYP2E1*c1/*c1*, *CYP2E1*c1/*c2* and *CYP2E1*c2/*c2* of *Pst*I/*Rsa*I RFLP in the 5′ flanking region were 69.8%, 25.8%, and 4.4% respectively; the allele frequency of *CYP2E1*c1* was 0.827; the genotype