

**F307**      **Sequence Analysis of the Gene Operon for Hydrolytic  
Dechlorination of 4-Chlorobenzoate from *Pseudomonas* sp.  
DJ-12**

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Dechlorination is one of the critical steps for degradation of 4-chlorobenzoate (4CBA). *Pseudomonas* sp. DJ-12 is a natural isolate capable of degrading 4CBA via hydrolytic dechlorination. The genes responsible for hydrolytic dechlorination of 4CBA were sequenced from the chromosome of the organism and they are organized as an operon. The *fcba* (1518 bp), *B* (810 bp), and *C* (429 bp) genes encoding 4CBA-CoA ligase, 4CBA-CoA dehalogenase, and 4HBA-CoA thioesterase, respectively, were organized in the order *fcba-fcbB-fcbC* with an intergenic space between the *fcba* and *fcba*. A promoter-like sequence which is dependent on RpoD type sigma factor ( $\sigma^{70}$ ) was shown upstream of the *fcba*. Other ORFs (981 bp, 546 bp, and 1320 bp) homologous to transmembrane proteins (transporter) are located in the intergenic region.

**F308**      **DIFFERENCE IN TRANSACTIVATION OF RAR P53 MUTANTS cDNAs  
FROM KOREAN BREAST TUMORS**

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Single-strand conformation polymorphism analysis of polymerase chain reaction products (PCR-SSCP) was used for detection of mutations of the p53 gene in surgical specimens of human breast tumors. Six of 36 breast tumors showed mobility shifts in the analyses. These six tumors also showed loss of a normal allele. The samples were examined further by DNA sequencing. Results showed that five of them had missense mutation at codons 199, 240, 243, 250, 285 and 291, and the other one had nonsense mutation at codon 199. The mutant p53 cDNAs were generated by oligonucleotide-directed mutagenesis and expressed under the control of cytomegalovirus (CMV) promoter. The mutant p53 expression vectors were transiently cotransfected into Saos-2 or HeLa cells along with the p53-responsive reporter CosX1CAT containing multiple copies of a specific p53 binding sequence. The transcriptionally active mutant p53<sup>250A</sup> showed transcription transactivation activity indistinguishable from that of the wild type protein, while little activity was detected for the inactive mutants (p53<sup>179R</sup>, p53<sup>Δ199</sup>, p53<sup>Δ240I</sup>, p53<sup>Δ285K</sup>) in Saos-2 cells. The mutant p53<sup>Δ199</sup> having C-terminal truncation showed some transactivation in HeLa cells.