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Identification of the deletion endpoints in yeast *Saccharomyces cerevisiae* mitochondrial *oxi3* mutants

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Previous studies (genetic mapping) in our laboratory showed that large deletions were exceptionally frequent in the *oxi3* gene, a large mosaic gene coding for subunit I of cytochrome oxidase. The nature of one of these large deletions was physically analyzed by PCR and sequencing. PCR were carried out on the whole mitochondrial genomes of several mutants isolated by CsCl density-gradient ultra-centrifugation. About 450 bp fragments which are presumed to contain deletion endpoints were successfully amplified and cloned in the pUC vector. DNA sequencing around these deletion endpoints were carried out and possible deletion mechanisms underlying these large deletions will be discussed.

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Isolation and Genetic Charaterization of Absolute Polyamine-Auxotrophic *Escherichia coli* K-12 Mutants

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In an attempt to elucidate the physiological function of polyamine *in vivo*, it was sought to isolate mutant strains showing absolute polyamine-requirement for the growth. Strain KL527(Δ(*speAspeB*)Δ*speC*) was mutagenized by Tn*10* and λ*plac*Mu53. The mutant pools were screended to find mutant strains showing absolute polyamine-dependent growth(Spe$^-$). Two representative mutant strains, JL183(*speY*, *zzz*::Tn*10*) and JH6044(*speZ*::λ*plac*Mu53), were found to be Spe$^-$. They both showed no growth in the absence of added polyamine in glucose minimal medium, but restored their growth by addition of spermidine or putrescine in the medium. The strain JL183 isolated by Tn*10* mutagenesis was found to have no linkage between Spe$^-$ phenotype and Tn*10*(Tc^r). Therefore, it is likely that this strain acquired *speY* mutation spontaneously. The *speY* locus was genetically mapped to locate between 90.75 min and 91.5 min in the *E. coli* chromosome. The *speY* muation was transduced in KL527 using the *malE*::Tn*10kan* as a nearby positive selection marker. All of the *speY* transductants showed Spe$^-$ phenotype like JL183. Therefore, it is concluded that the *speY* mutation in the Δ(*speAspeB*)Δ(*speC*) background gives strong Spe$^-$ phenotype. The other Spe$^-$ mutant JH6044 showed 100% linkage between λ*plac*Mu53(Km^r) and Spe$^-$ phenotype. Although the genetic map location of the *speZ*::λ*plac*Mu53 was not determined, it was not co-transducible with the *malE*::Tn*10kan* nearby the *speY* gene. Therefore, it is unlikely that *speY* and *speZ* mutations locate in the same gene.