NOVEL MECHANISM OF PRECISE EXCISION OF COMPOSITE TRANSPOSONS: RECOMBINATION EXCISION OF TRANSMIDS

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Precise excision of composite transposons bearing long inverted repeats (of Tn5 type) is provided by a slippage mechanism (Brunier et al., 1988). For transposons with IS elements in direct orientation (of Tn9 type) an alternative recombination mechanism has been shown (Goryshin et al., 1991). Making use of the experimental systems with mobile elements localized on plasmids the evidence for recombination mechanism of the precise excision of Tn5 or its IS50 module has been given.

The recombination *in trans* occurs between short direct repeats of transposons localized on different plasmids which results in formation of a "specialized dimer structure". The dimer is resolved with the aid of homologous recombination. The latter does not depend on the RecA protein or transposase. Moreover the insertion of a plasmid *ori* of replication into transposon (we termed such a structure as "transmid") results in domination of the recombination mechanism of excision even for transposons with long inverted repeats. This *ori* creates conditions which appear to be unfavorable for stem-loop formation preceding the slippage process.

The transmids bearing originally a short direct repeat flanked by IS elements appeared to be a powerful tool for the analysis of topological problems arising during the binding of tansposase complex with the ends of IS elements. Varying the size of the "short direct repeat" it became possible to find the optimal length of the donor DNA site which is necessary to initiate transposition.