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The Xer site-specific recombination system catalyses a cut and paste rearrangement of circular DNA. The system s role in the bacterial cell requires that it is able to distinguish a substrate in which two participating recombination sites are on the same circle, from one in which the participating sites are on separate circles. We have addressed the basis of this topological selectivity experimentally and mathematically.

The *in vitro* system converts an unknotted substrate into a 4-crossing link. In order to expand the topological data available, knots and links of different topologies were constructed experimentally, tested as substrates and the crossing number of the reaction products was determined. We wish to find a single reaction mechanism which accounts for the topological change common to all of the reactions catalysed by Xer. We used the tangle model for site-specific recombination (based on low-dimensional topology) to compute a finite list of possible enzymatic mechanisms consistent with the experimental data. We give arguments to favor some mechanisms over others. In the case of unknotted substrates we show that the three tangle solutions may correspond to three different projections of the same 3-dimensional topological mechanism. (Received October 05, 2004)