Modeling DNA unlinking by Xer recombination

Mariel Vazquez
Mathematics Department, San Francisco State University
mariel@math.sfsu.edu

Multiple cellular processes, such as DNA replication and transcription, affect the topology of DNA. Controlling these changes is key to ensuring stability inside the cell. Changes in DNA topology are mediated by enzymes such as topoisomerases and site-specific recombinases. We use techniques from knot theory and low-dimensional topology, aided by computational tools, to analyze the action of such enzymes. I will here present recent advances in our study of DNA unlinking by XerCD-FtsK.
XerC and XerD are site-specific recombinases of *Escherichia coli*. Xer recombination is responsible for resolving DNA dimers formed by homologous recombination, thus allowing proper segregation of chromosomes and DNA plasmids at cell division. In Grainge et al. (2007) it was shown that, when coupled with FtsK, in addition to resolving DNA dimers, the site-specific recombinases XerC/XerD can unlink DNA catenanes. The authors proposed a stepwise model of unlinking. We show that, under suitable assumptions, the proposed model is the only mathematical solution to the XerCD/FtsK tangle equations.
This is joint work with D.J. Sherratt, I. Grainge, K. Shimokawa and K. Ishihara. Research is funded by NIH grants 2S06 GM052588 and 20123550 U56.