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Analysis of the inhibition mechanism of extension of RecA filament by RecX.

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Homologous recombination is responsible for maintenance of genomic integrity, because it repairs the serious DNA damage such as double strand DNA (dsDNA) break (DSB) by using homologous region of the genome as template. On the other hand, result of homologous recombination sometimes causes genomic rearrangement such as chromosomal translocation. Therefore, initiation and termination of homologous recombination must be regulated strictly. In bacteria, RecF, RecO and RecR protein enhance the filament formation of RecA on the SSB-coated ssDNA at DNA damage site. Followed by extension of the RecA filament, homologous recombination is progressed. In contrast to RecFOR, RecX protein is known as a negative regulator of homologous recombination. It has been reported that RecX inhibits several RecA activities such as DNA strand exchange, coprotease, and ssDNA-dependent ATP hydrolysis activity by blocking of filament extension of RecA. In general, it is thought that the inhibition mechanism of the extension of RecA filament is capping of the growing end of RecA filament by RecX (Ref., Figure 1). However, detailed mechanism of the capping model is still unclear. In this study, we examined RecX–protein interaction by NMR analysis. Based on the results of this study, we discuss the inhibition mechanism of filament extension of RecA by RecX.

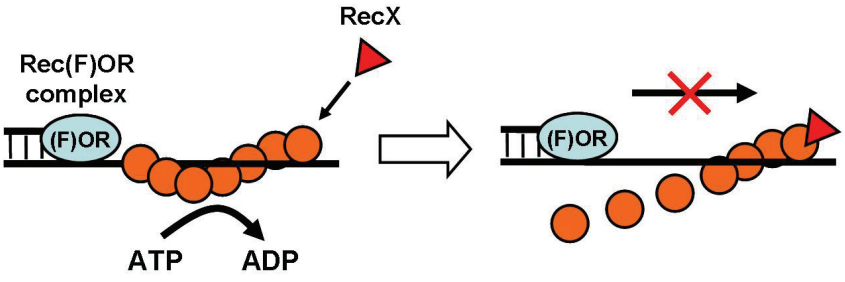

Figure 1. Model of RecA capping mechanism by RecX

Reference

Drees *et al*., (2004) *Mol. Cell*. **15**. 789-98