

P005 A simple model of intragenomic conflict explains the evolution of recombination hotspots

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The discovery of the molecular mechanisms behind recombination hotspots has unveiled an evolutionary puzzle. Namely, the self destructive nature of recombination hotspots (in their most accepted definition) raises the question how did they evolve and how are they maintained.

Redfield and others showed that if the initiation of recombination is stimulated in cis, recombinogenic sites are lost by gene conversion. Consequently, hotspots would be weeded out from the population in a short period of time. In Redfield's model recombination is not only evolutionarily unstable but also cannot invade.

Archetti argued that because it is a topoisomerase that induces the initiation of recombination, recombinogenic alleles may be considered "selfish" and intragenomic conflict can explain the persistence of hotspots. In his model recombination can both invade and be maintained, even at a cost for the rest of the genome. Archetti's model however failed to explain why the "hot" alleles are lost as a result of gene conversion and why the distribution of hotspots varies.

Here we present a new model that not only explains the origin and maintenance of recombination but also the loss of hot alleles by gene conversion and the rapid change in the distribution of hotspots in the genome.