

P043 Live hot, die young: transmission distortion in recombination hotspots

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There is increasing evidence that hotspots of meiotic recombination in humans, as well as in other organisms, are transient features of the genome. It has been suggested that this observation could be explained by biased gene conversion in favour of alleles that locally disrupt hotspots. We investigate the implications of such bias on hotspot evolution, concentrating on the human case. Our results demonstrate that biased gene conversion is a sufficiently strong force to produce the observed lack of sharing of intense hotspots between species, even if there are few sites where hotspot-disrupting alleles can arise, but that sharing may be much more common for weaker hotspots. Even direct selection for higher recombination rates, such as to ensure correct segregation during meiosis, is ineffective at conserving individual hotspots. Under simple models of hotspot genesis, only models where hotspots are initially shielded from drive are found to be consistent with the observed distribution of hotspot intensities. We investigate the affect of an allele that influences the intensity of a hotspot on patterns of linkage disequilibrium, and find that such alleles will be difficult to observe based solely on the patterns found in population data.