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Selected Publication

Garcia, V., Gray, S., Allison, R.M., Cooper, T.J., and M. J. Neale. Tel1/ATM-mediated interference suppresses clustered meiotic double-strand break formation. *Nature* 520, 114-118 (2015) <http://dx.doi.org/10.1038/nature13993>

Research Aims and Interests

Research in the Neale lab concerns the mechanism, evolution, and regulation of DNA recombination during meiosis. At the heart of this fundamental, evolutionarily-conserved process resides a fascinating array of chromosome gymnastics: chromatin remodeling; pairing of maternal and paternal chromosomes; reassortment of DNA segments within these pairs; and, ultimately, chromosome segregation. Collectively, these steps generate unique, recombinant, haploid genomes suitable for sexual reproduction, and ensure the propagation of genetically diverse populations.

Our highlighted work investigates the spatial regulation of meiotic recombination — specifically dissecting those mechanisms that ensure that recombination events are distributed relatively evenly across all chromosomes. We identified the DNA damage response kinase, Tel1/ATM, as a negative regulator of local recombination activity — an activity that appears critical to prevent recombination events from clustering within discrete regions of the chromosome defined by higher-order structure. We study these phenomena in the sexually-reproducing single-celled eukaryote, *Saccharomyces cerevisiae*, which acts as both a fascinating example of how hierarchical layers of regulation “imprinted” within the chromosome can influence diverse processes, and also as a generalized model of the mechanisms that arise in many other organisms, including mammals.