Genome Sciences Seminar

Wednesday, 10.25.17 | 3:30 | Foege Auditorium

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“Centromeres impersonating telomeres, and other forms of deception at the chromosome end”

Cooper Lab:

We study the mechanisms by which chromosome integrity is maintained starting at the telomere, the nucleoprotein complex that forms the end of the chromosome. Our lab focuses on how telomeres protect chromosomes from degradation and fusion, as well as alternative strategies for chromosome end protection in the absence of canonical telomeres. We also study the newly emerging roles of telomeres in controlling both spindle and centromere assembly during meiosis. Most of our studies utilize fission yeast, a versatile model system with telomeres and other heterochromatic regions that are highly conserved with those of human. Telomeres protect our genomes and choreograph chromosome movements; they affect a range of biological processes from cancer avoidance to healthy gamete formation. They prevent the degradation and fusion of chromosome ends and ensure that natural chromosome ends do not elicit the cell-cycle arrest pathways that respond to damage-induced DNA breaks. Telomeres are a particular focus of our research on tumorigenesis (which is associated with genomic instability and telomerase activation) and aging (which is accompanied by a progressive decline in telomere length). However, the complete repertoire and underlying mechanisms of telomere function are not yet understood. The telomeres of fission yeast, the model system we use most in our lab, are remarkably similar to those of humans but provide precise genetic manipulability. Concepts we have developed by studying fission yeast telomeres have repeatedly been shown to predict observations in mammals.

Topics we are exploring include the cell-cycle regulation of telomere function, the fascinating roles taken on by telomeres in controlling spindle formation and centromere assembly during meiosis, and surprising ways in which some cells (such as telomerase-minus cancer cells) can survive in the absence of canonical telomeres.