Johns Hopkins University

Department of Biology Seminar Series

Thursdays, 4:00pm

For more information go to: https://bio.jhu.edu/events

Zoom link: https://zoom.us/j/97925356454?pwd=bjNuTlY1dU9BcXcvRFdleis2TVNadz09

May 18th, 2023 - Mudd 100

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Host: Mark Van Doren

"Non cell autonomous control of germline stem cell homeostasis"

My lab is interested in the role of autophagy in development. Specifically, we focus on the role of autophagy in of the germline development. Autophagy is a conserved cellular recycling process, where cellular material destined for degradation is enclosed in the autophagosome, which fuses with the lysosome for degradation and recycling. Although typically upregulated in response to cellular stress conditions, basal levels of autophagy can also function as a quality control mechanism that serves to maintain cellular homeostasis in response to developmental and environmental cues. We recently described roles for autophagy genes in establishing the stem cell population in the proliferative zone (Ames et al., 2017). We are now investigating the mechanism(s) by which autophagy genes promote meiotic fidelity and DNA damage repair in germline development. Our current work seeks to elucidate where autophagy genes function to regulate germline development, and how autophagy is required for meiotic fidelity and the DNA damage response.

The extracellular matrix is important for stem cell biology. Heparan sulfate (HS) is an extracellular glycan of great molecular diversity, derived from complex, non-uniform modifications of the glycan chains. The sugar residues along the chains can be modified by dedicated HS modifying enzymes, which introduce modifications such as sulfation, deacetylation and epimerizations in various positions, with nearly boundless combinatorial possibilities. We have found that distinct combinations of heparan sulfate modifications are involved in regulating the homeostasis of C. elegans germ line stem cells. Specifically, mutations in certain modification enzymes increase, whereas mutations in genes coding for other modification enzymes decrease the number of stem cells in the progenitor zone of the C. elegans adult germline. Intriguingly, these effects may be mediated by regulating several signaling pathways, such as Fibroblast Growth Factor signaling and GLP-1/Notch. At least some of the functions of HS are non-autonomously controlling proliferation of stem cells in the germline. Collectively, these findings suggest that distinct HS modification patterns display differential effects on germline stem cell homeostasis, likely by modulating signaling between the germline niche and the germline stem cells.