Identifying Kinase Genes Involved in Regulation of the Stem Cell versus Meiotic Fate Decision in the C. elegans Germline

Vahag Kechjian

Washington University in St. Louis

Follow this and additional works at: https://openscholarship.wustl.edu/wushta_spr2017

Recommended Citation
https://openscholarship.wustl.edu/wushta_spr2017/63

This Abstract for College of Arts & Sciences is brought to you for free and open access by the Washington University Senior Honors Thesis Abstracts at Washington University Open Scholarship. It has been accepted for inclusion in Spring 2017 by an authorized administrator of Washington University Open Scholarship. For more information, please contact digital@wumail.wustl.edu.
Germline stem cells make a decision between maintaining the stem cell fate or entering meiosis. In the nematode *Caenorhabditis elegans*, germ cells adjacent to the somatic niche, the distal tip cell (DTC) of the adult germline, maintain the stem cell fate through the action of the GLP-1 Notch signaling pathway, while germ cells away from the influence of the DTC enter meiosis. *glp-1* maintains the stem cell fate through repression of the GLD-1 and GLD-2 pathways, which function redundantly to promote meiotic entry. The mechanism whereby *glp-1* signaling represses the GLD-1 and GLD-2 pathways is not completely understood, although it is known that regulation of the GLD-1 and GLD-2 pathways is post-transcriptional. I postulated that individual kinase genes, which act post-translationally, may function to promote meiotic entry in the *C. elegans* germline by promoting GLD-1 and/or GLD-2 pathway activity or inhibiting *glp-1* activity. With the exception of *glp-1*, due to genetic redundancy, loss of function of known single genes does not have a significant effect on the stem cell versus meiotic fate decision. Therefore, I used a sensitized genetic background, *glp-1*(ar202), to identify kinases, following RNAi knockdown, that result in a tumorous germline due to failure to switch to the meiotic fate. RNAi of 276 *C. elegans* kinase genes identified 26 that show incompletely penetrant germline tumor phenotypes. A deletion mutant for one kinase, *unc-82*, crossed into *glp-1*(ar202) background, significantly increased germline overproliferation, confirming that *unc-82* acts to promote GLD-1 and/or GLD-2 activity or inhibit *glp-1*. Future experiments will determine if deletion mutants of any of the remaining kinase genes results in the formation of germline tumors.