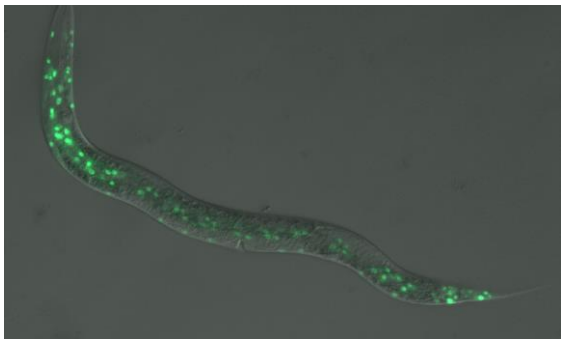
GUEST SPEAKER SEMINAR

Molecular Genetics
UNIVERSITY OF TORONTO

FOXO/*daf-16* coordinates multipotency and quiescence in *C. elegans* stem cell models



Multipotency, the ability of stem cells to produce multiple cell types, is critical for the development and maintenance of tissues and organs. Upon the completion of development, adult stem cells retain their multipotency during lengthy periods of cellular quiescence (reversible cell cycle exit) to replace cells lost through tissue damage or normal turnover. Maintenance of multipotency during quiescence is critical to stem cell function, yet the mechanisms linking multipotency to quiescence remain poorly understood. One candidate for this role is the FOXO family of transcription factors that promotes quiescence and stem cell maintenance. In *C. elegans*, *daf-16*, the FOXO ortholog, promotes entry into the stress-resistant and developmentally arrested dauer larva stage. Dauer is adopted midway through development in response to adverse environmental conditions, and can serve as a model for quiescence. Remarkably, phenotypes caused by mutations in developmental pathways can be corrected during dauer arrest, suggesting the existence of active mechanisms that re-set cell fate during dauer. Using two *C. elegans* progenitor cell types, we show that *daf-16* is part of these mechanisms. *daf-16* blocks the activity of signal transduction and microRNA pathways in order to maintain or re-establish multipotency during dauer. Thus, *daf-16*/FOXO coordinates quiescence with multipotent cell fate.

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Host: Dr. Julie Claycomb

Date: Thursday August 16, 2018
Time: 10:30 AM
Place: MaRS2 Rm.1522