LIN-61 regulates growth and lipid homeostasis via drl-1 suppression in C. elegans

Dietary (caloric) restriction in mammals is associated with reduced risk of disease/cancer for incompletely understood reasons. DR can be induced in a C. elegans model to better examine the molecular controls of the metabolic tradeoffs between growth, reproduction, and lipid homeostasis. Mutation of drl-1, a MAP kinase orthologous to human MEKK-3, mimics a dietary restricted state in C. elegans. drl-1 mutants have been previously demonstrated in the Dowen lab to produce a slow-growing, lipid-devoid animal with down-regulation in vitellogenin expression. lin-61 is a chromatin factor identified in an EMS screen of drl-1 mutants which is a genetic suppressor of DRL-1. Genetic mutation and RNAi knockdown of lin-61 in drl-1 mutants suggest that LIN-61 antagonizes vit gene expression and growth rates in C. elegans. Further experimentation will be required to elucidate the exact mechanisms by which LIN-61 interacts with DRL-1 to regulate the metabolic tradeoffs between these key processes.