Potential ufd-2 control of vitellogenesis in C. elegans

As of yet, ubiquitination proteins play an unidentified role in the mTORC2 pathway in C. elegans nematodes, though their role in mammals is better understood. Preliminary testing in genetic backgrounds mutant for groundhog 3 and 4 proteins (GRD-3/4), which enhance loss of vitellogenesis phenotypes, have suggested that ufd-2 RNAi results in a reduction in vitellogenin expression, possibly by reducing the activity of the mTORC2 pathway. In this study we investigate this relationship by recreating a deletion mutation in ufd-2 and attempt to determine whether mutation of ufd-2 enhances grd-3; grd-4 mutants of if ufd-2 alone is required for vitellogenesis, both in a grd-αæ mutant background and in a wild type background. Preliminary testing indicated that loss of ufd-2 function by RNAi induced a mild loss of vitellogenin production; however, isolation and characterization of ufd-2 genetic mutants is ongoing. This talk discusses the phenotypes observed through RNAi knockdown of ufd-2, the phenotypes observed due to the mutation of the ufd-2 pathway, and the implications of these for the function of UFD-2 in the mTORC2 pathway.