

Investigating the Contribution of prp-17 and Other Splicing Factors to Maintaining Fertility in *Caenorhabditis elegans*

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Caenorhabditis elegans hermaphrodites seed stem cells of their germlines to reproduce. The proliferation of these stem cells is controlled in a large part by the FBF genes (*fbf-1* and *fbf-2*). The goal of this research was to investigate the role of *prp-17* in maintaining fertility in *C. elegans* and its possible interaction with FBF. The original assumption was that *prp-17* was either a cofactor of one of the FBF genes or was providing independent regulatory input separable from FBF function. These hypotheses were tested using three worm mutants (*rrf-1*, *rrf-1;fbf-1*, and *rrf-1;fbf-2*) as well as transgenic versions of each mutant. The transgene used was designed to reflect the expression of *fog-1*, one of the genes that FBF downregulates in the germline's stem cells. Assays were conducted in which *prp-17* was silenced in each worm mutant using *Escherichia coli* bacteria to deliver dsRNA of *prp-17* to the worms, triggering RNAi. Assays revealed higher levels of sterility among the FBF mutants than among the *rrf-1* mutants, as well as higher levels of Masculinization of the Germline (MOG). MOG is the phenotype associated with FBF function disruption; therefore this data suggests that *prp-17* is involved somehow with both FBF genes. Assays using transgenic worms showed that removal of *prp-17* hindered *fbf-1* and *fbf-2*'s ability to downregulate *fog-1* albeit in different ways (possibly indicating differences in how they function). The data points to the conclusion that *prp-17* promotes activity of both FBF genes, but is not a selective cofactor of either one of them. Assays using *rrf-1;fbf-2* transgenic worms showed increased *fog-1* levels in the pachytene zone of the germline, indicating *fbf-1* activity farther down the germline than previously thought.