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.