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

Novak, Preston

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.

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