

Loss of *mir-244* Suppresses *alg-1* Developmental Timing Phenotype

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In order to identify pathways regulated by microRNAs in *Caenorhabditis elegans*, multiple strains were built composed of deletions in a specific microRNA gene and *alg-1*. *alg-1* encodes an Argonaute protein which functions in the biosynthesis and activity of miRNAs. In an *alg-1* background, expression levels of miRNAs are generally reduced and thus single *alg-1* mutant worms display developmental timing defects as seen through defective alae formation, misexpression of *col-19::GFP*, and extra seam cells. Malformed alae are characterized by gaps in which it is irregularly patterned along the anterior-posterior axis of the worm. *col-19::GFP* is misexpressed in a discontinuous fashion among hypodermal cells, namely *hyp7* and seam. Typically two additional seam cells at the L4 stage of development suggests these single mutant worms repeat L2 stage programming. Contrary to *alg-1* worms, *alg-1*; miR-244 double mutant worms display less developmental timing defects. These worms generally display more complete alae patterning and continuous GFP expression in addition to reflecting wild type total seam cell numbers at the L4 stage. These observations have led to the assumption that developmental timing errors are suppressed in double mutant worms. The goal of this project was to characterize the pathway in which miR-244 functions and, in the future, identify specific downstream targets for miR-244.