

*Screening for and characterization of
mir-48 suppressors in C.elegans*

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Control of developmental timing is a subject of interest because there are many precisely orchestrated changes an organism must go through before it can reach its mature state. The question of how developmental events are timed within organisms has been a question asked by biologists for many years. The nematode *C. elegans* is one of the best model organisms for this type of research. They are easy to grow, transparent, and the developmental fate for each cell has been mapped out. The essentially invariant cell lineage allowed for the discovery of heterochronic genes that time specific events as the organism grows. Among these genes were the first microRNAs discovered in any system. These ~21 nt RNAs act post-transcriptionally to regulate gene expression. Overexpression of one *C. elegans* miRNA, *mir-48*, causes adult cuticle to form precociously, during larval stages, and also alters vulva development, ultimately disabling egg-laying. To identify additional players in *mir-48*-mediated control of developmental time, we searched for suppressors of the *mir-48* overexpression phenotype. We performed a chemical mutagenesis and screened for animals that had regained the ability to lay eggs. The genes identified by these mutations could encode transcriptional activators of *mir-48*, proteins involved in miRNA processing, and *mir-48* target genes with mutations in their miRNA binding sites. We identified over 20 mutants with restored egg-laying and characterized their phenotypes. We are in the process of outcrossing these mutants with wild-type animals to determine if the mutations cause timing phenotypes in the absence of *mir-48* overexpression. One mutation causes a phenotype on its own, and turned out to be an allele of *lin-66*. We hope that by continuing our research we will identify additional members of the pathway.

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