CHARACTERIZATION OF THE CLASS A SYNTHETIC MULTIVULVA GENES *LIN-8* AND *LIN-56*

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The receptor tyrosine kinase/Ras pathway essential for vulval induction in *C. elegans* is negatively regulated by two redundant pathways. Hermaphrodites mutant in only one of these two pathways (A or B) appear wild-type for vulval induction. Hermaphrodites mutant in both pathways (A and B) exhibit the synthetic Multivulva (synMuv) phenotype. Various screens for multivulva animals have defined four genes in the synMuv class A pathway: *lin-8*, *lin-15A*, *lin-38*, and *lin-56*. Of these genes, only the *lin-15A* locus was cloned previously; *lin-15A* encodes a novel protein with no recognizable functional or structural motifs (1).

The class B synMuv genes inhibit Ras-mediated vulval development via an Rb/E2F/DP-mediated pathway, which suggests that the transcriptional repression of genes required for vulval development is the molecular mechanism of this inhibition (2). The class A synMuv genes function in parallel to this Rb pathway, but the molecular mechanism by which they inhibit vulval development is not known. The recent identification of *egr-1* and *egl-27* as possible class A synMuv genes suggests that the synMuv class A pathway may also act via transcriptional repression, as *egr-1* and *egl-27* are homologous to MTA1, a component of the mammalian NURD chromatin remodeling complex (3). In addition, evidence from two-hybrid experiments suggests that the products of class A and class B synMuv genes may be associated in a complex *in vivo* (4).

To further our understanding of the mechanism by which the class A synMuv genes inhibit the Ras pathway, we have cloned *lin-56* and *lin-8.* Both appear to encode novel, highly-charged proteins. We have identified molecular lesions associated with both *lin-56* alleles and with all but one of the nine *lin-8* alleles. We are working to characterize the expression patterns of *lin-56* and *lin-8* and plan to determine their sites of action using mosaic analysis.

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