**51. Characterization of the Cell Deaths Caused by Mutations in *lin-24* and *lin-33***

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Mutations in the genes *lin-24* and *lin-33* can semidominantly cause the inappropriate deaths of Pn.p cells. Some Pn.p cells are vulval precursor cells, and *lin-24* and *lin-33* mutations can result in a vulvaless phenotype. The Pn.p cell deaths caused by *lin-24* and *lin-33* mutations are morphologically distinct from both programmed cell deaths and the necrotic deaths observed in strains containing certain mutant alleles of genes that encode degenerin ion channels (*mec-4*, *deg-1, mec-10,* and *unc-8*). Interestingly, the inappropriate deaths of the Pn.p cells in *lin-24* and *lin-33* mutants require a subset of the genes necessary for programmed cell death, including at least some of the genes required for corpse engulfment.

We have cloned *lin-24* and *lin-33. lin-24* encodes a protein containing an Aerolysin toxin domain, while *lin-33* encodes a novel protein. Aerolysin-like toxins are cytolytic toxins made predominantly by bacteria and act by destroying the membrane permeability barrier of eukaryotic cells and causing osmotic lysis.

A deletion allele we isolated of *lin-24* and an intragenic suppressor we isolated of *lin-33(n1043)* each result in a wild-type phenotype. Genetic analyses suggest that the deaths caused by a semidominant mutation in one gene require the function of the other gene. Additionally, the deletion allele of *lin-24* can dominantly suppress the semidominant Pn.p death phenotypes of both *lin-24* and *lin-33* mutants. To characterize the *lin-24* and *lin-33* Pn.p cell deaths, we are analyzing these deaths by electron microscopy. We are also analyzing the expression of the *lin-24* and *lin-33* genes using *gfp* reporter constructs and an antibody raised against LIN-24. The results of ectopic expression studies, mosaic analysis, and dosage studies of these genes will be presented.