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***lin-55* DP and an E2F-like gene act in the *lin-35* Rb pathway to antagonize *let-60* ras signaling during vulval induction**

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The Ras signaling pathway of vulval induction is antagonized by the synthetic multivulva (*synMuv*) genes. On the basis of genetic interactions, the *synMuv* genes have been grouped into two classes, A and B. An animal is Muv if it has mutations in both a class A gene and a class B gene, whereas an animal has a wild-type vulva if a mutation is present in a gene or genes of a single class. *lin-35*, a class B *synMuv* gene, encodes a protein similar to the mammalian tumor suppressor pRb and the related proteins p107 and p130 (Lu and Horvitz, 1998, *Cell* **95**: 981-991). A well-characterized binding partner of pRb is the DP/E2F heterodimeric transcriptional activator. pRb/DP/E2F complexes are thought to repress transcription of DP/E2F-responsive genes, including genes required for S phase cell-cycle progression. We are determining the roles of DP and E2F proteins in the specification of vulval cell fates.

We cloned the class B *synMuv* gene *lin-55* and have found that it encodes a protein similar to the DP family of transcription factors. To investigate the null phenotype of *lin-55*, we isolated a deletion allele, *n3316*. In addition to a *synMuv* phenotype, *lin-55(n3316)* causes maternal-effect lethality.

We have also identified two *C. elegans* E2F-like genes. A deletion allele, *n3318*, of a gene we are temporarily calling *C.e.E2F-1* behaves like a class B *synMuv* mutation. *n3318* also causes maternal-effect lethality. We are currently comparing the lethal phase of *n3318* embryos with that of *lin-55(n3316)* embryos and are investigating the role of the other E2F-like gene in vulval development and embryogenesis.

We propose that LIN-55 and *C.e.E2F-1* form a complex with LIN-35 Rb and other class B *synMuv* proteins and that this complex represses transcription of genes that promote vulval development.