Abstract/Session Information for Program Number 699C

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Abstract Content

Program Nr: **699C**

The "Green Pharynx" Phenotype of Transgene Misexpression Yields New Insight into the synMuv Genes. **Hillel Schwartz**, Dawn Wendell, Bob Horvitz. HHMI, Dept. Biology, Cambridge, MA 02139, USA.

In the course of a screen to identify mutants defective in the control of the sex-specific deaths of the CEM neurons using the reporter *pkd-2::gfp* (see abstract by Schwartz and Horvitz), we found 29 independent isolates that had strong GFP expression in the pharynx, a tissue that does not normally express this reporter. From this screen and further clonal and nonclonal screens, we have identified 68 mutants with the green pharynx phenotype. This transgene misexpression is not dependent on chromosomal integration, high transgene copy number, or choice of co-injection marker, and the phenotype can be seen with at least one other GFP reporter that contains a different promoter.

We found that mutations in certain synthetic Multivulva (synMuv) genes (see abstracts by Andersen and Horvitz and by Harrison and Horvitz) could produce the green pharynx phenotype. The synMuv genes act to inhibit vulval development. Animals mutant in both a class A gene and a class B synMuv gene, but not animals mutant in one or more class A genes or in one or more class B genes, display a Multivulva phenotype. Several class B synMuv genes have been cloned and shown to encode genes with homologs implicated in transcriptional silencing and chromatin remodeling. The synMuv genes able to cause *pkd-2::gfp* expression in the pharynx include one class A synMuv gene and two class B synMuv B genes, a result that contrasts with the finding that genes in the A and B synMuv pathways act separately and in parallel to prevent vulval cell fates. Mutations in 26 other synMuv genes have been tested and do not cause the green pharynx phenotype. Of the 68 mutations isolated based upon this phenotype, 67 appear to be alleles of these three genes.

A fourth gene, defined by a single allele, *n3599*, caused an identical phenotype of transgene misexpression. *n3599* mutants are not synMuv A or synMuv B. Interestingly, *n3599* is synthetically lethal with a subset of synMuv B mutations, including *lin-35 Rb*. This subset does not correspond to other subsets associated with other phenotypes, including the subset of synMuv B mutants defective in regulation of the cell cycle (Boxem and van den Heuvel, Current Biology 12: 906-11, 2002; Fay, Keenan, and Han, Genes and Development 16: 503-17, 2002). It is possible that the synMuv genes that are synthetically lethal with *n3599* share a normal function distinct from both vulval development and cell cycle regulation but nonetheless involving transcriptional repression.

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