

A New Genetic Screen for Hermaphrodites with Inappropriately Surviving CEM Neurons

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Two sexually dimorphic cell deaths occur during *C. elegans* development. The HSN neurons survive in hermaphrodites, in which they are required for egg laying, but die in males. The CEM neurons survive in males, in which they may play a role in chemotaxis towards hermaphrodites, but die in hermaphrodites. Genetic studies have identified one gene, *ceh-30*, that plays a key role in regulating the CEM neuron sexually dimorphic survival. *ceh-30* is required for CEM survival in males, and *ceh-30* gain-of-function mutations cause CEM survival in hermaphrodites (see abstract by Schwartz and Horvitz).

We have begun a screen to identify new genes that regulate the hermaphrodite-specific programmed cell death of the CEM neurons. Previous screens for CEM survival in hermaphrodites recovered numerous mutations that masculinized hermaphrodites. The CEM neurons of these animals survived because they adopted the male CEM survival fate, not because of a defect in programmed cell death. As *ceh-30* appears to act downstream of the sex-determination genes, mutations that masculinize *ceh-30* loss-of-function (lf) mutant hermaphrodites should not cause CEM survival. By screening for CEM survival in a *ceh-30*(lf) mutant background we should be able to eliminate the recovery of a large number of mutations in genes functioning in sex determination.

To date, we have recovered at least 27 independent mutations that may disrupt the execution of programmed cell death or prevent proper CEM neuron cell fate. We are currently performing complementation tests between these isolates and alleles of the programmed cell death genes *egl-1*, *ced-4*, and *ced-3*, which are known to cause CEM neuron survival in hermaphrodites.

Poster

Session Topic: Cell death and neurodegeneration

Second Session Topic: Cell fate specification - embryo

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