Abstract/Session Information for Program Number 99

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Abstract Content

Program Nr: **99**

The *BarH* Class Homoeodomain Gene *ceh-30* is Directly Regulated by *tra-1* to Specify the Sexually Dimorphic Survival of the CEM Neurons. **Hillel Schwartz**, Bob Horvitz. HHMI, Dept. Biology, Cambridge, MA 02139, USA.

While many genes involved in the execution of cell death have been identified, the mechanisms that control the commitment of specific cells to undergo programmed cell death are poorly understood. To identify genes that act in the specification of cell death, we performed a screen for hermaphrodites in which the male-specific CEM neurons, which die during normal hermaphrodite development but survive in males, fail to die. Using *pkd-2::gfp* as a marker for CEM survival, we screened 60,000 mutagenized haploid genomes and recovered at least 154 independent mutations that cause survival of the CEMs, including at least 42 alleles of known cell-death genes and 64 mutations that cause sexual transformation.

Three mutations from this screen, *n3713*, *n3714*, and *n3720*, semidominantly cause CEM survival in hermaphrodites, but cause no other obvious defects in programmed cell death or sex determination. We found that these mutations affect a previously uncharacterized gene on LGX. The CEM survival caused by these mutations is not affected by loss of the fem genes, the most downstream genes required for masculinization, indicating that this gene may act downstream of sex determination. The *n3714* CEM survival phenotype is not affected by a duplication, indicating *n3714* causes increased wild-type function or altered function.

In a screen for suppressors of *n3714*, we found one mutation, *n4111*, that is tightly linked to *n3714* and acts as a dose-sensitive suppressor of the CEM survival phenotype of *n3714*. In contrast to *n3714* hermaphrodites, which inappropriately have surviving CEMs, *n4111 n3714* males are inappropriately lacking CEMs. The CEMs of *n4111 n3714* males are restored by mutations preventing programmed cell death, but not by a null mutation in *tra-1*, a gene required for feminization and the most downstream gene in the sex determination pathway. Other deaths and sexually dimorphic characteristics are not affected by *n4111 n3714*.

By performing transformation-rescue experiments and determining DNA sequences, we found that *n4111* is a nonsense mutation in the BarH class homeodomain gene *ceh-30*. *n3713*, *n3714*, and *n3720* mutate an evolutionarily conserved TRA-1 binding site in an intron of *ceh-30*. We propose that *ceh-30* is specifically required for CEM survival in males and that in hermaphrodites *ceh-30* is prevented from protecting the CEMs by direct transcriptional repression by TRA-1. It remains to be determined how *ceh-30* protects the CEMs and to what extent this function of *ceh-30* is shared by *BarH* class homeodomain genes in other organisms.

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Abstract Information

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