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1999 International Worm Meeting abstract 389

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## ***n3194*, identified in a screen for suppressors of *ced-9(n1950)*, may define an ion channel required for cell viability**

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To identify genes that may interact with the cell-death protector gene *ced-9*, we screened for suppressors of the Ced phenotype of *ced-9(n1950gf)* animals by looking for the reappearance of corpses in *ced-9(n1950)* embryos. We identified one candidate, *n3194*, with a large number of refractile bodies in embryos. *n3194* maps to LGIII, complements *ced-9(lf)*, and causes a recessive maternal-effect lethal phenotype. A second allele, *n3264*, was isolated in a screen for engulfment-defective mutants (see abstract by Z. Zhou) and does not cause maternal-effect lethality. A third allele, *zu223*, was isolated in an unrelated screen for maternal-effect lethals (J. Priess, personal communication). Neither the accumulation of refractile bodies nor the lethality of *n3194* is suppressed by loss-of-function mutations in *ced-3* or *ced-4*. The extra dying cells are acridine-orange positive but not TUNEL-positive, distinguishing them from normal programmed cell deaths. We examined the morphology of the refractile bodies in *n3194* mutants using electron microscopy and determined that the bodies resemble degenerative deaths rather than apoptotic deaths.

*n3194* resides in the gene R13A5.1, which encodes a protein highly similar to the products of human and mouse ESTs of unknown function with weak similarity to K<sup>+</sup> and Ca<sup>2+</sup> channels. We identified nonsense mutations in *n3194* and *zu223* and a missense mutation altering a conserved residue in *n3264*.

An R13A5.1-GFP fusion capable of rescuing *n3194* is expressed on the plasma membrane of all cells in early embryos. Later the GFP marker becomes restricted to the excretory canals and several neurons in the head. These neurons have cell bodies directly posterior to the anterior bulb of the pharynx and extend processes into the nose.

R13A5.1 appears to be required for cell viability during embryogenesis. Its observed localization in the excretory canal and its homology to ion channels suggest a role in maintaining ionic homeostasis.