***ced-11* Is Required for the Morphological Appearance of Apoptotic Cell Corpses**

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Programmed cell death is a fundamental process required for proper development and tissue homeostasis in many organisms. Genetic analyses of programmed cell death in *C. elegans* led to the discovery of an evolutionarily conserved genetic pathway that regulates the activation of apoptosis. A cell dying by apoptosis undergoes a series of morphological changes resulting in the appearance of round refractile cell corpses, as visualized by Nomarski optics. *ced-11* was identified in a screen for mutations that alter the morphological appearance of cell corpses. The corpses of *ced-11* mutant embryos are non-refractile as visualized by Nomarski optics.

We have found that while mutations in *ced-11* do not prevent cells from dying, they can enhance the ventral-cord cell-death defect of weak alleles of other cell-death genes. This observation indicates that *ced-11* plays a role in the cell death process. *ced-11* acts downstream of the CED-3 caspase and appears not to have an effect on engulfment. *ced-11* encodes a protein with similarity to members of the TRP family of non-selective cation channels. As TRP channels are often permeable to calcium, we tested if *ced-11* regulates calcium during apoptosis. We used GCaMP3, a genetically-encoded calcium indicator, to monitor calcium in dying cells. In wild-type embryos refractile corpses that express GCaMP3 have bright fluorescence throughout the corpses. In *ced-11* mutants corpses that express GCaMP3 have bright fluorescence in the cytoplasm but a reduction of fluorescence in the nucleus. In addition, in wild-type embryos a long-lasting increase of fluorescence coincides with the onset of refractility of the corpses. By contrast, *ced-11* corpses have occasional transient bursts of fluorescence in the cytoplasm. This observation suggests that *ced-11* might act as a calcium-permeable channel to regulate the entry of calcium into the nuclei of cells undergoing apoptosis. Alternatively, *ced-11* might regulate the breakdown/integrity of the nuclear envelope and thus allow calcium into the nucleus of apoptotic corpses. We plan to determine how *ced-11* affects the entry of calcium into the nucleus of apoptotic corpses. Better understanding of the role of *ced-11* in apoptosis might help elucidate the role of calcium downstream of caspase activation and the mechanism of nuclear degradation in apoptotic cell death.

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