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Meeting abstract

Regulation of programmed cell death in plant embryogenesis

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As plants grow they not only form new tissues and structures using highly coordinated cell-division and cell-differentiation programs but also continuously kill many of their own cells through activation of programmed cell death (PCD). The earliest functions of PCD in plant life are fulfilled during embryogenesis. Here PCD governs two major developmental processes. First is the elimination of a transient embryonic structure – suspensor, which functions at early stages of embryo development as a conduit of nutrients and growth factors, but is not required at later stages [1]. Another function of embryonic PCD applies to embryo abortion, which is not only a response to stress or mutagens, but also a normal feature of those plant species, which produce polyembryonic seeds. In the latter case competition among multiple embryos for survival often induces PCD resulting in the elimination of all but one embryo in a seed [2]. At the demise, both suspensor and entire embryos display a gradient of successive stages of PCD along apical-to-basal axis [2,3]. This PCD implicates active role of autophagy in complete removal of cell protoplast. Autophagosomes are formed via Golgi and proplastids [1]. Autophagocytosis depends on the dynamic reorganization of the cytoskeleton. Microtubule network is disrupted early in PCD pathway. F-actin is gradually reorganised into thick longitudinal cables and is present till the vacuole collapse and fragmentation of nuclear DNA [1,3]. Type II metacapase is critically involved in the regulation of the cell death pathway, which is essential for normal embryo development. Metacaspase gene silencing results in the suppression of cell death and failure of embryonic pattern formation [4].

References

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