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Mechanisms of developmentally controlled cell death in plants

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During plant development various forms of programmed cell death (PCD) are implemented by a number of cell types as inherent part of their differentiation programmes. Differentiation-induced developmental PCD is gradually prepared in concert with the other cell differentiation processes. As precocious or delayed PCD can have detrimental consequences for plant development, the actual execution of PCD has to be tightly controlled. Once triggered, PCD is irrevocably and rapidly executed accompanied by the breakdown of cellular compartments. In most developmental PCD forms, cell death is followed by cell corpse clearance. Devoid of phagocytic mechanisms, dying plant cells have to prepare their own demise in a cell-autonomous fashion before their deaths, ensuring the completion of cell clearance *post mortem*. Depending on the cell type, cell clearance can be complete or rather selective, and persistent corpses of particular cells accomplish vital functions in the plant body. The present review attempts to give an update on the molecular mechanisms that coordinate differentiation-induced PCD as vital part of plant development.

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Introduction

Diverse instances of programmed cell death play fundamental roles in plants' responses to biotic and abiotic stresses, as well as during plant vegetative and reproductive development. In animals, the molecular biology of PCD has been studied comprehensively, revealing extensive networks regulating different types of PCD. In comparison, the molecular regulation of plant PCD is far less understood. Distinct forms of plant PCD can be induced by environmental stresses (ePCD) or developmental cues (dPCD). Knowledge on ePCD stems mainly from research conducted on the pathogen-triggered hypersensitive response, and on ePCD triggered by diverse abiotic stresses [1,2]. During regular plant development, dPCD is triggered as the ultimate step of cell-type specific differentiation programmes, for example, in various xylem cell types, the tapetum layer, or the root cap [3]. Furthermore, and irrespective of specific differentiation programmes, plant cells die in a developmentally controlled fashion during the final stage of senescence of plant organs. Interestingly, although dPCD does occur independent of environmental cues, the timing of differentiation-induced or senescence-related dPCD can be influenced by certain stresses [4].

The present review gives an update on the mechanistic regulation of differentiation-induced dPCD. First, we will discuss the differentiation programme that various cell types have to run through in order to achieve PCD competency (Phase I). Such plant cells will become receptive for internal or external signals that trigger a rapid PCD execution at the right place and time by still poorly understood mechanisms (Phase II). Finally, after breakdown of cellular compartmentalisation, the cellular corpse is either partially or completely removed by cell clearance processes that were initiated in Phase II, but are finalised *post mortem* (Phase III). The transitions between these phases are not always easily recognised, and processes initiated in one phase might be finalised only in the next. In this review, we will follow this chronological outline to highlight some of the molecular mechanisms involved in the control of dPCD processes in plants cells.

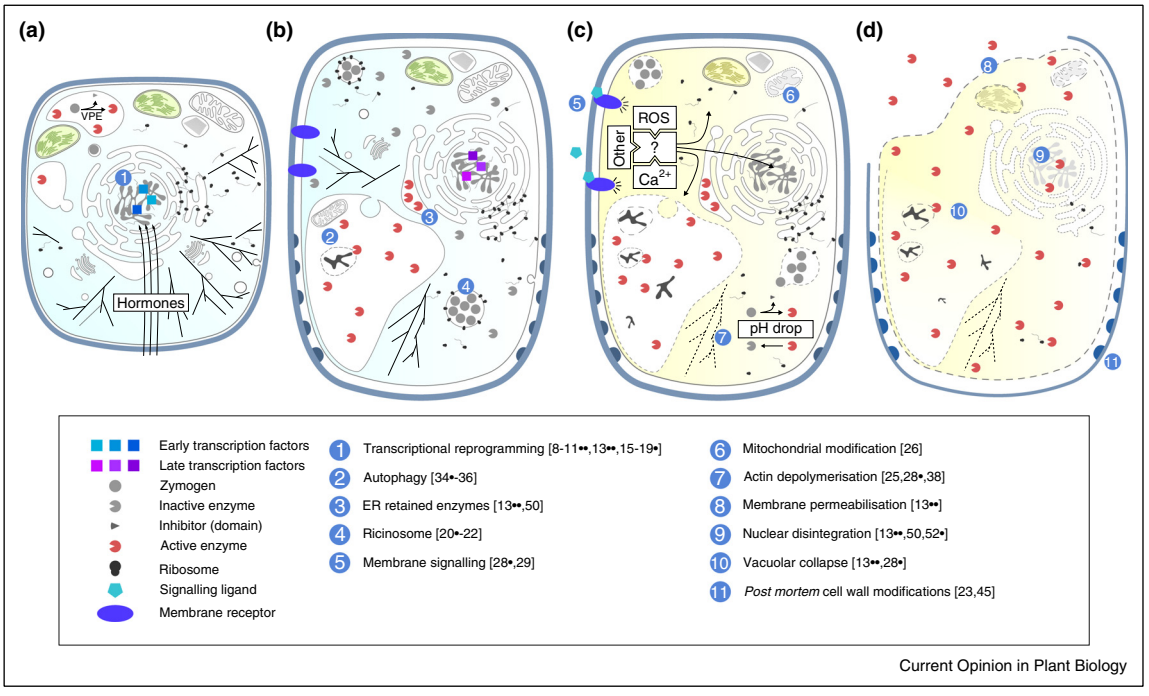
Phase I: preparation for PCD in the course of cellular differentiation

Some plant cell types undergo a terminal differentiation event in which the cell is killed and — partially or totally — removed through dPCD processes [3]. Plant hormones guide different aspects of plant development including cell differentiation and death. Notable examples include the complex crosstalk of ethylene, jasmonic acid, abscisic acid and gibberellic acid during seed development and germination, or the involvement of auxin, ethylene, cytokinin and brassinosteroids during xylogenesis (reviewed in [5,6]). Because hormones and hormonal crosstalk pleiotropically affect many aspects of plant development it is difficult to separate direct from indirect hormonal effects on cell death. One challenge of future research will be to determine the specific signalling

pathways that are employed by different hormones to control particular dPCD processes.

Downstream of hormone signalling a transcriptionally regulated differentiation programme is set in motion (Figure 1a) [7]. In *Arabidopsis*, NAC (NAM, ATAF and CUC) transcription factors (TFs) control the differentiation of xylem and lateral root cap (LRC) cells. While recessive loss-of-function mutations in *VASCULAR-RELATED NAC-DOMAIN6* (*VND6*) and *VND7* do not lead to obvious phenotypes, dominant-repressive mutants lead to a reduction in xylem formation [8]. Both VND6 and VND7 ectopically trigger tracheary element-like secondary cell wall (SCW) formation and cell death upon inducible systemic expression [8]. Among their target genes are genes that control different cellular processes such as SCW deposition and protoplast clearance during xylogenesis [9,10]. Interestingly, VND-homologous TFs (VNS) of the moss *Physcomitrella patens* control the formation of hydroids, water-conducting cells functionally analogous to xylem but devoid of tracheary SCW formation. While *vns* mutants display impaired autolysis during hydroid cell differentiation, VNS proteins are able to activate the same sets of downstream targets as VND6 and VND7 when expressed in *Arabidopsis* [11••]. These findings suggest an evolutionary conserved mechanism controlling xylogenesis and dPCD in plants, supporting the concept that SCW deposition is an add-on to an ancestral dPCD and autolysis programme to form effective water-transporting conduits [12]. Analogous to xylem NAC TFs, a root cap-specific NAC TF, SOMBRERO (SMB) promotes root cap differentiation and dPCD in *Arabidopsis*. SMB controls the expression of xylem and root cap expressed cell corpse clearance factors such as the nuclease BFN1, and of the death-associated aspartic protease PASPA3. Root cap cell death in *smb* loss-of-function mutants is delayed and aberrant [13••]. Similar to xylem and the root cap, the tapetum layer within the anther undergoes dPCD at the end of its differentiation programme. The precise timing of this process is crucial for pollen development: in *Arabidopsis* and rice, mutations in key TFs alter tapetum differentiation and dPCD resulting in male sterility (reviewed in [14]). The *Arabidopsis* TF MYB80 directly controls the transcription of the mitochondria-localised

Figure 1



Summary of the molecular mechanisms governing the different phases of differentiation-induced dPCD. **(a)** Undifferentiated cell: In a young cell a differentiation programme is imposed by developmental cues. The complex crosstalk of hormones trigger early transcriptional reprogramming by TFs. In the young cell proteases are present in the vacuole for steady-state cell catabolism. Several of these proteases require processing to be active. **(b)** Phase I: a different set of TFs activates the PCD module of the differentiation programme. During Phase I the differentiated cell starts to prepare for cell death by accumulating lytic enzymes (or their zymogens) in different (specialised) compartments. **(c)** Phase II: signal transduction by second messengers (Ca^{2+} and ROS) and other (unelucidated) signalling cascades triggers the initiation of cell death execution. In the dying cell the vital components of the cell are degraded through the action of autophagy and lytic enzymes that are released from various stores. A drop in cytosolic pH activates the released enzymes. **(d)** Phase III: tonoplast rupture and plasma membrane permeabilisation can be regarded as the moment of death. Lytic processes that have been initiated in Phase II are completed after cell death, so that the cell corpse is degraded by action of released enzymes. In some cell types all remaining debris is removed (upper half). In other cells (lower half) the cell wall remains and allows the cell corpse to carry out important functions.