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Chapter

Programmed Cell Death (PCD) in Plant: Molecular Mechanism, Regulation, and Cellular Dysfunction in Response to Development and Stress

Raju Mondal, Sreya Antony, Sovan Roy and Sanjib Kumar Chattopadhyay

Abstract

Programmed cell death (PCD) or apoptosis is a genetically programmed cellular process. Though in the plant, a true caspase system is lacking, still PCD can occur throughout the life cycle at any cell type, tissue, and organ part in response to a wide range of stimuli. Here we have discussed the current understanding of plant PCD in terms of different pathways, cellular dysfunction, regulation, and signaling mechanisms. Our present study discussed how and to what extent PCD is involved in pre-zygotic and post-zygotic plant life cycle and emphasized to what extent PCD modulated in response to abiotic and biotic stress. Additionally, the expression profile of different PCD-associated genes that are modulated by developmental stage, biotic-abiotic stress, cellular metabolites are also elucidated. Hence, this study will be helpful for understanding the molecular and structural instincts of PCD in different stages of plant growth and development, response to biotic/abiotic stimuli, and cellular dysfunction.

Keywords: Plant Development, PCD, Signaling, Stress, Caspase-like, metacaspase-like proteases

1. Introduction

Plant cells can cope up with fluctuating environments by modulation of different cellular processes [1]. Programmed cell death (PCD) is the process in which the cell(s) voluntarily commit suicide on several occasions among which the most prominent reasons are morphogenic or developmental changes and stress responses. In plants and animals, PCD plays significant role during development, structure formation, or removing unwanted tissues [2, 3]. It has been also reported that PCD act as a defense mechanism of an organism to prevent the pathogen spread from the infected cell [4].

In general, PCD is grouped into three broad categories; *viz.* apoptosis, autophagy (vacuolar PCD), and necrosis [5]. The detailed cellular mechanism of apoptosis is

well studied in the animal system but current knowledge of the execution processes leading to PCD in plants is very scarce [6, 7]. In general, the fundamental mechanism of apoptosis involves chromatin condensation followed by nuclear fragmentation and the formation of the apoptotic body [8]. Besides that, autophagy involves the formation of vacuoles and rupture of vacuoles that results from the release of hydrolyses enzyme and sudden disappearance of cytoplasm [9]. Such autophagic vacuoles contain degenerating organelles and cytosolic content [10]. Lastly, the process of necrosis is an energy-independent process that results in cytoplasmic swelling and rupture of the plasma membrane [5].

Our present study emphasized on current understanding of plant PCD in different dimensions such as understanding the PCD response towards stress, types of PCDs occurs, cellular-hormones signaling mechanism, and proteases involved in different stress conditions and developmental processes. Additionally, our comparative studies showed that the process of true apoptosis is absent in plants but has several similarities, hence this process is termed apoptosis-like PCD (AL-PCD). The *cys*-protease which is responsible for triggering animal PCD is absent in plants but similar kinds of proteases particularly vacuolar processing enzymes (VPEs) and papain-like cysteine proteases (PLCPs), known as metacaspase play the main roles in PCD [6, 11, 12].

2. Central role of developmental PCD (dPCD) in plant

Inductions of morphogenetic changes are an integral part of the development of an organism. PCD is one of the major fundamental cellular processes that plays crucial role in morphogenetic changes in plant systems [13]. In plants, the occurrence of cell death during development is termed developmental PCD (dPCD). PCD can occur throughout the life cycle at any cell type, tissue, and organ part of the plant (**Table 1**).

Plant Part	Mechanism	Reference
Pollen	ROS mediate	[14]
	Osatg7–1 mediate	[15]
	ROS mediate	[16]
	PERSISTENT TAPETAL CELL2 mediate	[17]
	AtLSD1 deathosome mediate	[18]
Female floral buds	*AMC9,* MeGI, BAG5, AifA, and HSP70 mediate	[19]
Female gametophyte	Auxin efflux	[20]
Fruit	Ca^{2+}-dependent nuclease	[21]
Double Fertilization	ROS mediate	[22]
Vegetative cells	*PrpS-PrsS* modulate PCD vegetative cells	[23]
Zygotic Embryogenesis	—	[24]
Somatic Embryogenesis	K^{+}_{ATP} channel activity	[25]
	Phenol-storing cells	[26]
Embryonic Suspensor Cell	—	[27]

Table 1.
List of important plant parts/sites of PCD in a vascular plant suggested developmental PCD (dPCD) involved cellular differentiation of specific cell types.

Among the two other types of cell death, apoptosis is the most understood type of PCD [5]. In plants, the process of true apoptosis is absent but has a similar kind of programmed cell death termed apoptotic like programmed cell death (AL-PCD). AL-PCD is morphologically and biochemically similar to apoptosis, but due to the structural and functional differences of plant cells, there are some changes in the execution process. The general similarities between apoptosis and AL-PCD include (1) cell shrinkage (2) chromatin condensation, (3) mitochondrial permeabilization and depolarization, (4) cytochrome c release, (5) vacuole leakage and fusion with plasmalemma. In the case of mitochondrial permeabilization, the release of cytochrome *c* leads to the formation of a caspase-like protein termed metacaspase. This in turn results in the release of nuclease and several protease enzymes which finally leads to DNA fragmentation and protein degradation [28]. The fundamental phenomenon of PCD in plants occurred across the whole life cycle depicted in **Figure 1**.

Developmental PCD (dPCD) is triggered by vascular cell death [30]. Vascular cell death is mainly associated with developmental stages includes morphogenesis and senescence. The existence of vascular system in the plant cell is the key difference from an animal cell. Vacuolar cell death in plants resembles autophagy in animals. A large portion of the plant cell is occupied by vacuoles and the presence of these lytic vacuoles plays a major role in plant cell death. This type of cell death is known as vacuolar cell death [30]. The mechanism involved in this vacuolar cell death involves

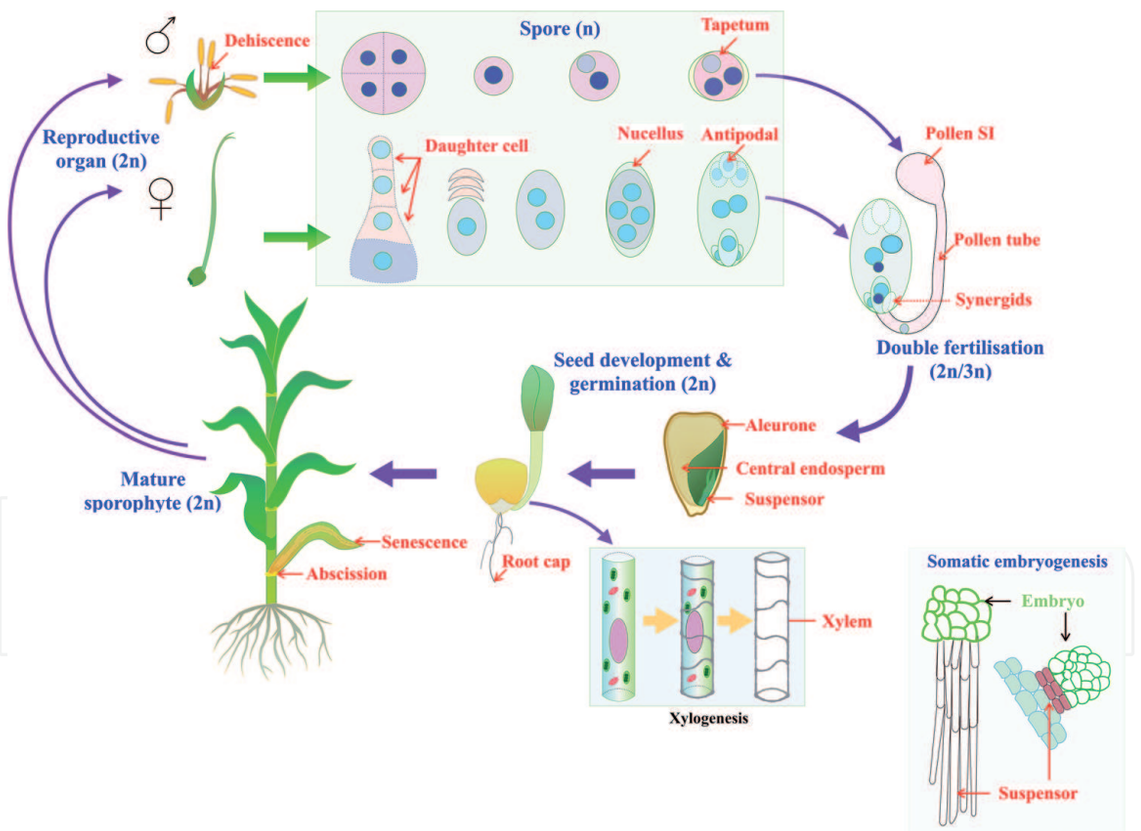


Figure 1.
Central role of PCD across the plant life cycle. Pre-zygotic and post-zygotic developmental changes such as tapetum layer degeneration, daughter cell degeneration, antipodal-synergids cell degeneration, pollen tube degeneration, aleurone layer, central endosperm, suspensor cell death are controlled by PCD. The process of xylogenesis is also influenced by PCD [29]. During the differentiation of tracheary elements, vacuole swelling and rupture are coordinated with the thickening and restructuring of the cell wall. The final collapse of the vacuole immediately precedes nuclear DNA fragmentation, which occurs at the late stages of the cell-death process before the final autolysis of the cell. In mature sporophyte senescence, abscission and dehiscence are regulated by PCD. PCD is also observed during in vitro *somatic embryogenesis [25]. Symplastic cell-to-cell trafficking connection between the somatic embryo and mother explant is broken due to PCD. Degeneration of suspensor cell of somatic embryo mediated by PCD.*

the increase in the size of the vacuoles by fusing small vacuoles and results in the formation of larger vacuoles. Autolytic PCD occurs due to tonoplast rupture and the clearance of cytoplasm. It is not clear the exact reason for the rupture of tonoplast but the clearance of cytoplasm is due to the flow of hydrolases enzyme that degrades the cytoplasm [31, 32] which is released to aid differentiation of tracheary elements [30, 33]. The dPCD involved to develop various organs like integuments [34], megasoprogenesis [35], pollen tube development [36], leaf senescence [37].

Plant cells contain two types of vacuoles in different types of tissues, lytic vacuole (LV) and protein storage vacuole (PSV) [38]. PSV may contain many kinds of proteins especially defense and stress-related, and the pH value found close to neutral [39–41]. Unlike the PSV, LV helps in ion and water homeostasis of the cell [42]. It is reported that PSV of cereal aleurone transformed from storage compartments to lytic organelles and fusion of several PSV, acidification takes place in the vacuolar lumen [43].

Studies also suggest that a particular cysteine protease called the 'vacuolar processing enzyme' (VPE) functions as a key regulator or the executioner of plant vacuolar PCD during development and also during stress [44]. Located in the vacuole, VPE ruptures the lytic vacuolar membrane. VPE is a cysteine protease and involved in cleaves the peptide bond at the C-terminal side of asparagine and aspartic acid [45]. The up-regulation of VPE genes was associated with various types of cell death under stressed conditions. This is essential in processing seed storage proteins and for mediating the susceptible response of toxin-induced cell death [46]. The collapse of the vacuolar membrane allows the release of hydrolytic content and causes the destruction of other organelles [47]. For example degradation of nuclear and chloroplast DNA, without condensation of DNA, can be completed immediately within 20 minutes of vacuole rupture, but chlorophyll degradation takes more time. Even the plasma membrane can be completely degraded as observed in endosperm tissue. However lignified tissues are exempted from this degradation. Furthermore, PCD in tracheary cells and fiber cells is delayed so that more lignification can take place to allow those cells to become more rigid for structural support [47].

Arabidopsis genome codes for at least 4 VPE homologous αVPE, βVPE, γVPE, δVPE. All of which are located in the vacuole [48]. The last one (δVPE) is only found in dicots [49]. Among these βVPE is specifically located to seeds, whereas αVPE and γVPE are specific to vegetative organs [50] and the δVPE is expressed in seed coat development at the early stage of seed formation [51]. However, the number of VPE genes in various plant species differs, for example four genes have been described in Arabidopsis [52], eight in the case of barley [53], 5 in rice [54], and 14 in tomato [55]. In the recent advancement of the genomic and transcriptomic data, the activities of VPE genes along with their expression pattern will become clearer. In an experiment, it has been proved that when all 4 VPE genes are mutated in Arabidopsis (VPE null mutant) and detectable activities of caspase-1 or VPE in the fungal toxin fumonisinB1 (FB1)-treated leaves whereas wild-type leaves had the caspase-1 and VPE activities [46]. It demonstrated that in planta VPE is solely responsible for caspase-1 activity [52].

3. Types of cellular dysfunction and molecular mechanism lead to PCD in plant

In plants, there are mainly three types of PCD that have been reported *viz.* (1) Apoptotic-like cell death (AL-PCD), (2) senescence-associated death, and (3) vacuole-mediated cell death which resembles autophagy [56]. The cellular dysfunction of above mentioned process has been illustrated in **Figure 2**.

Although presence of rigid cell wall associated with cell membrane prevents to form apoptotic body and lack of true caspase as well as phagocytic cells are main reason for absence of true apoptosis in plants but plants exhibit another mechanism that shows striking similarity to apoptosis which is known as apoptotic-like cell death (AL-PCD) [57, 58]. Like animals, when a plant cell is subjected to PCD several changes occur for example cell shrinkage, condensation of chromatin, chromosome fragmentation, mitochondrial permeabilization, cytochrome C release, etc. **Figure 2A** represents these events schematically [59, 60]. Chromatin condensation and chromosome fragmentation are two characteristic features that are also observed during necrotic and autophagic mode cell death.

Hypersensitive reaction (HR) also results of protoplast shrinkage, similar to apoptotic cell shrinkage. But in the case of animal apoptosis, there is a distinct morphology by which apoptosis can be recognized, the plasma membrane retains its integrity, while the cell shrinks. Animal cells form apoptotic bodies -a vesicle containing segments of a dying cell and apoptotic bodies are formed during the execution phase of the apoptotic process, where the cell's cytoskeleton collapse and causes the membrane to bulge outward surrounding cells which might cause damage (like an inflammatory response) to them. However, unlike animal cells, in plant

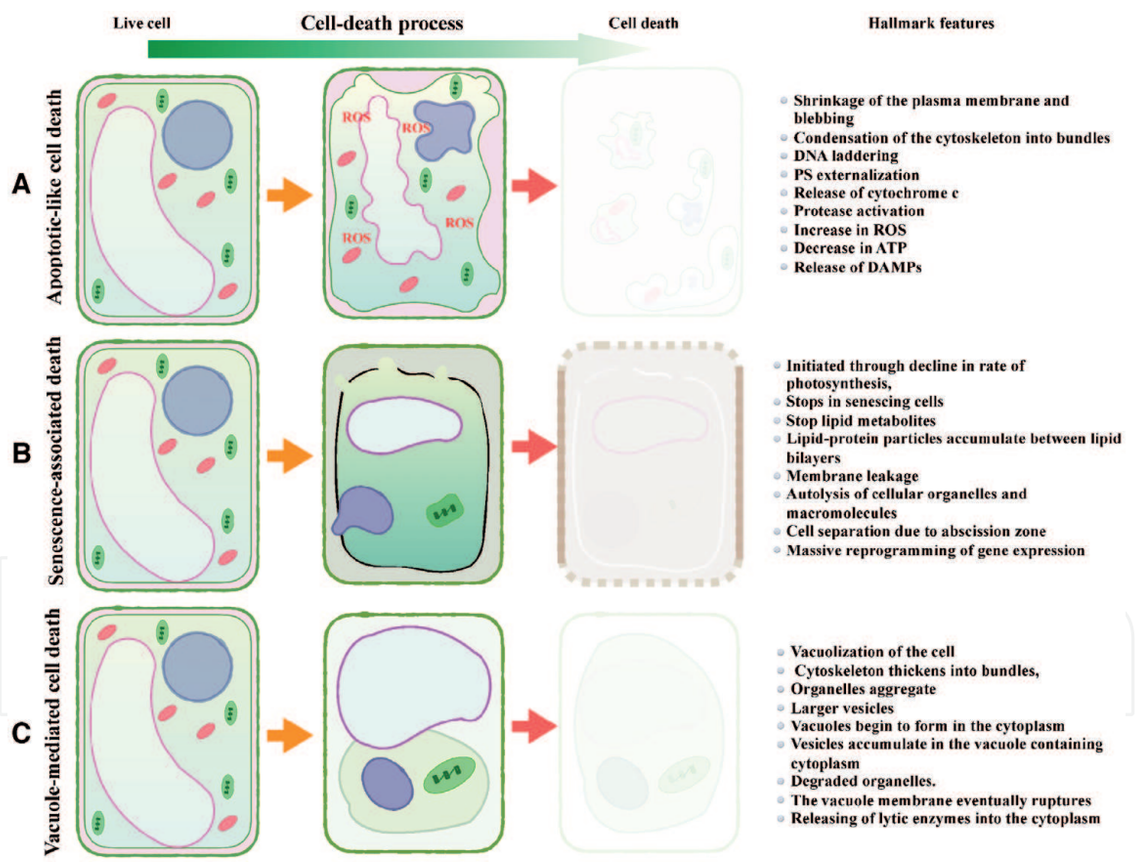


Figure 2.
Morphological comparison, the hallmark features of types of cellular dysfunction in response to PCD. (A) Apoptosis-like PCD mediated by shrinkage of the plasma membrane move away from the cell wall, membrane blebbing, condensation of the cytoskeleton into bundles, DNA laddering, PS externalization, cytochrome c release, protease activation, increase in ROS, decrease in ATP, the release of DAMPs. (B) Senescence-associated death initiated through the decline in the rate of photosynthesis, stops in senescing cells, stops lipid metabolites, lipid-protein particles accumulate between lipid bilayers, causing the membrane to become leaky. In the degenerative phase, autolysis of cellular organelles and macromolecules takes place. And in the terminal phase cell separation takes place at the abscission accumulation by massive reprogramming of gene expression. (C) Vacuole-mediated cell death or autophagic PCD can be characterized by vacuolization of the cell on a large scale. The cytoskeleton thickens into bundles, organelles aggregate, and larger vesicles and vacuoles begin to form in the cytoplasm. Vesicles accumulate in the vacuole containing cytoplasm and degraded organelles. The vacuole membrane eventually ruptures, releasing lytic enzymes into the cytoplasm and furthering cell death.

cells, the content of dead cells remains in the cell itself and there is no membrane blabbing and the process of phagocytosis is also absent [57, 61]. In comparison to animal PCD the true detailed mechanism of AL-PCD is unclear [45]. Plant cells do not undergo 'classic' form of apoptosis because of their rigid cell walls that rule out the necessity or possibility of a breakdown of plant cells into apoptotic bodies and also there are no phagocytic cells in plants [57].

Activation of PCD triggering proteases occurs may be due to a result of a certain change in the cellular environment. It is reported that the activation and dimerization of cysteine C13 protease legumain occur during the low pH. The evidence supporting this includes wheat homolog triticain-α is activated in low pH [62]. In *N. tabacum* reactive carbonyl species (RCS), a ROS product, increased the activity of caspase-like proteases (C1LP and C3LP). This is similar to animal cell where ROS trigger PCD by activating specific proteases [63].

In response to heat stress in cucumber cotyledons, releases of cytochrome c from mitochondria indicate that cyt-c functions differently in plants to initiate PCD in the absence of Bcl-2 proteins [64–66]. The presence of all the cyt-c is

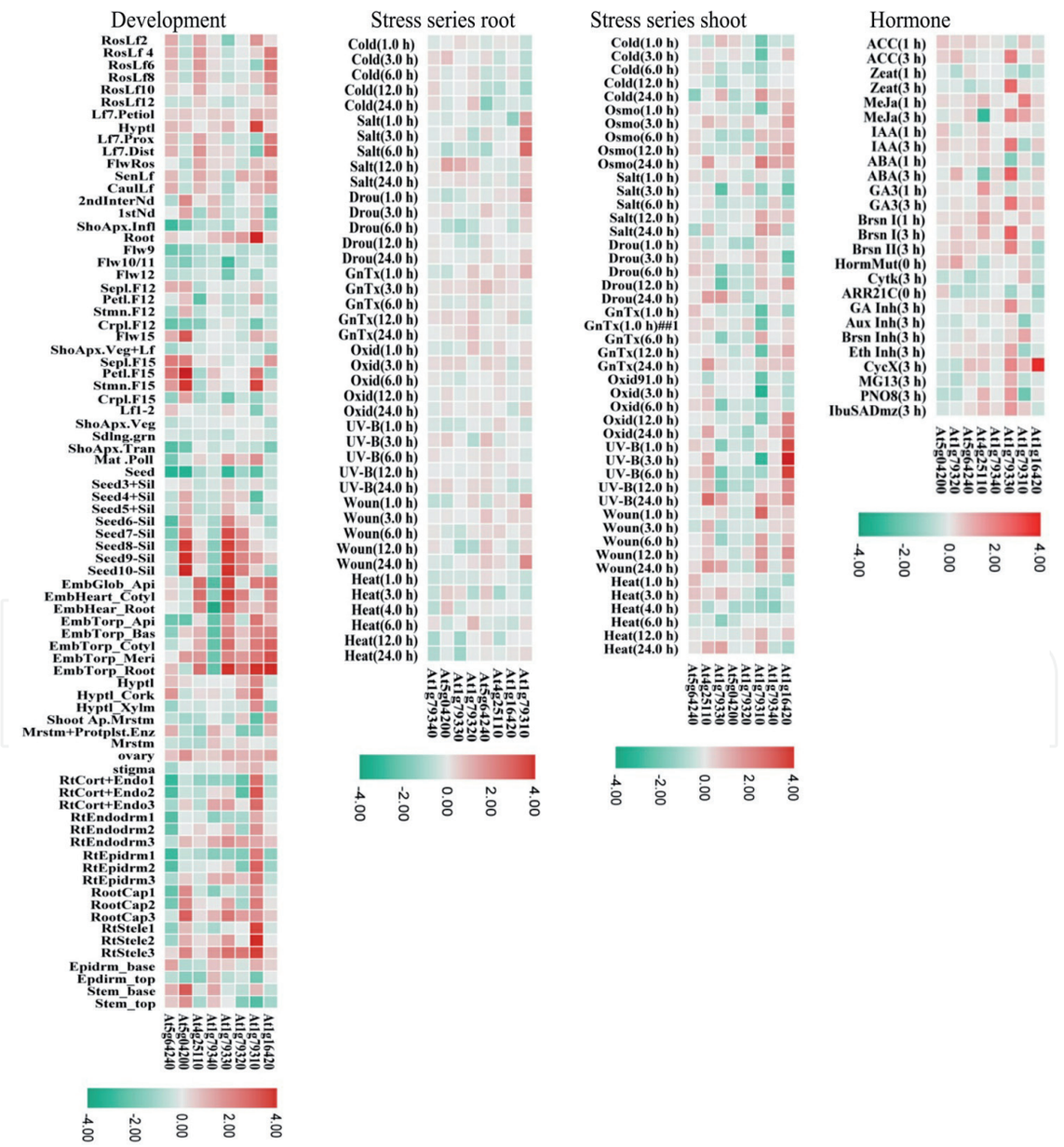


Figure 3.
Differential expression pattern of caspase genes in response to developmental stage, abiotic stress, and metabolic stimuli like plant growth regulators.

detected in the cytoplasm after 1 hour of heat stress although it is completely absent in mitochondria after 3 hours of the heat stress. Self-incompatibility (SI) induction in incompatible pollen tubes is also stimulated by the cytochrome c relocation from mitochondria to cytosol [67]. Like animals, cytochrome c is released in response to stress in plants, but studies suggest that it is functionally different as in the case of apoptosis [60, 64]. Caspase-driven cell death is the process that only present in animal kingdom, but the plant genome lacks core apoptotic proteins like BCL-2 family and caspase [68]. Two caspase-like protein families have been recognized, *viz*. (I) paracaspases, (II) metacaspases [69]. Some metacaspase prodomain comprises a zinc finger motif that resembles the plant hypersensitive response (HR) protein Isd-1 [69]. Metacaspases are members of the C14 class of cysteine proteases and thus related to caspase, orthocaspase, and paracaspase. The metacaspase is recognized as Type I and Type II, both are arginine/lysine-specific, in contrast to caspase, which is aspartate-specific [70]. Differential expression patterns of caspase genes are observed in different developmental stages, abiotic stress, and metabolic stimuli like plant growth regulators (**Figure 3**). Thus, we can assume that plant PCD is a most complex events with coordinated regulation.

Unlike animal cells, the formation of the apoptotic body is absent in plant cells but in response to the biotic and abiotic stress, the apoptotic body-like structures are also observed in the plant cell [71]. According to the most recent proposed model by Thanthrige *et al*. [72], plant PCD is controlled by conserved protein family B cell lymphoma 2 (Bcl-2) associated athanogene (BAG). Subcellular localization and probable function of different BAGs are presented in **Figure 4**.

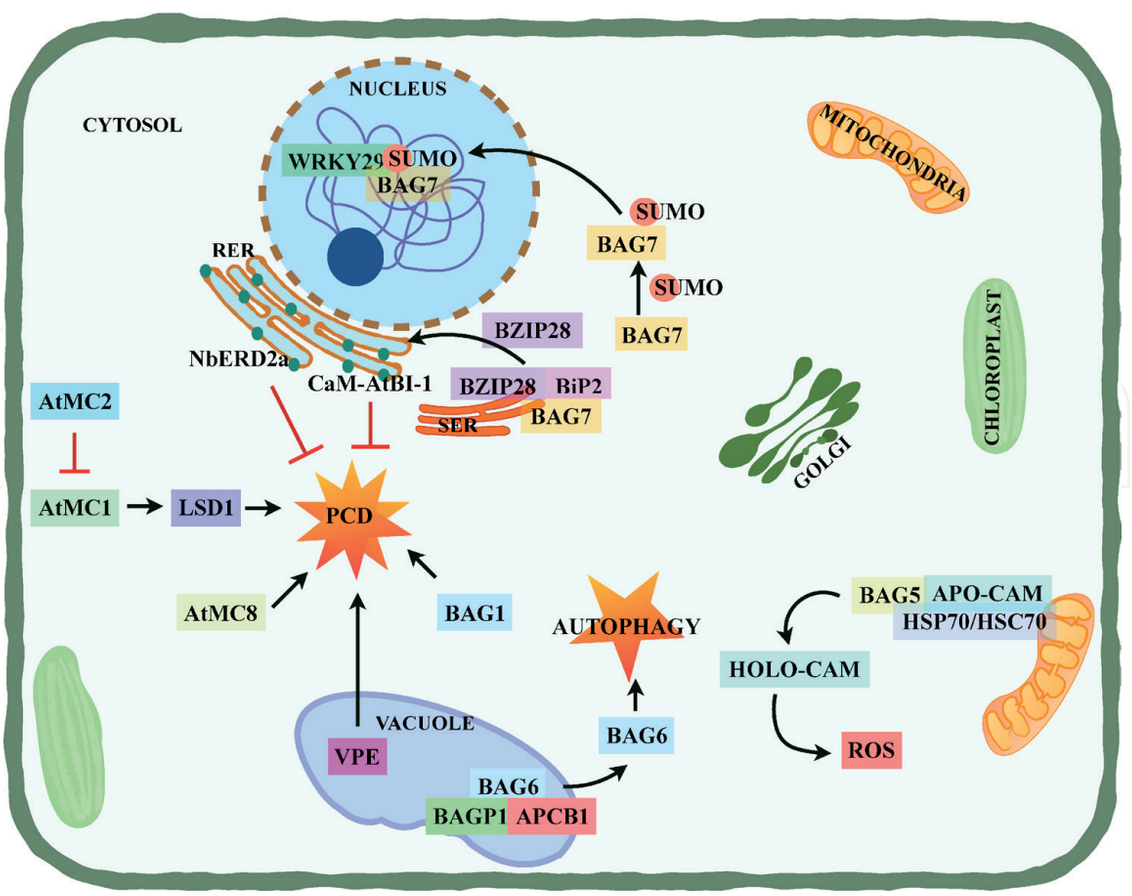


Figure 4.
Pictorial presentation of molecular signature of plant PCD. Seven Arabidopsis BAG members (BAG 1–7) were localized throughout the cell in organelles. AtBAG1–4 (cytoplasm) AtBAG5 (mitochondrion), AtBAG6 (Vacuole, nucleus) and AtBAG7 (ER, nucleus) mechanized PCD in plant system.

In *A. thaliana* there are seven BAG genes identified and they are localized in the cytoplasm, vacuole, nucleus, endoplasmic reticulum (ER), mitochondria [72]. Cytosolic protein AtBAG1, AtBAG2, and AtBAG3 interact at the C-terminus of the HSC70-interacting protein (CHIP). AtBAG1 and AtBAG2 involved proteasomal degradation and plant development process respectively. Though the function of AtBAG3 remains unknown. Cytosolic AtBAG4 interacts with heat-shock protein 70 (HSP70) to repress cell death in response to abiotic stress. AtBAG5 is mitochondrion-localized and interacts with HSP70 and calmodulin (CAM). At a low concentration of cellular Ca^{2+}, the AtBAG5-CAM-HSP70 complex produces reactive oxygen species (ROS) and fasten leaf senescence, but in presence of high cellular Ca^{2+} concentration senescence is inhibited. Vacuole and nucleus localized AtBAG6 play an important role in autophagy. AtBAG6 is bind with AG-associated GRAM protein 1 (BAGP1) and adenomatous polyposis coli B1 (APCB1) are involved in basal defense mechanism against necrotrophic fungi. ER and nucleus localized protein AtBAG7 accelerate heat and cold tolerance by interacting with small ubiquitin-like modifier (SUMO) and WRKY, a DNA-binding protein/transcription factor 29 (WRKY29). Moreover, the BAG co-chaperone family played a potential role in response to a wide range of stress stimulation during plant PCD. Though the future systematic investigation is required to enrich understanding of BAGs function that may help to develop improved stress-tolerant crops.

4. Plant PCD in response to biotic and abiotic stress

Plant PCD occurs mainly during the time of plant-pathogen interaction as well as in response to different abiotic stresses [73]. There are several indentified major causal biotic and abiotic factors that induce plant PCD are presented in **Figure 5**.

Plant-pathogen interaction mediated hypersensitivity response (HR) is a mode of broad-spectrum resistance in plants. Whether the pathogen is compatible or incompatible is determined by the interaction between resistance genes in the host and the avirulence gene in the pathogen as explained in Flor's hypothesis [74]. Hypersensitive cell death is the localized rapid death of cells at the point of infection, and it serves not only to restrict the growth but also stop the invasion of pathogens to other cells/tissue of the plant by undergoing suicide of the infected cells. The complex signalling of this hypersensitive reactions involves cascade of events such as protein phosphorylation, reactive oxygen species (ROS) production, and modification of ion fluxes [75].

HR is known as a defense mechanism against biotrophic plant pathogens or microbes, which depend on nutrients for survival on the host. However, it is observed that HR might be beneficial in the early stages for local adaptation against the infection but not in the later stages [76]. HR is observed in most plant species and it can be influenced by a wide range of plant pathogens such as oomycetes, viruses, fungi, and even insects [77]. All HR-induced PCD have one feature in common which distinguishes it from vacuolar-type, that is, this type of PCD is not initiated by vacuolar swelling and release of lytic enzymes inside plant cell (i.e. non-destructive). However, this type of response may include the release of vacuolar content by fusion with the plasma membrane [78]. Even it has been proved that VPE is essential for a virus-induced HR that involves PCD [11]. Thus it leaves no doubt that vacuolar rupture might take place during HR. Besides that, necrotic cell death is mainly triggered by abiotic stress in plants. The cell death via AL-PCD or necrosis is dependent upon the severity of the stress that the higher heat stress

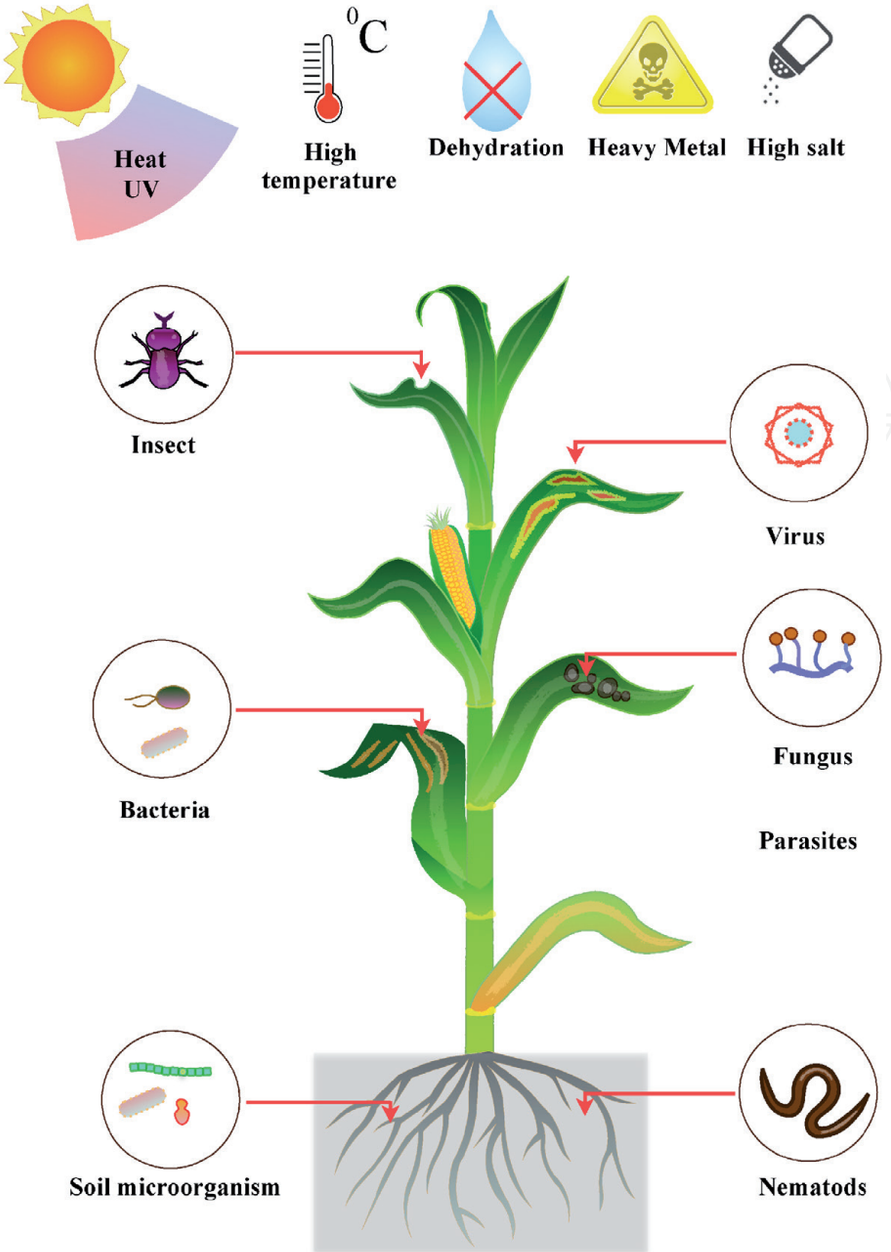


Figure 5.
Diagrammatic presentation of various biotic and abiotic factor-induced PCD in the plant.

leads to necrosis, while moderate heat stress leads to AL-PCD [57]. Previous study also suggests some difference between AL-PCD and necrosis. These include (1) shrinking of protoplast is absent in necrosis while in AL-PCD protoplast shrinks from the cell wall; (2) unlike apoptosis, necrosis is an energy-independent process which proceeds through sudden permeabilization of the plasma membrane and plasma membrane leakage. Additionally, a array of researcher revealed that PCD can induced by a range of abiotic stress tolerance. For more details description see **Table 2**.

5. Metabolic regulation of plant PCD

Hormones play an important role in plant PCD. Ethylene, methyl jasmonate, and salicylate have been identified as key regulators which involved in the

Stress	Studied plants	Cellular responses	Reference
Heat shock	Carrot Arabidopsis Tobacco Soybean	Cytoplasmic condensation	[79–83]
H_2O_2	Arabidopsis	AL-PCD	[79]
Toxin victorin	Oats	Cell shrinkage associated with death	[84]
TMV	Tobacco	DNA Cleavage into 50-kb fragments	[85]
Fungal infection	Cowpea	Internucleosomal cleavage	[86]
D-mannose treatment	Arabidopsis root Maize suspension culture	Oligonucleosomal fragmentation of DNA	[87]
Ultraviolet light or H_2O_2	Metacaspase-8 knockout lines of Arabidopsis	Reduced cell death	[88]
Mycotoxin	Arabidopsis	Detected the presence of Vascular processing enzyme	[52]

Table 2.
List of studies depicted the environmental stress stimulate PCD in plants.

different developmental process like senescence [89]. The increased production of ethylene and salicylic acid associated with ROS-dependent PCD has been investigated [90]. In response to PCD, identified up-regulated genes and associated metabolism processes are autophagy transport, response to ROS, ABA signaling, metal-ion binding, DNA/Protein binding, carotene metabolism, glutamine synthase 2, caspase activity, pectinesterase activity, ethylene signaling, and lipid catabolism. Simultaneously down-regulated genes and associated metabolism processes amino acid metabolism, chlorophyll biosynthesis, carotenoid biosynthesis cytokinin-mediated signaling, glycine metabolism,

PGRs/Metabolites	Identified pathways/mechanism	Plant species	Reference
Auxin	Auxin may restrict the cellulose biosynthesis inhibitor such as Thaxtomin A (TA) and protect PCD.	*Arabidopsis thaliana*	[93]
	Modulate ROS accumulation, anthocyanin production, and the release of mitochondrial cytochrome *c* pathways by auxin antagonist auxinole, auxin inhibitor NPA.	*Aponogeton madagascariensis*	[94]
Nitric oxide	Pathogenesis-related 1 (PR-1) and phenylalanine ammonia-lyase genes and cyclic GMP (cGMP) and cyclic ADP-ribose (cADPR), found to be involved in PCD signaling pathways in abiotic stress.	—	[95]
ROS	*LASTIDIAL NAD-DEPENDENT MALATE DEHYDROGENASE* (*plNAD-MDH*), chloroplastic *DICARBOXYLATE TRANSPORTER 1* (*DiT1*) and *MITOCHONDRIAL MALATE DEHYDROGENASE 1* (*mMDH1*) involved in rescues ROS accumulation and mutant of MOSAIC DEATH 1 (MOD1), leads to the accumulation of ROS and PCD.	*Arabidopsis thaliana*	[96]

PGRs/Metabolites	Identified pathways/mechanism	Plant species	Reference
Salicylic acid (SA)	SA-regulated alternative oxidase (AOX) plays a crucial role in the reduction of mitochondrial ROS and cell death mechanisms.	—	[97]
Ethylene	Ethylene-responsive element binding factors 2 (ERF2) in *Petunia* involved in PCD	*Petunia* sp.	[98]
	Ethylene-mediated ROS signaling plays a role in aerenchyma formation.	*H. annuus*	[99]
Myo-inositol (MI)	MI modulate ROS-induced PCD through SA-dependent and ethylene-dependent pathways.	*Arabidopsis thaliana*	[100]
Phenolic compound	Pyrogallic acid induce PCD in cyanobacteria (*M. aeruginosa) in* caspase-3 (–like)-dependent manner.	*Microcystis aeruginosa*	[101]
Rosmarinic acid	Rosmarinic acid modulates ROS and mitochondrial dysfunction	*Arabidopsis thaliana*	[102]

Table 3.
List of plant growth regulators (PGRs), other metabolites, and their role in plant PCD.

photosynthesis, glutamine synthase 1 [91]. In response to PCD expression profile of ROS-scavenging enzymes such as peroxidase, ascorbate peroxidase (APX), superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPX)) and non-enzymatic antioxidants ascorbic acid (AsA) and glutathione (GSH) also be studied in *Triticum aestivum* [12]. Yang *et al*, [92] investigated that Acyl-CoA Synthetase (ACOS) is one of the enzymes involved in fatty acids metabolic and protect tapetum cells from PCD and maintain reproductive fitness in rice. Some important findings refers that PGRs, different metabolites are played key role in PCD in plant (**Table 3**). Thus the complex metabolic network is involved in PCD in the plant system. The further systematic metabolic study will be required to understand and enrich our current understanding.

6. Conclusion

In the present study, we have discussed the recent status of occurrence, cellular dysfunction and molecular mechanism of the most complicated fundamental processes of PCD in the plant. Due to the lack of sufficient study on PCD in plant systems, we are unable to identify and describe the exact molecular mechanism. The complex process of PCD is triggered in normal development as well as in response to stress. To understand (1) how plant cell sense or become competent towards PCD? (2) what are the specific signaling pathways correspond to different types of plant PCD?, integrated omic study is helpful in the near future. In our recent study, we have identified novel Cell division cycle and apoptosis regulator 1 (CCAR1) protein in plant system and computationally characterized their function in plant PCD [103]. Thus spectaculars study will be required for understanding the PCD in plant system.

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