Env.Biodiv. Soil Security **Vol. 8**, pp: 165- 173 (2024)

Environment, Biodiversity & Soil Security
http://jenvbs.journals.ekb.eg/

Decomposition of Dead Cells in Plants and Their Role in Self-Organic Nutrition

Ayman M. El-Ghamry[1]; Ahmed A. Mosa[1], Hassan R. El-Ramady[2], Dina A. Ghazi[1], Mohamed A. El-Sherpiny[3*] and Zein El-Abedin A. Mohamed

[1] *Soil Sciences Department, Faculty of Agriculture, Mansoura University, 35516, Egypt*
[2] [3] *Soil and Water Dept., Faculty of Agriculture, Kafrelsheikh University, Kafr El-Sheikh 33516, Egypt*
[4] *Soil & Water and Environment Research Institute, Agriculture Research Center, Giza, 12619, Egypt*
Agric. Botany Department, Faculty of Agriculture, Mansoura University, 35516, Egypt.

THE role of dead cells in plants is a critical area of study, as these cells facilitate the breakdown and absorption of nutrients within plant tissues. This highlights the occurrence of self-organic nutrition during various stages of plant growth, with the process differing based on the plant's age and type. Programmed cell death (PCD) is a fundamental biological mechanism essential for the development, homeostasis, and stress response in all organisms. It is a highly regulated and complex process, and its misregulation can have detrimental effects. While significant progress has been made in understanding cell growth and proliferation, the contribution of PCD to cellular homeostasis in plants has only recently become a focus of research, uncovering considerable knowledge gaps. This review explores the concept of PCD, contrasting developmental PCD (dPCD) with environmental PCD (ePCD) in plant cells. It also emphasizes the importance of recycling processes associated with PCD, both in developmental stages and as a response to environmental stressors.

Keywords: Programmed cell death, PCD; Necrosis; dPCD; ePCD and Nutrients recycling

1. Introduction

Programmed cell death (PCD) refers to a self-initiated and carefully controlled process in which a cell orchestrates its own demise. This phenomenon is tightly regulated at both genetic and biochemical levels, indicating that cells possess the necessary molecular tools to execute their destruction. The evolutionary origins of PCD date back to ancient times, as several genes and molecules associated with this process are conserved across diverse organisms from different taxa. According to Lockshin and Zakeri (2004), PCD is characterized as a series of organized events culminating in the disintegration of the cell.

The fundamental morphological and biochemical characteristics of programmed cell death (PCD) appear to be conserved across both plants and animals. However, recent research highlights the role of organelles like the vacuole and chloroplast in regulating cell death in plants, suggesting that plants have developed their unique mechanisms for PCD. This process in plants relies on a complex network of signaling pathways, involving various molecular signals such as plant hormones, calcium, cyclic nucleotides, reactive oxygen species (ROS), and reactive nitrogen species (RNS) (Ma et al., 2009; de Pinto et al., 2012; Huysmans et al., 2016). Additionally, the execution of PCD requires interaction between different subcellular structures, including mitochondria, chloroplasts, vacuoles, the endoplasmic reticulum, and the nucleus. These interactions may involve anterograde and retrograde signaling pathways,

*Corresponding author e-mail: melsherpiny2010@gmail.com.
Received: 29/11/2024; Accepted: 21/12/2024
DOI: 10.21608/jenvbs.2024.340327.1258

although many of these signals remain to be fully identified (Ng et al., 2014; Van Aken and Pogson, 2017).

Cell death can be classified into distinct types, including apoptosis, autophagy, and necrosis, based on their morphological characteristics. Apoptosis, first described by Kerr et al. (1972), is defined by specific structural changes such as cell shrinkage, condensation and fragmentation of the nucleus, and ultimately the formation of apoptotic bodies. Unlike apoptosis, necrosis is an uncontrolled and traumatic process that typically occurs when cells are subjected to extreme physiological conditions beyond their tolerance. Key attributes of necrosis include disruption of membrane integrity, imbalance of ionic homeostasis, random DNA degradation, and loss of subcellular organelle compartmentalization, organelle swelling, and eventual rupture of the cell or organelle (Proskuryakov et al., 2003).

A key distinction between apoptosis and necrosis, particularly in mammals, lies in the inflammatory response and the release of toxic cellular debris associated with necrosis. Interestingly, the same agents that induce apoptosis can also trigger necrosis, with the outcome in cell cultures often determined by the severity of the stress applied (Mammone et al., 2000). While programmed cell death (PCD) is orchestrated by regulated intracellular signals, necrosis is a spontaneous and unregulated form of cell death. Rapid and precise techniques, such as the TUNEL assay, comet assay, and the measurement of caspase-like enzyme activity, are instrumental in distinguishing PCD from necrosis (Sychta et al., 2021).

2. What is programmed cell death?

Programmed cell death (PCD) is a deliberate process where cells intentionally undergo self-destruction. This mechanism often occurs during developmental or morphogenic changes and as a response to various stress factors. It entails a well-orchestrated series of events resulting in the systematic breakdown of the cell (Lockshin and Zakeri, 2004). The primary goal of PCD is to eliminate damaged or unnecessary cells while supporting evolutionary adaptations and enhancing organismal fitness.

3. Contexts of PCD in plant cells

Since plant PCD that fully mirrors the hallmarks of apoptosis has not been identified, the term "apoptotic-like cell death" (ALPCD) was introduced to describe instances of plant PCD exhibiting some features similar to animal apoptosis (Reape and McCabe, 2008). The nature of PCD in plants varies depending on how it is triggered, making it challenging to classify plant PCD events solely based on morphological and molecular characteristics. As a result, many researchers now categorize PCD from a functional perspective into two types: developmentally regulated PCD (dPCD) and environmentally induced PCD (ePCD) (Huysmans et al., 2016). dPCD is integral to vegetative and reproductive development, while ePCD is associated with responses to environmental stresses. Internal factors regulate dPCD, whereas ePCD is driven by external stimuli. Traditionally, dPCD and ePCD have been studied separately, and comparative analyses have revealed only limited similarities in their transcriptional profiles (Olvera-Carrillo et al., 2015).

Programmed cell death (PCD) plays a vital role in various aspects of plant development. Examples of its involvement include the differentiation of tracheary elements, the formation of endosperm and aleurone cells in cereal grains, trichome development, differentiation of the female gametophyte, the transition from bisexual to unisexual flowers, and responses related to self-incompatibility (Bosch and Franklin-Tong, 2008). Additionally, PCD contributes to certain types of leaf morphogenesis, such as the fenestrated leaf patterns seen in lace plants (Gunawardena, 2008). Differentiation events driven by dPCD can be categorized depending on their developmental context (Beers, 1997). Differentiation-induced dPCD represents a terminal step in the specialization of certain cells, such as xylem, anther tapetum, or root cap cells (Escamez & Tuominen, 2014; Kumpf & Nowack, 2015). In other instances, certain cell types can activate dPCD facultatively through cell-to-cell signaling mechanisms during responses like self-incompatibility (Wilkins et al.,

2014) or based on positional cues during processes such as aerenchyma formation or leaf perforation (Gunawardena, 2008).

Environmental-induced programmed cell death (ePCD) is initiated by external stressors, including pathogen attacks and abiotic stresses such as heat, salinity, drought, and flooding. Through ePCD, plants sacrifice certain cells to ensure the survival of the entire organism in response to these stresses (Petrov et al., 2015) or biotic threats such as pathogens (Wu et al., 2014). In some cases, ePCD triggers a hypersensitive response (HR), a specialized form of cell death activated at the site of biotrophic pathogen attacks. HR serves as a defense mechanism by allowing plants to recognize specific pathogen effectors, thereby limiting pathogen access to plant metabolic resources at the site of invasion. This strategy prevents the spread of pathogens throughout the plant. Both abiotic and biotic stressors induce necrosis—classified as ePCD—by initiating oxidative stress through the excessive production of reactive oxygen species (ROS). These ROS, primarily generated by mitochondria, contribute to phytotoxic responses and modulate PCD pathways. The interaction between ROS, plant hormones such as ethylene, nitric oxide (NO), and calcium ions triggers complex signaling pathways that activate PCD mechanisms. Proteases with caspase-like activity execute PCD in plant cells in response to these signals (Sychta et al., 2021).

In certain instances of plant cell death, notable similarities to apoptosis are observed, although key differences also exist. As a result, it has been proposed that this distinct form of programmed cell death in plants be referred to as apoptotic-like PCD (ALPCD) (Danon et al., 2000). ALPCD is thought to be regulated by specific signaling molecules, gene expression patterns, and caspase inhibitors, distinguishing it from necrotic or autophagic cell death, which do not typically involve these regulatory factors.

4. Roles of PCD in plants

Similar to animals, cell death is as vital to plant biology as cell division, playing a fundamental role in development and defense mechanisms. Programmed cell death (PCD) is crucial for proper development in higher plants and for initiating robust defense responses against invading pathogens (Greenberg, 1996). PCD encompasses a wide range of plant processes, including reproductive development, senescence, morphogenetic patterns, adaptive responses to environmental stressors, and immune responses against pathogens. PCD is therefore critical for various aspects of plant life, influencing growth, development, and responses to unfavorable environmental conditions, with potential implications for crop productivity (Petrov et al., 2015). Furthermore, plants employ PCD in senescent tissues such as leaves, petals, and sepals to recycle nutrients prior to their removal. From a developmental perspective, selective cell elimination through PCD is vital during embryogenesis and seed germination to facilitate the emergence of new plants (Domínguez and Cejudo, 2014). Differentiation-induced dPCD represents the final differentiation step in certain plant cells, such as xylem, anther tapetum, or root cap cells (Escamez & Tuominen, 2014; Kumpf & Nowack, 2015).

5. PCD nutrients recycling and reallocation of resources

Understanding how nutrient recycling operates in plants could reveal new molecular strategies to enhance crop yields by optimizing nutrient use. As autotrophic, stationary organisms, plants cannot access external mineral sources for sustenance, necessitating efficient internal recycling to support reproduction or the development of new organs, particularly during resource-limited conditions. Plants achieve this by utilizing processes such as senescence and programmed cell death (PCD) to redistribute resources. Although cell death is often viewed as the elimination of individual cells, the remnants resulting from these processes can, in certain situations, contribute to maintaining tissue stability and homeostasis.

Senescence is essential for plant development and survival, playing a key role in nutrient recycling and redistribution throughout a plant's lifecycle. Leaf senescence, in particular, facilitates the transfer of nutrients from senescing leaf tissues to support the development of new plant structures or seasonal growth in perennial species.

The primary purpose of leaf senescence is to break down and recycle cellular components

accumulated during the leaf's growth and maturation into reusable, transportable nutrients. These nutrients are then redirected to sink organs, optimizing nutrient use. This efficient recycling process enhances nutrient management, supports the formation of new plant organs, and promotes overall plant health and productivity.

The autophagy pathway, which operates through a vesicular process, has recently been implicated in chloroplast degradation and nutrient reallocation during both developmental and stress-induced senescence. Autophagy is a highly conserved vesicular process responsible for degradation and recycling across all eukaryotic cells. This pathway allows the breakdown of cytoplasmic components in the vacuole to eliminate damaged structures and recycle essential materials, thereby supporting cellular maintenance and the development of new cells. Autophagy is categorized into two main types: macroautophagy and microautophagy (Bassham et al., 2006). Of these, macroautophagy, the most extensively studied form, involves the formation of a specialized organelle called the autophagosome, which plays a central role in this process (Avila-Ospina et al., 2014).

In macroautophagy, bulk cytosolic components and organelles are encapsulated within a double-membrane structure known as an autophagosome. The outer membrane of the autophagosome subsequently fuses with the vacuolar membrane, delivering its inner membrane and enclosed cargo, referred to as the autophagic body, into the vacuolar lumen for degradation. In contrast, microautophagy involves the direct digestion of portions of the cytoplasm or specific organelles. This process occurs through the invagination of the vacuolar membrane, which leads to the formation of intravacuolar vesicles, also known as autophagic bodies, which are broken down by vacuolar hydrolases (Avila-Ospina et al., 2014).

Evidence highlights the importance of nutrient recycling in plants, as deficiencies in macro-nutrients like nitrogen significantly accelerate senescence and shorten the plant lifespan. For instance, in a study by Wingler et al. (2004), the addition of glucose to a low-nitrogen medium markedly hastened leaf senescence compared to plants grown in a low-nitrogen medium without glucose. The researchers proposed that glucose supplementation likely enhanced nitrogen utilization, depleting nitrogen from the medium more rapidly and consequently triggering increased nitrogen remobilization. This study underscores the critical role of the carbon/nitrogen balance in regulating the process of leaf senescence.

Chloroplasts contain up to 70% of the leaf proteins and house the majority of metabolic enzymes involved in critical processes such as photosynthesis, photorespiration, nitrogen assimilation, and amino acid biosynthesis. During senescence-induced chloroplast degradation, the processes of carbon and nitrogen assimilation are supplanted by the breakdown of chlorophyll and macromolecules like proteins and membrane lipids. Figure (3) presents a schematic representation of protein degradation events, highlighting their role in feeding electrons to the mitochondrial electron transport chain, supplying carbon skeletons to the TCA cycle, and recycling NH_4^+ to support nitrogen remobilization toward sink organs.

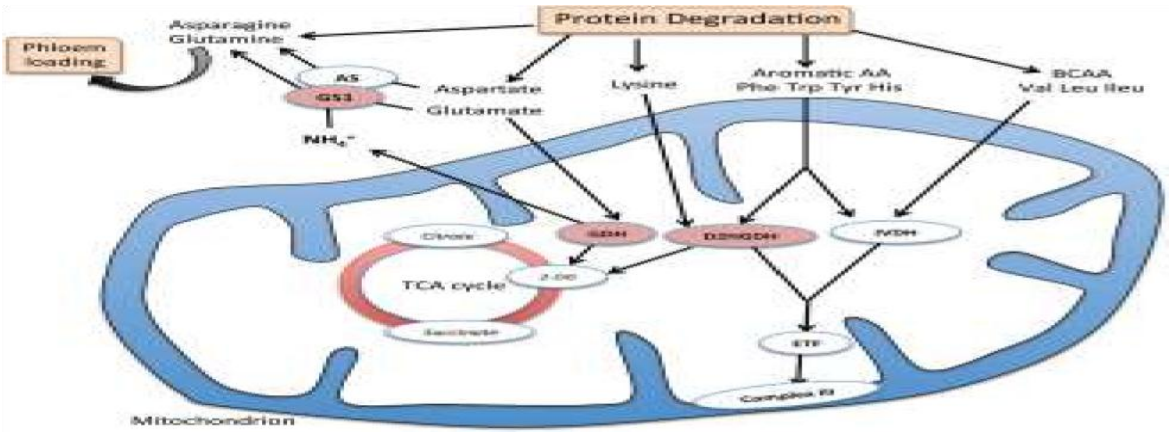

Fig (1). Schematic representation of the protein recycling comprising: providing carbon skeletons entering the TCA cycle, recycling NH4 + to support nitrogen remobilization to sink organs, and feeding electrons to the mitochondrial electron transport chain (Adapted from Araujo et al., 2011). ETF, electron transfer flavoprotein; IVDH, isovalerylCoA dehydrogenase; D2HGDH, 2-hydroxyglutarate dehydrogenase; GDH, glutamate dehydrogenase; GS1, cytosolic glutamine synthetase; AS, asparagine synthetase; BCAA, branched chain amino acids; AA, amino acids; TCA, tricarboxylic acid cycle (Avila-Ospina et al.2014).

Recent studies have demonstrated that autophagy is essential for the senescence-dependent degradation of Rubisco (Ishida et al., 2008). Additionally, it plays a crucial role both prior to and during leaf senescence, facilitating nutrient remobilization and the removal of cellular waste (Wingler et al., 2009).

Phosphorus poses significant nutritional challenges for plants due to its low solubility and limited mobility in soils, often necessitating the formation of mycorrhizal associations. For plants operating with marginal phosphorus availability, nucleic acids represent a substantial investment, particularly in species with large genomes. Beyond their primary roles as carriers of genetic information, nucleic acids can be considered valuable phosphorus storage compounds. Evidence for this comes from experiments where seeds were grown in a hydroponic medium devoid of phosphorus, producing mature but highly stunted plants with very few fertile seeds. This striking demonstration highlights the plant's ability to reuse phosphorus efficiently during development, achieved by continuously recycling the phosphorus present in the original seed through successive leaves and ultimately into the new seeds.

While RNA serves as the primary target for phosphorus remobilization, many plant nucleases can act on both RNA and DNA substrates (Yupsanis et al., 1996). When DNA is targeted by phosphorus-mobilizing nucleases, nicks and ladder-like fragmentation are likely to occur. The resulting DNA fragmentation patterns or TUNEL staining, often observed during these processes, may resemble markers of apoptotic or programmed cell death-like mechanisms in animal cells. However, it would be misleading to infer functional parallels between apoptosis in animals and senescence in plants based solely on these observations. In fact, Lee and Chen (2002) determined that cell death during rice leaf senescence does not follow an apoptosis-like pathway, as seen in animals.

It is well established that a protein like Rubisco not only performs an enzymatic function but also serves as a storage protein, with its nitrogen content being redistributed during senescence. Furthermore, programmed cell death (PCD) in roots facilitates the recycling of nucleotides and amino acids into the shoots, contributing to plant recovery and enhancing tolerance to stress conditions (Katsuhara and Shibasaka, 2000).

Nutrient recycling through programmed cell death (PCD) is particularly evident in the functional relationship between tapetal cells and developing pollen grains. Tapetal cells, with their short lifespan, remain fully functional despite being enucleate. During pollen development, the tapetum undergoes degradation to supply nutrients, metabolites, and sporopollenin precursors essential for microspore development. Defects in tapetal degradation have been linked to abnormal pollen coat and grain formation, ultimately causing severe male sterility (Zhang et al., 2008; Ariizumi and Toriyama, 2011). This degradation process is tightly regulated and exhibits hallmarks of PCD, including chromatin condensation, cell shrinkage, endoplasmic reticulum (ER) swelling, and mitochondrial persistence (Rogers et al., 2005).

6. Conclusions

Programmed cell death (PCD) is a cornerstone of plant life, contributing to both general and specific processes such as organ shaping, morphological adaptations, and defense mechanisms against abiotic and biotic stressors. The induction of PCD in plants exhibits distinct hallmarks, with its characteristics varying based on the activation trigger. PCD is often categorized functionally into developmental-regulated PCD (dPCD) and environmental-induced PCD (ePCD), as the complexity of its morphological and molecular features makes traditional classification challenging. A key component of the PCD process is the vesicular-mediated autophagy pathway, which facilitates the degradation of cytoplasmic components within the vacuole. This pathway is crucial for eliminating damaged materials and recycling them into raw resources, thereby sustaining new cell formation and maintaining vital cellular functions.

7. References

Ameisen, J. C. (2004). Looking for death at the core of life in the light of evolution. Cell Death & Differentiation, 11(1):4–10.

Araújo, W. L., Tohge, T., Ishizaki, K., Leaver, C. J., & Fernie, A. R. (2011). Protein degradation–an alternative respiratory substrate for stressed plants. Trends in plant science, 16(9), 489-498.

Ariizumi, T., & Toriyama, K. (2011). Genetic regulation of sporopollenin synthesis and pollen exine development. Annual review of plant biology, 62(1), 437-460.

Avila-Ospina, L., Moison, M., Yoshimoto, K., & Masclaux-Daubresse, C. (2014). Autophagy, plant senescence, and nutrient recycling. Journal of Experimental Botany, 65(14), 3799-3811.

Bassham, D. C., Laporte, M., Marty, F., Moriyasu, Y., Ohsumi, Y., Olsen, L. J., & Yoshimoto, K. (2006). Autophagy in development and stress responses of plants. Autophagy, 2(1), 2-11.

Beers, E. P. (1997). Programmed cell death during plant growth and development. Cell Death & Differentiation, 4(8), 649-661.

Bosch, M., & Franklin-Tong, V. E. (2008). Self-incompatibility in Papaver: signalling to trigger PCD in incompatible pollen. Journal of experimental botany, 59(3), 481-490.

Bury, M., Novo-Uzal, E., Andolfi, A., Cimini, S., Wauthoz, N., Heffeter, P., ... & Locato, V. (2013). Ophiobolin A, a sesterterpenoid fungal phytotoxin, displays higher in vitro growth-inhibitory effects in mammalian than in plant cells and displays in vivo antitumor activity. International Journal of Oncology, 43(2), 575-585.

Daneva, A., Gao, Z., Van Durme, M., & Nowack, M. K. (2016). Functions and regulation of programmed cell death in plant development. Annual review of cell and developmental biology, 32(1), 441-468.

Danon, A., Delorme, V., Mailhac, N., & Gallois, P. (2000). Plant programmed cell death: a common way to die. Plant Physiology and Biochemistry, 38(9), 647-655.

de Pinto, M. C., Locato, V., & De Gara, L. (2012). Redox regulation in plant programmed cell death. Plant, cell & environment, 35(2), 234-244.

de Pinto, M. C., Locato, V., Paradiso, A., & De Gara, L. (2015). Role of redox homeostasis in thermo-tolerance under a climate change scenario. Annals of Botany, 116(4), 487-496.

de Pinto, M. C., Locato, V., Sgobba, A., Romero-Puertas, M. D. C., Gadaleta, C., Delledonne, M., & De Gara, L.

(2013). S-nitrosylation of ascorbate peroxidase is part of programmed cell death signaling in tobacco Bright Yellow-2 cells. Plant Physiology, 163(4), 1766-1775.

Delledonne, M., Xia, Y., Dixon, R. A., & Lamb, C. (1998). Nitric oxide functions as a signal in plant disease resistance. Nature, 394(6693), 585-588.

Domínguez, F., & Cejudo, F. J. (2014). Programmed cell death (PCD): an essential process of cereal seed development and germination. Frontiers in plant science, 5, 366.

Escamez, S., & Tuominen, H. (2014). Programmes of cell death and autolysis in tracheary elements: when a suicidal cell arranges its own corpse removal. Journal of Experimental Botany, 65(5), 1313-1321.

Galluzzi, L., Pedro, B. S., Vitale, I. G. O. R., Aaronson, S. A., Abrams, J. M., Adam, D., ... & Kroemer, G. (2015). Essential versus accessory aspects of cell death: recommendations of the NCCD 2015. Cell Death & Differentiation, 22(1), 58-73.

Gómez Ros, L. V., Paradiso, A., Gabaldón, C., Pedreño, M. A., de Gara, L., & Ros Barceló, A. (2006). Two distinct cell sources of H 2 O 2 in the lignifying Zinnia elegans cell culture system. Protoplasma, 227, 175-183.

Greenberg, J. T. (1996). Programmed cell death: a way of life for plants. Proceedings of the National Academy of Sciences, 93(22), 12094-12097.

Gunawardena, A. H. (2008). Programmed cell death and tissue remodelling in plants. Journal of experimental botany, 59(3), 445-451.

Hussain, J., Chen, J., Locato, V., Sabetta, W., Behera, S., Cimini, S., ... & Vandelle, E. (2016). Constitutive cyclic GMP accumulation in Arabidopsis thaliana compromises systemic acquired resistance induced by an avirulent pathogen by modulating local signals. Scientific Reports, 6(1), 36423.

Huysmans, M., Coll, N. S., & Nowack, M. K. (2017). Dying two deaths—programmed cell death regulation in development and disease. Current opinion in plant biology, 35, 37-44.

Ishida, H., Yoshimoto, K., Izumi, M., Reisen, D., Yano, Y., Makino, A., ... & Mae, T. (2008). Mobilization of rubisco and stroma-localized fluorescent proteins of chloroplasts to the vacuole by an ATG gene-dependent autophagic process. Plant physiology, 148(1), 142-155.

Katsuhara, M., & Shibasaka, M. (2000). Cell death and growth recovery of barley after transient salt stress. Journal of Plant Research, 113(3), 239.

Kerr, J. F., Wyllie, A. H., & Currie, A. R. (1972). Apoptosis: a basic biological phenomenon with wideranging implications in tissue kinetics. British journal of cancer, 26(4), 239-257.

Klimešová, J., Nobis, M. P., & Herben, T. (2015). Senescence, ageing and death of the whole plant: morphological prerequisites and constraints of plant immortality. New Phytologist, 206(1), 14-18.

Koyama, T. (2014). The roles of ethylene and transcription factors in the regulation of onset of leaf senescence. Frontiers in plant science, 5, 650.

Kumpf, R. P., & Nowack, M. K. (2015). The root cap: a short story of life and death. Journal of experimental botany, 66(19), 5651-5662.

Lam,E.,Kato,N.&Lawton,M.(2001)Programmedcelldeath,mitochondriaandplanthypersensitiveresponse.Nature,14,848-853.

Lee, R. H., & Chen, S. C. G. (2002). Programmed cell death during rice leaf senescence is nonapoptotic. New Phytologist, 155(1), 25-32.

Liu, Y., Schiff, M., Czymmek, K., Tallóczy, Z., Levine, B., & Dinesh-Kumar, S. P. (2005). Autophagy regulates programmed cell death during the plant innate immune response. Cell, 121(4), 567-577.

Locato, V., Paradiso, A., Sabetta, W., De Gara, L., & de Pinto, M. C. (2016). Nitric oxide and reactive oxygen species in PCD signaling. In Advances in botanical research (Vol. 77, pp. 165-192). Academic Press.

Locato, V., Uzal, E. N., Cimini, S., Zonno, M. C., Evidente, A., Micera, A., ... & De Gara, L. (2015). Low concentrations of the toxin ophiobolin A lead to an arrest of the cell cycle and alter the intracellular partitioning of glutathione between the nuclei and cytoplasm. Journal of experimental botany, 66(10), 2991-3000.

Lockshin, R. A., & Zakeri, Z. (2004). Apoptosis, autophagy, and more. The international journal of biochemistry & cell biology, 36(12), 2405-2419.

Ma, L., Zhang, H., Sun, L., Jiao, Y., Zhang, G., Miao, C., & Hao, F. (2012). NADPH oxidase AtrbohD and AtrbohF function in ROS-dependent regulation of Na+/K+ homeostasis in Arabidopsis under salt stress. Journal of Experimental Botany, 63(1), 305-317.

Ma, W., Qi, Z., Smigel, A., Walker, R. K., Verma, R., & Berkowitz, G. A. (2009). Ca^{2+}, cAMP, and transduction of non-self perception during plant immune responses. Proceedings of the National Academy of Sciences, 106(49), 20995-21000.

Mammone, T., Gan, D., Collins, D., Lockshin, R. A., Marenus, K., & Maes, D. (2000). Successful separation of apoptosis and necrosis pathways in HaCaT keratinocyte cells induced by UVB irradiation. Cell biology and toxicology, 16, 293-302.

Moeder, W., & Yoshioka, K. (2008). Lesion mimic mutants: a classical, yet still fundamental approach to study programmed cell death. Plant signaling & behavior, 3(10), 764-767.

Mondal, R., Antony, S., Roy, S., & Chattopadhyay, S. K. (2021). Programmed cell death (PCD) in plant: molecular mechanism, regulation, and cellular dysfunction in response to development and stress. Regulation and dysfunction of apoptosis, 2, 1-20.

Ng, S., De Clercq, I., Van Aken, O., Law, S. R., Ivanova, A., Willems, P., ... & Whelan, J. (2014). Anterograde and retrograde regulation of nuclear genes encoding mitochondrial proteins during growth, development, and stress. Molecular plant, 7(7), 1075-1093.

Olvera-Carrillo, Y., Van Bel, M., Van Hautegem, T., Fendrych, M., Huysmans, M., Simaskova, M., ... & Nowack, M. K. (2015). A conserved core of programmed cell death indicator genes discriminates developmentally and environmentally induced programmed cell death in plants. Plant physiology, 169(4), 2684-2699.

Paradiso, A., de Pinto, M. C., Locato, V., & De Gara, L. (2012). Galactone- γ- lactone- dependent ascorbate biosynthesis alters wheat kernel maturation. Plant Biology, 14(4), 652-658.

Petrov, V., Hille, J., Mueller-Roeber, B., & Gechev, T. S. (2015). ROS-mediated abiotic stress-induced programmed cell death in plants. Frontiers in plant science, 6, 69.

Proskuryakov, S. Y., Konoplyannikov, A. G., & Gabai, V. L. (2003). Necrosis: a specific form of programmed cell death?. Experimental cell research, 283(1), 1-16.

Rabinovich, G. A. (2015). Essential versus accessory aspects of cell death: recommendations of the NCCD 2015.

Reape, T. J., & McCabe, P. F. (2008). Apoptotic- like programmed cell death in plants. New Phytologist, 180(1), 13-26.

Rogers, L. A., Dubos, C., Surman, C., Willment, J., Cullis, I. F., Mansfield, S. D., & Campbell, M. M. (2005). Comparison of lignin deposition in three ectopic lignification mutants. New Phytologist, 168(1), 123-140.

Sychta, K., Słomka, A., & Kuta, E. (2021). Insights into plant programmed cell death induced by heavy metals—Discovering a terra incognita. Cells, 10(1), 65.

Vacca, R. A., Valenti, D., Bobba, A., de Pinto, M. C., Merafina, R. S., De Gara, L., ... & Marra, E. (2007). Proteasome function is required for activation of programmed cell death in heat shocked tobacco Bright-Yellow 2 cells. Febs Letters, 581(5), 917-922.

Van Aken, O., & Pogson, B. J. (2017). Convergence of mitochondrial and chloroplastic ANAC017/PAP-dependent retrograde signalling pathways and suppression of programmed cell death. Cell Death & Differentiation, 24(6), 955-960.

Van Aken, O., & Pogson, B. J. (2017). Convergence of mitochondrial and chloroplastic ANAC017/PAP-dependent retrograde signalling pathways and suppression of programmed cell death. Cell Death & Differentiation, 24(6), 955-960.

van Doorn, W. G., Beers, E. P., Dangl, J. L., Franklin-Tong, V. E., Gallois, P., Hara-Nishimura, I., ... & Bozhkov, P. (2011). Morphological classification of plant cell deaths. Cell Death & Differentiation, 18(8), 1241-1246.

Van Hautegem, T., Waters, A. J., Goodrich, J., & Nowack, M. K. (2015). Only in dying, life: programmed cell death during plant development. Trends in plant science, 20(2), 102-113.

Wilkins, K. A., Poulter, N. S., & Franklin-Tong, V. E. (2014). Taking one for the team: self-recognition and cell suicide in pollen. Journal of experimental botany, 65(5), 1331-1342.

Wingler, A., Marès, M.,& Pourtau N (2004) Spatial patterns an metabolic regulation of photosynthetic parameters during le senescence. New Phytol 161:7.

Wingler, A., Masclaux-Daubresse, C., & Fischer, A. M. (2009). Sugars, senescence, and ageing in plants and heterotrophic organisms. Journal of experimental botany, 60(4), 1063-1066.

Wu, L., Chen, H., Curtis, C., & Fu, Z. Q. (2014). Go in for the kill: How plants deploy effector-triggered immunity to combat pathogens. Virulence, 5(7), 710-721.

Yupsanis, T., Eleftheriou, P., & Kelepiri, Z. (1996). Separation and purification of both acid and neutral nucleases from germinated alfalfa seeds. Journal of Plant Physiology, 149(6), 641-649.

Zhang, D. S., Liang, W. Q., Yuan, Z., Li, N., Shi, J., Wang, J., ... & Zhang, D. B. (2008). Tapetum degeneration retardation is critical for aliphatic metabolism and gene regulation during rice pollen development. Molecular plant, 1(4), 599-610.