Journal of Cell Science & Therapy
ISSN: 2157-7013

Nath and Lu, J Cell Sci Ther 2015, 6:2
DOI: 10.4172/2157-7013.1000202

Commentary Open Access

# A Paradigm of Reactive Oxygen Species and Programmed Cell Death in Plants

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**Rec date:** Feb 18, 2015; **Acc date:** Mar 28, 2015; **Pub date:** Apr 06, 2015



## Commentary

Plants are continuously exposed to a large variety of biotic and abiotic stresses during their successive stages of development [1-4]. Living organisms have different endogenous strategies to acclimate to a series of adverse environmental conditions. Animals can easily move from one place to another to avoid challenging environmental stresses. In contrast, due to immobile nature of plants, they cannot escape from adverse environmental conditions including high light, high temperature, chilling, drought, ultra-violet radiation during their consecutive stages of development [1-4]. Plants have evolved highly organized strategies to cope with such adverse environmental conditions [4-6]. Recent reports showed that upon certain threshold of these changes, plant cells can no longer maintain homeostasis due to malfunction of metabolic processes, and they initiate the defense program, the so called programmed cell death (PCD). PCD is a highly regulated and organized cell suicide process that is essential for all living organisms. PCD plays an important role in cell homeostasis maintenance, tissue specialization, removing of damaged or infected cells, and acclamatory response [3,7-9].

As previously mentioned, PCD is a tightly regulated and well organized process that controls the disassembly of cell, including condensation, shrinkage and fragmentation of both cytoplasm and nucleus and DNA laddering [7,10]. Necrotic cell death proceeds before cell swelling, lysis and leakage of cell content, [2,7,10]. Although PCD has been discovered for many years, the connection between abiotic stress-induced reactive oxygen species (ROS) and PCD is still poorly understood and remains to be investigated at the molecular level. Here, recent advancements on ROS-mediated PCD is summarized.

As shown in Figure 1, plant cells experience exogenous and endogenous stress during their successive stages of development including aging. ROS such as singlet oxygen ($^1O_2$), superoxide anion radical ($O^{2-}$), and hydrogen peroxide ($H_2O_2$) are constantly produced as byproducts of aerobic reactions during several metabolic processes, in cellular organelles, including chloroplasts, mitochondria peroxisomes. In addition to exogenous environmental factors such as different biotic and abiotic cues, plant cells are regulated by endogenous factors such as hormones. These endogenous factors also control ROS production and elicit other signaling molecules proceeding PCD (Figure 1). Depending on the concentrations, ROS may have a negative or positive role in cellular functions.

The negative role of ROS is related to oxidative stress which may result in damage to macromolecules. At an optimal concentration, ROS may play a positive role in cellular functions because it is also a secondary signaling molecule that controls PCD during unfavorable environmental condition [3,10-15]. Under unfavorable biotic and abiotic stress conditions, when ROS production exceeds the ROS scavenging capacity in plant tissues, excess ROS induce necrotic lesions as cytotoxins and alter the expression of genes involved in signal transduction and eventually cause cell death [11].

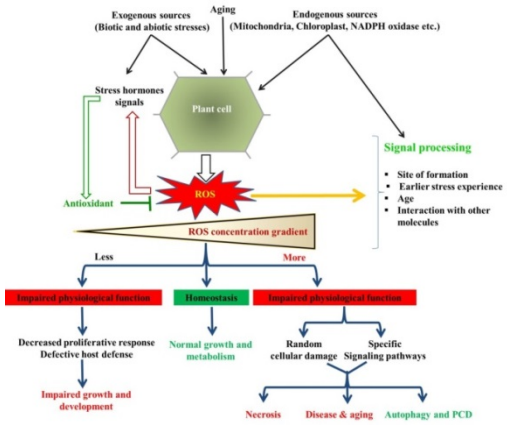


**Figure 1:** An overview of exogenous and endogenous factors which involve in the plant cell homeostasis and ROS production during PCD in plants. Several biotic and abiotic environmental factors and aging renders cell to produce ROS. Endogenously produced ROS, may influence a highly regulated signal transduction systems which provides the either positive or negative feedback control mechanisms. To keep cell in homeostasis condition, antioxidants system is always actively involves preventing elevation of ROS level in cell. As Different gradient levels of ROS decide the fate of the cell in plants. Less ROS leads impaired physiological function that causes defects in the plant growth. Optimum level of ROS keep cell in homeostasis condition for normal growth and development of plants. In contrast, more ROS level leads random cellular damage and specific signaling pathways for necrosis, disease and aging, and autophagy and PCD.

During plant aging, a highly regulated biological process called "autophagy" initiates PCD [2]. Senescence and autophagy are genetically controlled terminal stages of the plant life; these two processes play important roles on degradation and recycling of macromolecules, organelles, and cytoplasm during PCD [16,17]. It has been reported that the spatial-temporal correlation between increases in ROS contents and cell death serve as the initial indication of ROS-mediated PCD are [7,10]. ROS such as $O^{2-}$ and $H_2O_2$ are also known as key coordinators of senescence [14].

Antioxidants are essential to remove toxic ROS to defense plant against oxidative stress [11,18]. Chloroplasts, mitochondria and peroxisomes are the main cellular sites of ROS generation [11,12]. As above mentioned, accumulation of ROS under abiotic stresses affects

cellular functions by damaging nucleic acids and oxidizing proteins, and eventually causes cell death [1,11,13,14,19,20]. To minimize lethal effects, phototropic organisms employ an array of ROS-scavenging systems which involve superoxide dismutase, ascorbate peroxidase, catalase, peroxidase, and glutathione S-transferase [12,20]. In summary, adverse environmental conditions will cause over-accumulation of ROS, which leads to PCD; excess ROS could be removed via various ROS-scavenging systems.

## Acknowledgement

This work was supported by the U.S. National Science Foundation Grant MCB-1244008.

## References

1. Mittler R (2006) Abiotic stress, the field environment and stress combination. Trends Plant Sci 11: 15-19.
2. Pennell RI, Lamb C (1997) Programmed Cell Death in Plants. Plant Cell 9: 1157-1168.
3. Wituszynska W, Karpinski S (2013) Programmed Cell Death as a Response to High Light, UV and Drought Stress in Plants. In: Abiotic Stress-Plant Response and application in agriculture. InTech Publisher : 207-246.
4. Nath K, Jajoo A, Poudyal RS, Timilsina R, Park YS, et al. (2013) Towards a critical understanding of the photosystem II repair mechanism and its regulation during stress conditions. FEBS Lett 587: 3372-3381.
5. Nath K, Poudyal RS, Eom JS, Park YS, Zulfugarov IS, et al. (2013) Loss-of-function of OsSTN8 suppresses the photosystem II core protein phosphorylation and interferes with the photosystem II repair mechanism in rice (Oryza sativa). Plant J 76: 675-686.
6. Kirchhoff H (2014) Structural changes of the thylakoid membrane network induced by high light stress in plant chloroplasts. Philos Trans R Soc Lond B Biol Sci 369: 2013-2025.
7. Vranová E, Inzé D, Van Breusegem F (2002) Signal transduction during oxidative stress. J Exp Bot 53: 1227-1236.
8. Petrov V, Hille J, Mueller-Roeber B, Gechev TS (2015) ROS-mediated abiotic stress-induced programmed cell death in plants. Front Plant Sci 6: 69.
9. Talbot NJ, Kershaw MJ (2009) The emerging role of autophagy in plant pathogen attack and host defence. Curr Opin Plant Biol 12: 444-450.
10. Apel K, Hirt H (2004) Reactive oxygen species: metabolism, oxidative stress, and signal transduction. Annu Rev Plant Biol 55: 373-399.
11. Mittler R (2002) Oxidative stress, antioxidants and stress tolerance. Trends Plant Sci 7: 405-410.
12. Van Breusegem F, Dat JF (2006) Reactive oxygen species in plant cell death. Plant Physiol 141: 384-390.
13. Woo HR, Kim HJ, Nam HG, Lim PO (2013) Plant leaf senescence and death - regulation by multiple layers of control and implications for aging in general. J Cell Sci 126: 4823-4833.
14. Kim JH, Woo HR, Kim J, Lim PO, Lee IC, et al. (2009) Trifurcate feed-forward regulation of age-dependent cell death involving miR164 in Arabidopsis. Science 323: 1053-1057.
15. Coll NS, Smidler A, Puigvert M, Popa C, Valls M, et al. (2014) The plant metacaspase AtMC1 in pathogen-triggered programmed cell death and aging: functional linkage with autophagy. Cell Death Differ 21: 1399-1408.
16. Lim PO, Kim HJ, Nam HG (2007) Leaf senescence. Annu Rev Plant Biol 58: 115-136.
17. Karuppanapandian T, Moon JC, Kim C, Manoharan K, Kim W (2011) Reactive oxygen species in plants: their generation, signal transduction, and scavenging mechanisms. Aust J Crop Sci 5: 709-725.
18. Foyer CH, Noctor G (2005) Oxidant and antioxidant signalling in plants: a re-evaluation of the concept of oxidative stress in a physiological context. Plant, Cell & Environ 28: 1056–1071.
19. Asada K (2006) Production and scavenging of reactive oxygen species in chloroplasts and their functions. Plant Physiol 141: 391-396.
20. Gill SS, Tuteja N (2010) Reactive oxygen species and antioxidant machinery in abiotic stress tolerance in crop plants. Plant Physiol Biochem 48: 909-930.