

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

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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 ($O_2^{\cdot-}$), 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 preceding 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].

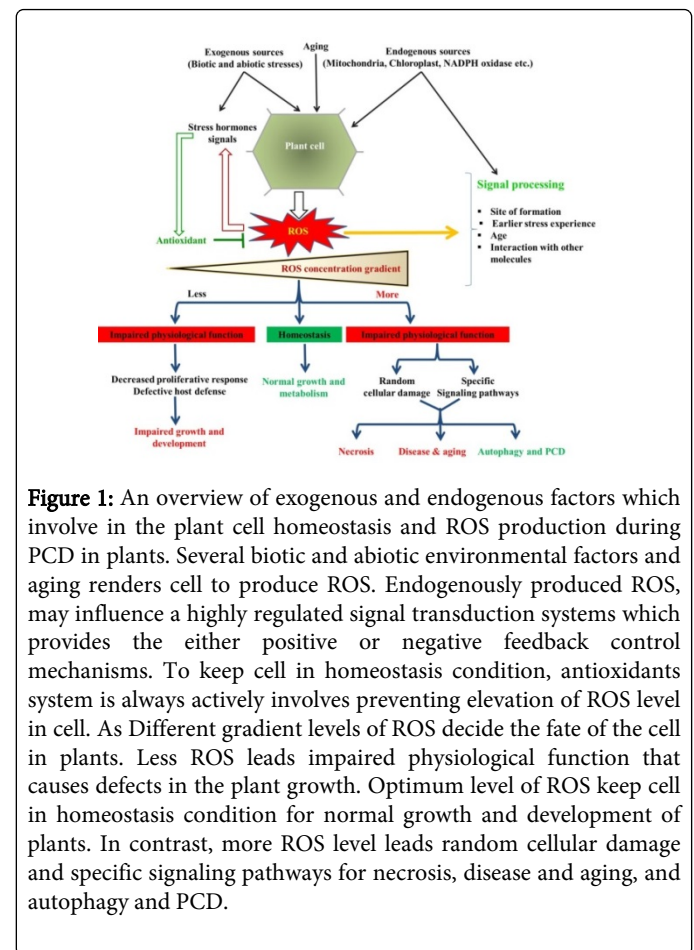


Figure 1: An overview of exogenous and endogenous factors which involve in the plant cell homeostasis and ROS production during PCD in plants. Several biotic and abiotic environmental factors and aging renders cell to produce ROS. Endogenously produced ROS, may influence a highly regulated signal transduction 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 $O_2^{\cdot-}$ 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.

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