

Research paper

ROS and oxidative burst: Roots in plant development

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ABSTRACT

Reactive oxygen species (ROS) are widely generated in various redox reactions in plants. In earlier studies, ROS were considered toxic byproducts of aerobic metabolism. In recent years, it has become clear that ROS act as plant signaling molecules that participate in various processes such as growth and development. Several studies have elucidated the roles of ROS from seed germination to senescence. However, there is much to discover about the diverse roles of ROS as signaling molecules and their mechanisms of sensing and response. ROS may provide possible benefits to plant physiological processes by supporting cellular proliferation in cells that maintain basal levels prior to oxidative effects. Although ROS are largely perceived as either negative by-products of aerobic metabolism or makers for plant stress, elucidating the range of functions that ROS play in growth and development still require attention.

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1. Introduction

Being sessile does not pose a curse to the plants, but opens the gate of opportunity for plants to utilize each aspect of every single entity available to it. The entity either becomes the part of its system or acts as a signal to specify function for maintaining the living state of the plant. Reactive oxygen species (ROS) qualify as an example to justify this viewpoint. ROS are the reactive products of oxygen that have the potential to damage the essential components of a living cell. Existence of ROS in the living system provides direct evidence to their origin (Czarnocka and Karpiński, 2018). Thus to acquire functional complexity, ROS gets enough time to deep down their rooting in origin and presence in all events of an organism life cycle. Shifting from its negative role to positive one has provided foundation to refresh our insight and to broaden it in its efficient and effective routes. Reactive oxygen species (ROS) are the reactive products of oxygen that damage essential components of living cells. More recently, ROS have been shown to play important roles in plant growth and development. For instance, ROS actively participate in germination and flowering, as well as the development of root apical meristem and shoot apical meristem, root hair

cells and pollen tubes, leaves, and lateral roots (Noctor et al., 2018; Mittler, 2017).

2. Sites of ROS production and ROS-scavenging

ROS production is important in plant cells because of its direct link with basic metabolic processes. ROS production is interlinked with ROS scavenging and cell survival depends on this link. The cell undergoes three states starting from ROS production, oxidative stress (ROS accumulation), and detoxification (Fig. 1) (Czarnocka and Karpiński, 2018). Whatever may be the reason of ROS production, the metabolic processes of life and natural selection does not eliminate the ROS production and detoxification. One important question is why ROS production is not naturally excluded? One of the more significant reasons has received much attention recently. Disruptions in the ROS production-scavenging cycle lead to ROS-mediated oxidative stress-induced damage, which may be lethal. ROS production sites in plant cells include chloroplasts, mitochondria, peroxisomes, and apoplasts (Table 1) (Corpas et al., 2015).

The chloroplast is a major source of ROS production in plants. Within the chloroplast, ROS production is largely caused by incomplete oxidation of water, plastosemi-hydroquinone H_2O_2 , and over-excitation of PSI (Fig. 2) (Pospíšil, 2016). H_2O_2 is produced via the action of chloroplastic superoxide dismutase on O_2^- and subsequently is converted to H_2O by ascorbate and glutathione (Chang

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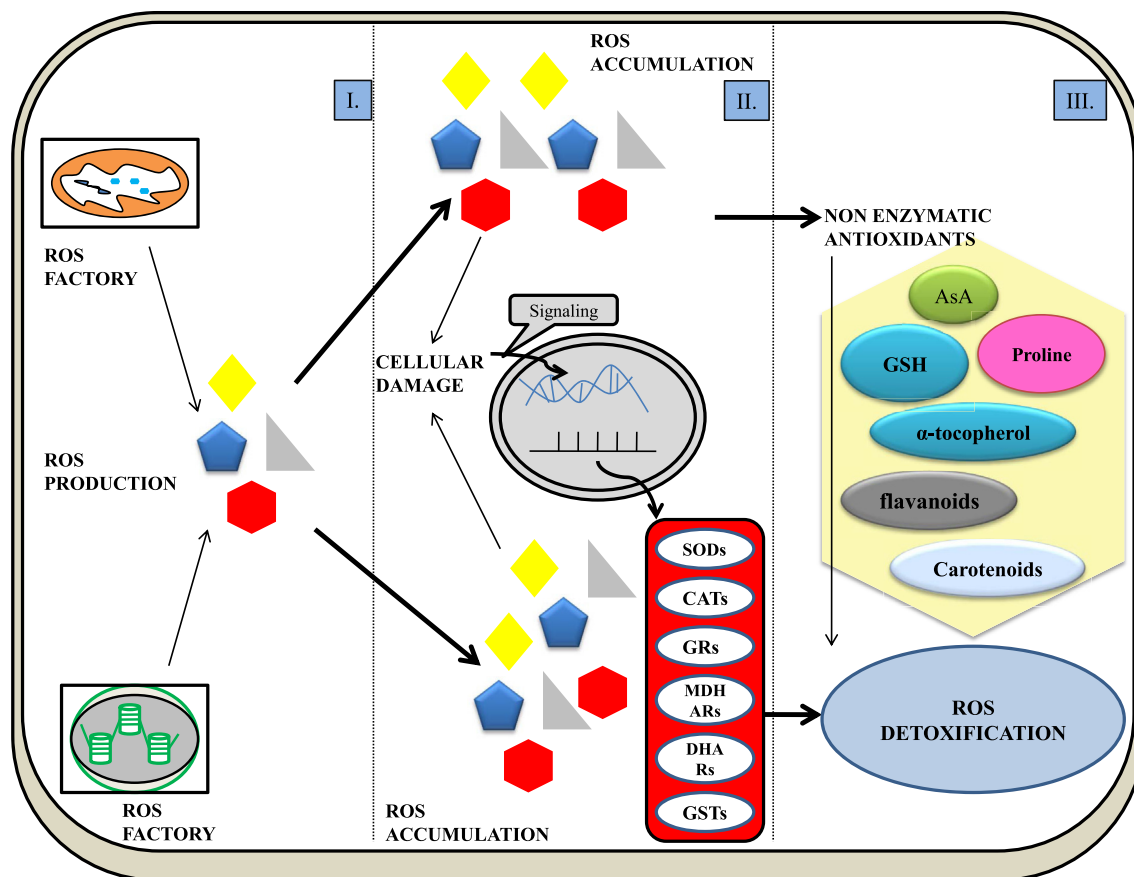


Fig. 1. Fate of ROS in cell. I. ROS production II. Oxidative stress: ROS accumulation-induced cellular damage causes the production of antioxidant components III. Detoxification: Enzymatic and non-enzymatic antioxidants (Proline, Carotenoids, Alpha-tocopherol, Glutathione, Ascorbic acid, Flavanoids and Carotenoids) act as ROS detoxicants. Superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPX), guaiacol peroxidase (POX), monodehydroascorbate reductase (MDHAR), glutathione-S-transferase (GSTs), dehydroascorbate reductase (DHAR), glutathione reductase (GR), ascorbate (AsA) and glutathione (GSH).

Table 1
ROS cellular localization and functions and the factors that elicit production.

S. no.	Site of production	Cause of ROS	ROS	Factors favoring ROS production	Functional status	References
1.	Cell wall	Class III peroxidases (PRXs), germin-like oxalate oxidases, amine oxidases, lipoxygenases, and quinone reductase	O_2 and H_2O_2	Ozone, high light, salinity, heavy metal, cold, heat, wounding and pathogen	Apoplastic polyamine-dependent programmed cell death induced by Ca^{2+} influx across the plasma membrane	Pottosin et al. (2014)
2.	Plasma membrane	NADPH oxidases	O_2 and H_2O_2	Ozone, high light, salinity, heavy metal, cold, heat, wounding and pathogen	Root hairs cell expansion, pollen tube growth, seed after-ripening, defense against pathogens, innate immunity and responses to abiotic stress	Wang et al. (2017)
3.	Chloroplast	Reaction centers	1O_2 , O_2 and H_2O_2	High light, UV radiation, low CO_2 , heat, cold, drought and pathogen	Local and systemic signaling, communication for proper non-photochemical quenching and develop systemic acquired acclimation or resistance	Wituszyńska and Karpiński (2013)
4.	Mitochondria	Mitochondrial electron transport chain	O_2 and H_2O_2	Heat, cold, drought, salinity, high light, UV radiation, heavy metal and hypoxia	Release of cytochrome c from mitochondria for programmed cell death	Aken and Van Breusegem (2015)
5.	Peroxisomes	Photorespiration and the fatty acid β -oxidation pathway	O_2 and H_2O_2	High light, low CO_2 , heat, salinity, drought and pathogen	Seed and pollen germination, and for stomatal movement senescence and fruit ripening	Corpas et al. (2017)

et al., 2009). Singlet oxygen (1O_2) is produced in the chloroplast under stress conditions (Wituszyńska and Karpiński, 2013). Excess light, for example, affects electron distribution stabilization, which

leads to the formation of triplet chlorophyll, generating 1O_2 (Tripathy and Oelmüller, 2012). 1O_2 provokes the loss of PSII via dysfunction of D1 polypeptide and pigment destruction.

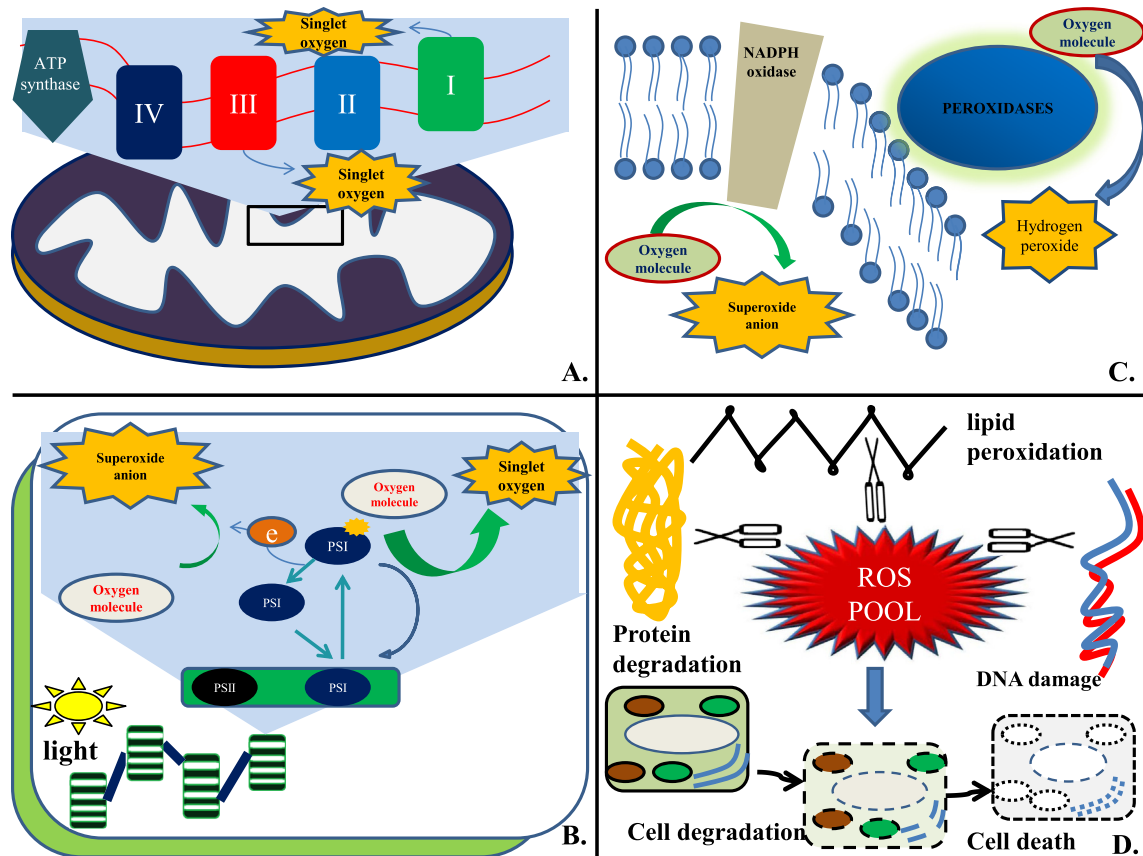


Fig. 2. Diagrammatic representation showing ROS production and cellular damage. A. Mitochondria (major site for ROS production via electron transport chain). B. Chloroplast (photosystems on thylakoids membrane generate ROS in the form of singlet oxygen and superoxide anions). C. Plasma membrane (NADPH oxidase and peroxidases at the membrane level produce ROS). D. Biomolecular damage (ROS accumulation causes protein degradation, lipid peroxidation and DNA damage).

Incomplete oxidation of water, plastosemi-hydroquinone H_2O_2 and overexcited PSI are the main causes involved in ROS production (Fig. 2) (Pospíšil, 2016). H_2O_2 results from the action of chloroplastic superoxide dismutase on O_2^- and subsequently convert to H_2O by ascorbate and glutathione (Chang et al., 2009).

Mitochondria are also a site of ROS such as O_2^- . The mitochondrial electron transport chain (ETC) has a sufficient supply of electrons to reduce O_2 to form ROS. Two main components of the mtETC that act as electron donor agents in the production of ROS are Complex I and Complex II (Fig. 2). Mitochondria generally produce ROS during respiration, but ROS production increases under stress conditions, which may lead to programmed cell death. To counteract oxidative

stress, ALTERNATIVE OXIDASE 1 (AOX1) and Mitochondrial SOD (Mn-SOD) are critical. AOX1 maintains a reduced state and decreases ROS production. The importance of AOX1 is clear from studies that show when AOX1 gene expression is decreased, cell death increases (Robson and Vanlerberghe, 2002). Mn-SOD acts in the matrix to detoxify O_2^- into O_2 and H_2O_2 (Navrot et al., 2007; Cvetkovska et al., 2013). Although ROS play significantly different roles in the mitochondria of animals and plants, ROS-mediated cell death in both requires cytochrome c (Mittler, 2017).

Peroxisomes, which are the sites of photorespiration and β -oxidation of fatty acid, also produce ROS. O_2^- is generated in the peroxisome matrix by the electron transport chain and the action of

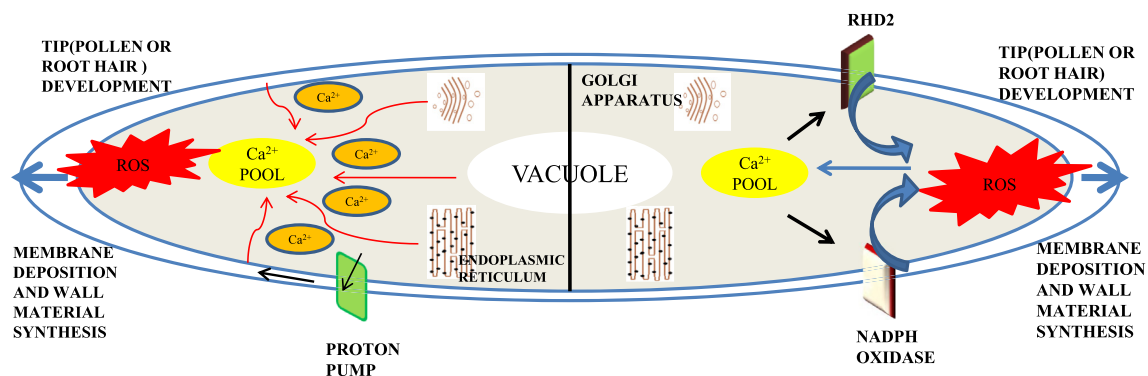


Fig. 3. Illustration showing the role of ROS and interacting components in tip development. Peak in calcium concentration in the cytoplasm activates the proton pump to acidify the cell wall and activate RHD2 and NADPH oxidase to produce ROS, thereby allowing tip development. RHD2 (Root hair defective gene 2), Ca^{2+} (Calcium).

xanthine oxidase. Other sources of ROS include the oxidation of glycolate and degradation of fatty acids. Glycolate, which is the final product of photorespiration in the chloroplast, enters the peroxisome where it is oxidized to glyoxylate with the aid of O_2 , leading to H_2O_2 production. The degradation of fatty acid by acyl-CoA oxidase also leads to H_2O_2 production. The ROS produced in the peroxisome are utilized in seed and pollen germination, fruit ripening, senescence, and stomatal movement (Corpas et al., 2017).

The apoplast is another source for ROS production, especially H_2O_2 . In the apoplastic plasma membrane, NADPH oxidase is the main source of O_2^- , which is further metabolized to H_2O_2 by superoxide dismutase (Fig. 2) (Marino et al., 2012; Mittler, 2017). Enzymes localized in the cell wall that are responsible for apoplastic ROS production include class III peroxidases, amine oxidases, germin-like oxalate oxidases, quinine reductase and lipoxygenases (Camejo et al., 2016).

The ROS scavenging system encompasses ROS homeostasis to ROS-dependent signaling under various environmental stresses (Noctor et al., 2018). ROS scavengers include ROS enzymatic as well as non-enzymatic systems that switch the cell from stressed to non-stressed phase. Enzymatic scavengers include Ascorbate Peroxidase (APX), Catalase (CAT), Glutathione Reductase (GRs), Superoxide Dismutase (SOD), Dehydroascorbate Reductase (DHAR), Glutathione-S-Transferase (GSTs) and Glutathione Peroxidase (GPX). The non-enzymatic scavenger system includes glutathione, α -tocopherol, flavonoids, carotenoids and proline. Both of these systems mitigate oxidative stress-induced damages (Wituszyńska and Karpinski, 2013).

3. ROS as a driver of cellular proliferation and differentiation during development

In multicellular organisms, the key events that determine growth and development are cell division and cellular differentiation. Disruption of the equilibrium between these two events leads to early termination of organogenesis, which may result in abnormal growth (Zhang et al., 2008). The transition from cellular proliferation to cell elongation during the earlier stages of differentiation are regulated by ROS homeostasis (Tsukagoshi et al., 2010). In *Arabidopsis* root, two ROS, oxygen radicals (O_2^-) and H_2O_2 , are differentially distributed. H_2O_2 accumulates in the elongation region, whereas oxygen radicals are located in the meristematic region (Dunand et al., 2007). In the zone of transition, however, the distribution of O_2^- and H_2O_2 overlap. The equilibrium between these two ROS is under the control of UPBEAT1 (*UPB1*), a basic helix loop helix transcriptional factor that is upregulated in the transition region of roots (Tsukagoshi et al., 2010). Mutant plants that overexpress *UPB1* have reduced root size due to smaller meristem size and fewer mature cells, while *upb1* mutants in which the *UPB1* transcription factor is absent are characterized by larger meristem size with elongated root cells. *UPB1* negatively regulates the genes that encode for a set of peroxidases. Interestingly, positional information in the transition region is provided by a gradient of oxygen radicals and H_2O_2 , which is required for cellular proliferation and differentiation. Hence, by mediating the gene expression of peroxidases, the requisite balance between oxygen radicals and H_2O_2 is maintained.

For the differentiation of root hairs *PFT1/MED25* (mediator complex PHYTOCHROME AND FLOWERING TIME 1) and *MED8* are essential. *PFT1/MED25* are preferentially confined for cell growth whereas *MED8* independently regulates the organ growth and ROS homeostasis in roots (Xu and Li, 2011). During the differentiation of root hairs, *PFT1* causes the production of ROS by promoting class III peroxidases to maintain balance elucidated by transcriptional profiling.

4. The function of ROS in diverse development stages

4.1. Seed dormancy and seed germination

ROS play a key role seed dormancy and germination in *Arabidopsis thaliana* (Leymarie et al., 2012), wheat (Ishibashi et al., 2008), barley (Bahin et al., 2011), sunflower (Oracz et al., 2007) and cress (Müller et al., 2009). In dry seeds, enzymatic activity is low and lipid peroxidation serves as a source of ROS. In hydrated seeds, increased metabolism is correlated with ROS production in chloroplasts, mitochondria, glyoxysomes, peroxisomes, and the plasma membrane (McDonald, 1999; Bailly, 2004). During seed imbibition, sub-cellular compartmentalization of ROS and their target molecule regulates the expression of various genes. Unlike dry seeds in which ROS production sites must be near targets (Bailly et al., 2008), during seed imbibition, water allows the translocation of ROS (e.g., H_2O_2) over greater distances. Therefore, in addition to altering the transcriptional activity of genes, ROS facilitate the oxidation of several components, stimulating transcription factors for signaling (Laloi et al., 2004). For instance, most labile H_2O_2 messenger depends upon the redox state of active proteins to accelerate the redox sensitive transcriptional factor for activating downstream cascades, triggering the MAPKs and oxidation of precise peptides (Petrov and Van Breusegem, 2012; Foyer and Noctor, 2013). Such oxidized proteins inhibit translation by oxidizing mRNA and also drive germination.

During seed germination, ROS are known to play roles in endosperm deterioration, seed reserve mobilization, pathogen defense, and programmed cell death (Fig. 6) (El-Maarouf-Bouteau and Bailly, 2008; El-Maarouf-Bouteau et al., 2013). ROS action during seed germination relies on interactions with abscisic acid (ABA), gibberellic acid (GA), and ethylene (ET), phytohormones that regulate seed dormancy and germination. In cereals, ROS-mediated effects on germination are inhibited by ABA via the promotion of ROS-scavenging enzyme activity. However, these effects are counteracted by GA, which downregulates ROS-scavenging enzymes and induce ROS-mediated programmed cell death. ROS mediate cell wall polysaccharide deterioration and the activation of calcium (Ca^{2+}) channels and MAPKs, which allows radicle enlargement (Diaz-Vivancos et al., 2013). The weakening of cell wall polysaccharide chains is regulated by apoplastic OH radicals, which mediate the breakdown of chitosan, pullulan-like polysaccharides, and hyaluronate (Stern et al., 2007). When GA interacts with the aleurone layer, it triggers α -amylase synthesis and releases other hydrolytic enzymes. In contrast, enzymes of the antioxidant system repress the production of hydrolytic enzymes. ABA acts as a negative regulator in seed germination via suppression of ROS production, although it also acts as a positive regulator that induces dormancy (Ishibashi et al., 2012; Finkelstein et al., 2008). Another phytohormone that plays a positive regulatory role in seed germination is ethylene. For instance, in soybean seeds ethylene production becomes elevated as a result of ROS production during imbibition (Ishibashi et al., 2013). During germination, ethylene and ABA antagonistically regulate seed germination (Fig. 4) (Arc et al., 2013).

The ratio of ABA and GA regulates seed dormancy. Disturbance to this balance directly affects seed dormancy. For instance, high ABA/GA ratios favour dormancy, whereas low ABA/GA ratios result in a break in dormancy. The exogenous application of H_2O_2 decreases ABA levels and increases GA concentrations, which triggers the release of dormancy (<https://www.frontiersin.org/articles/10.3389/fpls.2016.00864/full>, Graeber et al., 2010; Oracz and Karpinski, 2016). Studies on the crosstalk between ROS-ABA and Ethylene-GA explain the role of ROS in the alleviation of seed dormancy in barley (Bahin et al., 2011). The establishment of

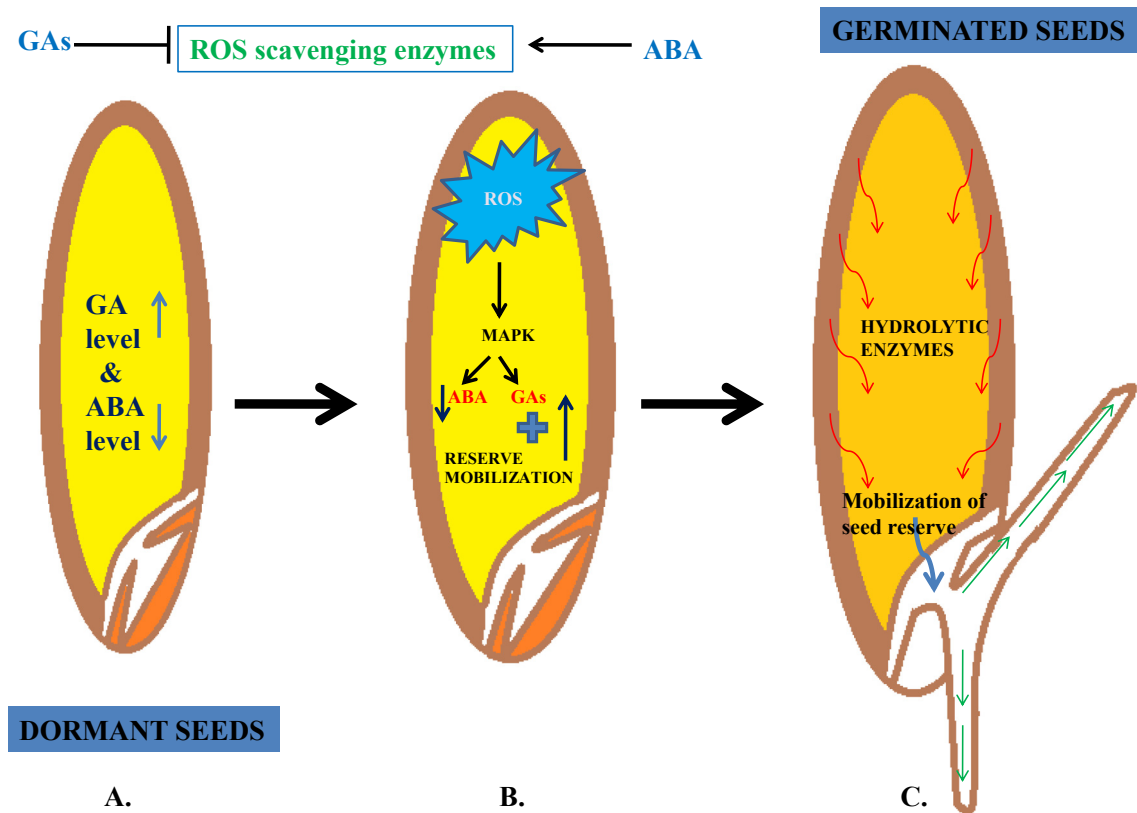


Fig. 4. Model depicting the role of ROS in seed dormancy and germination. The levels of phytohormones ABA and GAs regulate ROS scavenging. ROS signaling mediates transcription factor MAPKs to activate the hydrolytic enzymes responsible for reserve degradation and its mobilization. MAPK (mitogen activating protein kinase).

crosstalk between ROS and hormonal signaling in seeds also triggers dormancy release, after-ripening, and germination (<https://www.frontiersin.org/articles/10.3389/fpls.2016.00864/full>, Bahin et al., 2011). Accumulation of ROS takes place apoplastically in the radicle and endosperm region of seeds under the regulation of hormones. In *Lepidium sativum*, ABA inhibits endosperm rupture, whereas GA counteracts the effect of ABA in the radicle to stimulate seed germination (<https://www.frontiersin.org/articles/10.3389/fpls.2016.00864/full>, Graeber et al., 2010).

4.2. Meristem development

In both the shoot apical meristem (SAM) and the root apical meristem (RAM), stem cells are organized in a central zone (CZ) surrounding an organizing center called the organizing zone (OZ), or quiescent center (QC). The maintenance of the meristem relies on exchange of information between the OZ/QC and central zone as well as feedback from differentiated tissues. The chief differences between root and shoot meristem development are the gene networks that regulate their activity and their response to growth hormones. Notably, the activity of SAM and RAM has been shown to be affected by the interplay between ROS, redox components and phytohormones (Schippers et al., 2016).

RAM activity is responsive to changes in cellular redox status. For instance, application of exogenous H_2O_2 reduces the number of meristem cells in the RAM (Tsukagoshi et al., 2010). In addition, DNA damage leads to H_2O_2 accumulation through FLAVIN-CONTAINING MONOOXYGENASE 1, and decreases root meristem size, indicating that H_2O_2 acts as a negative regulator of the RAM. Gradients in ROS have been reported in the different zones of

roots, with H_2O_2 peaks in the zone of elongation, and O_2^- peaks in the zone of cell division (Fig. 5) (Rubio-Díaz et al., 2012; Tsukagoshi, 2016). Such distributions suggest that O_2^- and H_2O_2 act antagonistically (Zeng et al., 2017). Both O_2^- and H_2O_2 are associated with the maintenance and differentiation in the SAM by regulating the expression of the transcription factor WUS (Figs. 5 and 6; Table 2). O_2^- up regulates WUS expression, whereas H_2O_2 accumulates in the peripheral zone and is associated with cell differentiation (see Fig. 5).

QC cells remain in a highly oxidized environment. The oxidized forms of glutathione and ascorbate are present and NADPH is barely detectable. In adjacent cells, however, higher antioxidant capacities and a more reducing environment is detected. Moreover, ROS-associated genes are differentially expressed in specific SAM and RAM tissues (Tognetti et al., 2017).

Disrupted glutaredoxin (GRX) activity is closely associated with meristem deficiencies. In *Arabidopsis*, GRXS17 controls the translocation and sensitivity of auxin (Cheng et al., 2011). In maize (*Zea mays*), GRX ABERRANT PHYLLLOTAXY (ABPHYL2) regulates shoot meristem size and phyllotaxy through post translational modification the bZIP transcription factor FASCIATED EAR4 (Yang et al., 2015; Pautler et al., 2015). Moreover, *Arabidopsis* GRXs ROXY1 and ROXY2 reduce the disulfide bonds in the heteromeric TGA9/TGA10 transcription factor complex, a step that is required to activate gene expression during floral transition (Mурmu et al., 2010). The auxin-synthesizing Flavin monooxygenase YUCCA6 has thiol reductase activity, suggesting that there is a link between redox and auxin pathways (Cha et al., 2015).

The plastid thioredoxin TRXm3 regulates ROS homeostasis adjacent to plasmodesmata, targeting callose deposition that ultimately regulates transport through the plasmodesmata (Benitez-

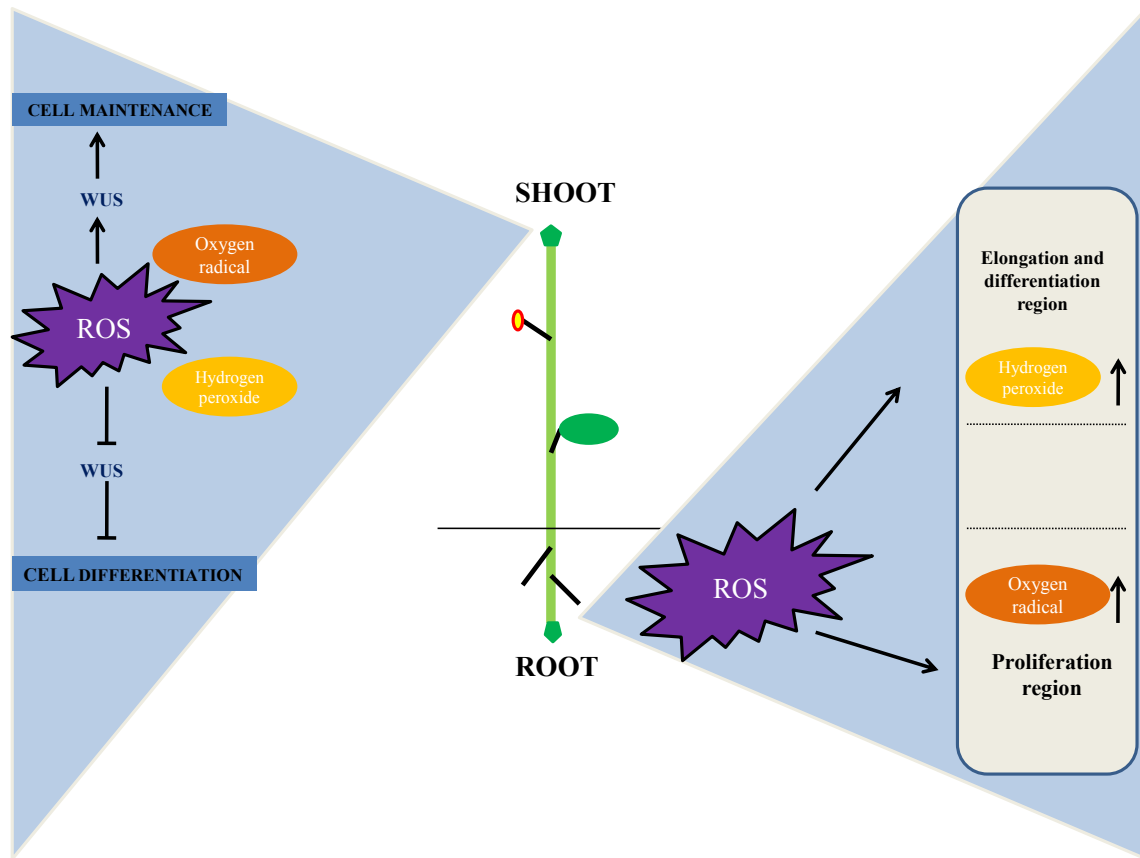


Fig. 5. Differential response of ROS components in meristem development. In the SAM, H_2O_2 and O_2 regulate the expression of the WUSCHEL (WUS) gene during cellular differentiation and maintenance respectively. In the root tip, increase in H_2O_2 induces elongation and differentiation whereas O_2 induces proliferation. SAM (Shoot apical meristem), WUS (WUSCHEL).

Alfonso et al., 2009). ROS also interact with the plant defense hormone salicylic acid (SA). In both *Arabidopsis* and rice, ABNORMAL INFLORESCENCE MERISTEM (*AIM1*) plays key role in the SA biosynthetic pathway and is required for meristem development. At the transcriptional level, SA downregulated WRKY transcription factors and thus alleviates the repression of anti-oxidative enzymes, such as glutathiones and catalases (Bussell et al., 2014; Xu et al., 2017).

4.3. Leaf development

The development of terminal plant organs entails a complex harmonization of cell proliferation and cell expansion (Lu et al., 2014). Leaf development is characterized by the proliferation of meristematic cells followed by expansion without further division (Beemster et al., 2005). This cell expansion requires changes to the structure and content of the plant cell wall. These cell wall changes are mediated by peroxidase-associated ROS gradients. Apoplastic peroxidases directly regulate the rigidity of cell wall by either restricting or promoting cellular extension (Lee et al., 2013). Cell wall peroxidases operate as cell wall loosening agent by producing O_2 , which break down cell wall polysaccharides (Müller et al., 2009). Cell wall stiffness is enhanced by the production of H_2O_2 , which increases cross linking (Table 2). Studies in *Arabidopsis* have shown that a repressor of peroxidases called KUODA1 (*KUA1*) acts as a promoter of cell expansion during leaf development by changing ROS homeostasis (Fig. 6). Mutant plants that overexpress *KUA1* are characterized by larger leaves with massive cells. In

addition, *kua 1* mutants have increased levels of H_2O_2 and increased class III peroxidase activities. Disruption of *KUA1* activity triggers peroxidase activity, which results in leaves with smaller cells. These findings indicate that *KUA1*-mediated ROS homeostasis regulates cell expansion and organ size during leaf development (Lu et al., 2014).

4.4. Tip development

Root hair cells and pollen tubes show tip expansion growth mediated by membrane deposition and wall material synthesis in the direction of elongating cells. Cell expansion must be precisely regulated to produce the correct size and shape. Polar cell growth is maintained by oscillatory feedback loops that consist of three main components. One of the main components is ROS that, with pH and Ca^{2+} ions maintain polar cell growth (Mangano et al., 2016). During cell expansion, NADPH oxidase and class III peroxidases regulate apoplastic ROS homeostasis to affect cell wall properties. Expansion in polar cells of root hair cells and pollen tubes are associated with higher cytoplasmic Ca^{2+} parallel with the apoplastic ROS production in the apical zone (Figs. 3 and 6) (Steinhorst and Kudla, 2013). Ca^{2+} is derived from vacuoles, ER, Golgi bodies, the cell wall and the exterior of the cell; the apoplastic pH is altered by plasma membrane-localized H^+ pump activation or deactivation, which releases Ca^{2+} from the cell wall and exterior into the cytosol from cell wall and exterior. Fluctuating Ca^{2+} concentrations are regulated by auto-inhibitory P-type II B Ca^{2+} ATPases, which facilitate the movement of Ca^{2+} into the apoplast, and $\text{H}^+/\text{Ca}^{2+}$ antiporters that supplement Ca^{2+} movement and H^+ influx into the cytoplasm.

Table 2
Role of ROS in plant development.

S. no.	Developmental event	Site of action	ROS action	ROS type	Plant	References
1.	Release of dormancy and germination	Seed	Oxidation of specific peptides and MAPKs activation	H ₂ O ₂	<i>Zinnia elegans</i> , maize, wheat and soybean	Singh et al., (2016); Basbous-Serhal et al., (2017)
2.	Seed germination	Aleurone cells	PCD of aleurone layer	—	Cereal seeds	Yadegari and Drews (2004)
3.	Leaf development	Leaves meristematic cells	Cell wall loosening and rigid cross-linking of cell wall components	H ₂ O ₂ and O ₂	<i>A. thaliana</i>	Lu et al. (2014)
4.	Leaf senescence	Senescent leaves	Crucial in regulation of senescence signaling	¹ O ₂ , H ₂ O ₂	—	Bhattacharjee (2012)
5.	Senescence	Foral meristems	Protect the initial growth of the ovule, sepals, and petals after accomplishing the death of petal cells	H ₂ O ₂	Daylily plant	Halliwell and Gutteridge (1989); Tripathi and Tuteja (2007)
6.	Development of trichome	Trichome initials	Switched the mitosis to endoreduplication, branching of cells, expansion, and cell death	H ₂ O ₂ burst	—	Hulskamp (2004)
7.	Development of male sex organs	Tapetal cells	Dictate the correct timing of tapetal PCD	H ₂ O ₂	<i>A. thaliana</i>	Durme and Nowack (2016)
8.	Development of pollen tube on pistil	Pistil	Attract and guide the pollen tube growth by deteriorating the cells of pistil in a programmed manner & activating Ca ²⁺ permeable channels to alter the cell wall extensibility	—	<i>A. thaliana</i> ,	Duan et al., (2014); Lassig et al., (2014)
9.	Self-incompatibility during pollination	Stigma	Induced PCD in incompatible pollen	H ₂ O ₂	<i>Papaver</i>	Wilkins et al. (2011)
10.	Development of xylem tracheary elements	Vascular bundle cells	secondary wall differentiation or trigger xylem differentiation by PCD	H ₂ O ₂	—	Ros Barceló (2005)
11.	Development of aerenchyma	Internode of stem	Induced PCD	H ₂ O ₂	rice	Steffens et al. (2011)
12.	Rhizogenesis	Root	—	O ₂ (oxygen radical)	—	Libik-Konieczny et al. (2015)
13.	Lateral root development	Root	Promoting transition from cellular proliferation to differentiation	H ₂ O ₂ and O ₂	<i>A. thaliana</i>	Manzano et al. (2014)
14.	Root hair development	Epidermal root cells	Activation of MAPKs cascade	—	<i>A. thaliana</i>	Mangano et al. (2016)

Such elevated levels of cytosolic Ca²⁺ in tip zone are required to activate NADPH oxidase (NOXs). NADPH oxidase generates ROS, which trigger the release of Ca²⁺ into the cytosol (Duan et al., 2014).

The ROOT HAIR DEFECTIVE 2 (*RHD2*) gene, which transfers of electrons to its acceptor from NADPH, leads to ROS production. *rhd2* (root hair defective 2) mutants properly initiate develop of protuberances on epidermal cells but do not show tip growth elongation (Foreman et al., 2003). *oxi 1* mutants have demonstrated that OXIDATIVE BURST INDUCIBLE 1 (*OXI1*), an important Ser–Thr kinase, plays a role in root hair elongation (Rentel and Knight, 2004). During root hair growth, *RHD2* triggers ROS resulting in the establishment of MAPKs cascade via *OXI1*. This ROS burst is essential for pollen tube rupture and sperm release (Duan et al., 2014). NADPH, Ca²⁺ and pH are the factors that oscillate in such a way that with their climax concentration, growth is favored (Lovy-Wheeler et al., 2006; Macpherson et al., 2008). For instance, in root hair, there is highest oscillatory fluctuation in cytoplasmic as well as apoplastic pH. Apoplastic ROS level increases the growth by 7–8s, whereas cytoplasmic Ca²⁺ fluctuation suspends the growth oscillation by approximately 5–6s (Monshausen et al., 2009). Pollen tube growth was found to be delayed by 11s with the oscillation in cytoplasmic Ca²⁺ concentration (Pierson et al., 1994). Application of H₂O₂ pauses the tip growth and its scavenging continues tip growth, thus suggesting the importance of ROS in cell wall rigidity (Monshausen et al., 2007).

4.5. Lateral root development

The interplay between auxin and ABA is crucial for the development of the lateral root. Auxin triggers the separation of the

pericycle initials and cellular expansion, whereas ABA is necessary to balance the equilibrium between cellular proliferation and cell differentiation in both the meristem and lateral primordia of the root (Table 2) (Lavenus et al., 2013). Both phytohormones induce ROS production to promote cell expansion in growing roots. IAA is degraded by peroxidases, decreasing auxin pools (Cosio et al., 2008). As observed in tobacco plants, decreased levels of free available IAA in the roots elicits significant changes in auxin that inhibit lateral root formation (Moriwaki et al., 2011). Almost all *gpx* (glutathione peroxidase) mutants have enlarged lateral root primordia, indicating that the redox-mediated GPX family plays a role in regulating root architecture (Passaia et al., 2014). GPX1 and GPX7 are the major peroxidases that regulate root architecture, lateral root development, and auxin-mediated lateral root formation. Several factors contribute to the development of later roots, including oxygen radicals (O₂) and H₂O₂ as well as increased ROS production initiated by enzymes such as lipoxygenases, cytochrome P450 carrier proteins and AtrbohC (Manzano et al., 2014). Overall, lateral root emergence is determined by peroxidases through ROS signaling that promotes the transition from cellular proliferation to cell differentiation (Fig. 6).

5. Negative consequences of ROS

Oxidative stress occurs when the production of enhanced reactive oxygen species exceeds their degradation. Numerous factors are capable of disturbing ROS equilibrium in plants, including drought, high light, and salinity. When ROS target vital biomolecules (i.e., DNA, lipids, and proteins), they affect cell

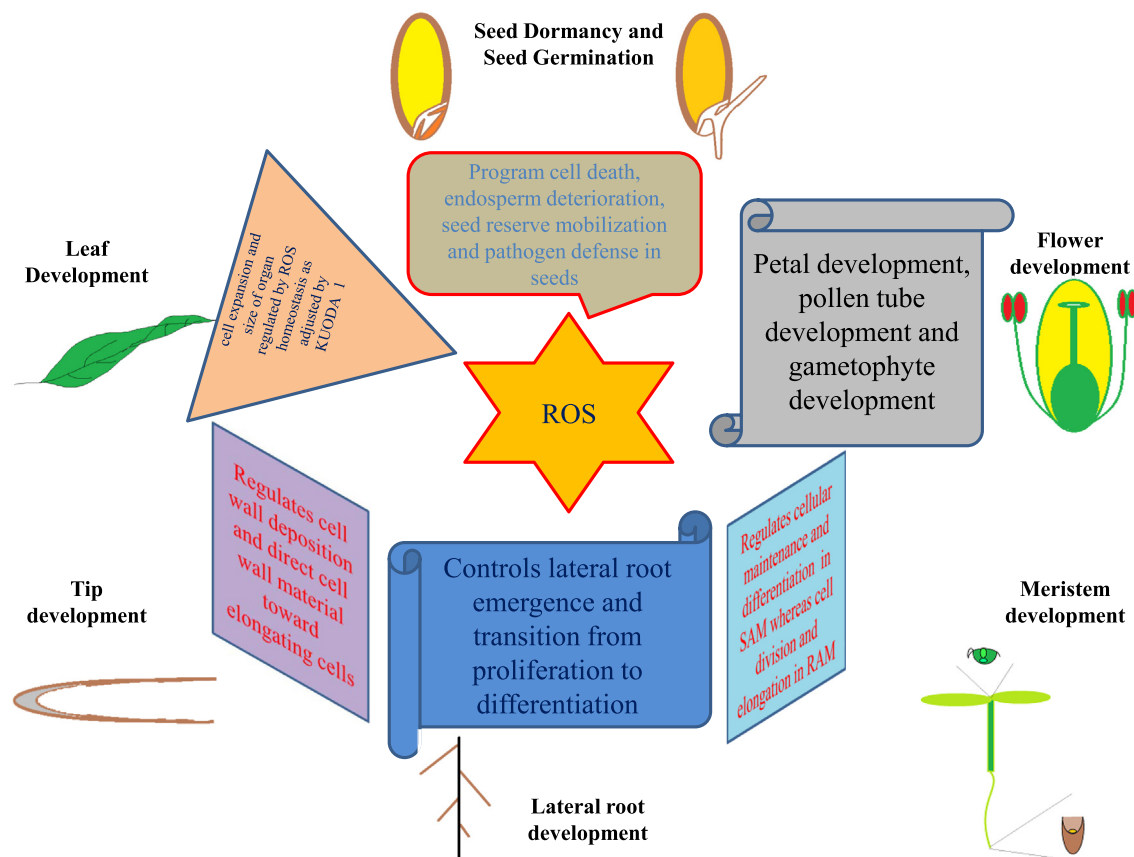


Fig. 6. Schematic overview of the ROS functional aspects at different phases of plant development.

Table 3

Life span, diffusion range and oxidative damage of ROS.

S. no.	ROS type	Half life	Diffusion range	Target of ROS	Oxidative damage	References
1.	$^1\text{O}_2$	3 μs	100 nm	Proteins, lipids, nucleic acids, and pigments	Lipid peroxidation, photosystem II activity loss and PCD	Das and Roychoudhury (2014)
2.	O_2^-	2–4 μs	30 nm.	Proteins	Oxidize enzymes containing the [4Fe–4S] clusters (aconitase or dehydratase) and reduce cytochrome C	Halliwell (2006)
3.	OH^-	1 μs	1 nm	Proteins, lipids and nucleic acids	Lipid peroxidation, production of cytotoxic lipid aldehydes, deoxyribose oxidation, removal of nucleotides, DNA-protein crosslinks, and strand breakage	Hossain et al. (2015)
4.	H_2O_2	1 ms	1 μm	Cysteine and methionine residues, and oxidize thiolates	Oxidation of Calvin cycle enzymes, transcription factors, signaling kinases, phosphatases, proteases, RNA-binding proteins and cell death	Waszczak et al. (2015)

physiological pathways, signaling cascades, membrane properties, which ultimately cause cell death (Fig. 2; Table 3).

5.1. DNA

DNA is a potential target of ROS damage and numerous environmental stresses have the potential to cause DNA degradation in plants (Das and Roychoudhury, 2014). Regardless of the source of DNA, damage ultimately causes aberrations in the resulting protein, which affects various aspects of cell physiology. ROS attacks break

DNA strands, remove and/or alter nucleotides, and oxidize deoxyribose.

ROS oxidize both deoxyribose and DNA base units. For instance, hydroxyl radicals can react with the deoxyribose backbone as well pyrimidine and purine bases. These oxidative attacks on DNA generally cause several mutagenic aberrations. For instance, hydrogen liberation from deoxyribose leads to sugar damage. However, when ROS removes hydrogen atoms from deoxyribose at C-4 position, additional radicals are produced, which leads to DNA single strand breaks (Evans et al., 2004). The addition of OH radicals

also damages bases (Halliwell, 2006; Das and Roychoudhury, 2014). Specifically, ROS directly cause mutations by altering G:C sites and indirectly damage DNA by generating potential products of macromolecules (lipids).

The reactivity of ROS varies for different molecules. Fe^{2+} is the most reactive towards ROS damage as it functions in the Fenton reaction to form hydroxyl radical (Mignolet-Spruyt et al., 2016). Hydroxyl ions target DNA and DNA binding proteins, which leads to cross-linking between the protein and DNA and is lethal without repair before transcription and replication. Chloroplastic and mitochondrial DNA are more vulnerable to ROS damage than chromosomal DNA. This is because of the absence of histone incorporation and presence of ROS production sites in these organelles. Thus, excessive ROS production leads to permanent DNA damage and ultimately to lethality in the cell.

5.2. Protein

In plants exposed to various stresses, protein modification increases. Generally, tissue rich in oxidative damage is recognized by the presence of high level of protein carbonylation, which acts as a marker for protein oxidation. ROS damage proteins in a variety of ways either directly or indirectly. Direct consequences of ROS attack on proteins include carbonylation, disulphide bond formation, glutathionylation and nitrosylation. Indirectly, ROS damage proteins via the products of fatty acid peroxidation, which bind with proteins to alter their activity (Yamauchi et al., 2008; Sharma et al., 2012). In addition, extreme ROS mediate the aggregation of products of cross-linked reactions, alter electric charge, fragment amino acid peptide chains, modify site-specific amino acids and increase the susceptibility of proteins to proteolysis.

The degree of ROS damage to proteins varies depending on amino acid composition. Sulfur- and thiol-containing amino acids are more vulnerable to ROS attack. For example, activated oxygen removes the H atom from cysteine amino acids to cross link with other equivalent cysteine amino acids via disulphide bonds. Furthermore, oxygen binds with methionine to generate methionine-sulphoxide derivative products. Oxygen radicals target iron-sulfur centers to permanently inactivate enzymes. Iron metal present in its binding form, located on the cation binding site and at this site metal undergoes Fenton reaction to generate hydroxyl radical in order to quickly oxidize the amino acids in the or nearby cation binding site of protein (Mittler, 2017).

5.3. Lipids

Lipid peroxidation increases ROS above threshold levels, which interfere with the functions of cellular and organelle membranes. Accordingly, lipid peroxidation is correlated with ROS production. Hence, lipid peroxidation levels during stress conditions are good indicators of ROS-induced damage to cell membranes. Such lipid peroxidation potentially damages DNA and proteins by generating the radicals of lipid derivatives, thus worsening oxidative stress. For instance, in the peroxidation of unsaturated fatty acids in phospholipids malondialdehyde damages the cell membrane.

ROS target double or unsaturated bonds and the ester bonds between fatty acid and glycerol resting on phospholipids (Sharma et al., 2012). Polyunsaturated fatty acids are more vulnerable to ROS attack. Membrane properties are altered due to polyunsaturated fatty acid peroxidation by ROS attack resulting in chain cleavage (Das and Roychoudhury, 2014). Furthermore, a single hydroxyl radical can mediate lipid peroxidation of a number of fatty acids.

The lipid peroxidation process is divided into three steps: initiation, progression, and termination. The initiation step is the rate limiting step that is activated by O_2 . Hydroxyl radicals and O_2

generate conjugating diene hydroperoxides and lipid peroxy radicals. These radicals, which are extremely reactive and capable of continuing the chain reaction, react with the methylene group of polyunsaturated fatty acids. The lipid hydroperoxides generated in the reaction undergo cleavage by reduced metals. Lipid hydroperoxide decomposes to produce numerous reactive species such as aldehydes, alkanes, alcohols, lipid alkoxyl radicals and lipid epoxides.

6. Concluding remarks and perspectives

Although ROS are considered messengers that lead to oxidative signaling, they may play two roles in plant biology. First, ROS trigger biological activities in response to stress. Second, evidence from several decades indicates that ROS are involved in plant growth and developmental processes. ROS regulate the cell cycle, seed dormancy and germination, root growth, pollen tube and leaf development and more. Much progress has been made in understanding the roles and mechanisms by which ROS regulate the plant life cycle. Despite our understanding of ROS production and activities, one emerging question is how ROS function and communicate between cell compartments. Finally, the range of ROS function still needs much attention and provides an opportunity for researchers interested in this area of plant physiology.

Declaration of Competing Interest

The authors declares that there is no conflict of interest.

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