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Review article

A REVIEW ON EFFECT OF SENESCENCE IN PLANTS AND ROLE OF PHYTOHORMONES IN DELAYING SENESCENCE

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ABSTRACT: Senescence is the final stage of organ development in plants. It is an oxidative process which involves a general deterioration of cellular metabolism in plants. The symptoms of senescence include loss in chlorophyll, carotenoids, proteins and increase in lipid peroxidation and membrane permeability which results in injury and leads to decrease in the photosynthetic output. During the period of senescence, a multiple factors such as hormones, environmental factors and senescence associated genes (SAGs) are involved. Plant hormones or phytohormones acts as chemical messengers which are involved in plant growth and developmental process and play a major role in regulation of senescence. The phytohormones such as salicylic acid, abscisic acid, jasmonic acid and ethylene promote senescence, where as others like cytokinins, gibberellins, and auxins delay this process. During the process of senescence generation of reactive oxygen species (ROS) which includes superoxide, singlet oxygen, hydroxyl radical and hydrogen peroxide radicals increased in thylakoids takes place. These free radicals are capable of inducing cellular damage by oxidation of proteins, inactivation of enzymes, alterations in the gene expression, and decomposition of biomembranes. Thus, plants have evolved a defense mechanism which scavange ROS by antioxidant enzymes such as superoxide dismutase (SOD) and catalase (CAT). Thus in the present review all factors that are responsible for causing or accelerating senescence have discussed, antioxidant activity which protects plants from ROS and role of phytohormones such as auxins, gibberillic acid and cytokinins have participated significantly in delaying or to control senescence have been discussed.
Key words: Antioxidant enzymes, Chlorophyll degradation, Lipid peroxidation, Phytohormones, ROS, Senescence.

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INTRODUCTION

Senescence is a genetically regulated process which is characterized by a network of molecular and biochemical mechanisms. It is a synchronized series of events which occur during the course of plant development, but any type of stress in plants may bring about the process of senescence [1]. In plants, senescence is a complex deterioration process which can lead to the death of whole organism or a single organ and is highly regulated by various factors such as age, reproductive development, and phytohormone levels and by environmental signals, including photoperiod, stresses such as drought, ozone, nutrient deficiency, wounding, and shading [2]. Senescence in leaf is the final step in the development of leaf and is characterized by degradation of biomolecules. Senescence is a synchronized and sequential process in which first undergoes the degeneration of chloroplast which is followed by the hydrolysis and remobilization of macromolecules to other plant parts and disintegration of nucleus as well as mitochondrian takes place [3].

It is a deteriorative process which includes the loss of chlorophyll (Chl), decrease in the total RNA and protein contents [4]. The process of degradation of chloroplast involves the disorientation of granum stacks and swelling of the thylakoids that results in the enlargement of plastoglobuli [7]. The most evident events triggered during senescence are the breakdown of chlorophyll, protein and oxidative stress [5,6]. The generation of ROS is one of the earliest responses of plant cells under abiotic stresses [9] and senescence which play a significant role in stress signaling [8]. The formation of ROS which generally causes oxidative damage to DNA, proteins and membrane lipids and also play an important role in cellular signalling pathways in plants [10].

Thus, plants have evolved some defense mechanisms for the protection against ROS production by the process of acclimation which involves decreased production of ROS coupled with an efficient antioxidant defense mechanism [11] and the activation of different signaling pathways [12,13]. Antioxidant enzymes such as superoxide dismutase, ascorbate peroxidase, glutathione reductase, peroxidise and catalase are involved in the scavenging of reactive oxygen species. Plant hormones are also known as phytohormones which play a critical role in plant developmental processes and play a significant role in the regulation of leaf senescence [14]. Phytohormones like auxins, gibberillins and cytokinins delays the process of senescence where as ethylene and abscisic acid that promote senescence [15]. Various factors like phytohormones and microRNAs participated significantly to control senescence under non-stress and stress conditions have also been discussed. (Fig: 1)

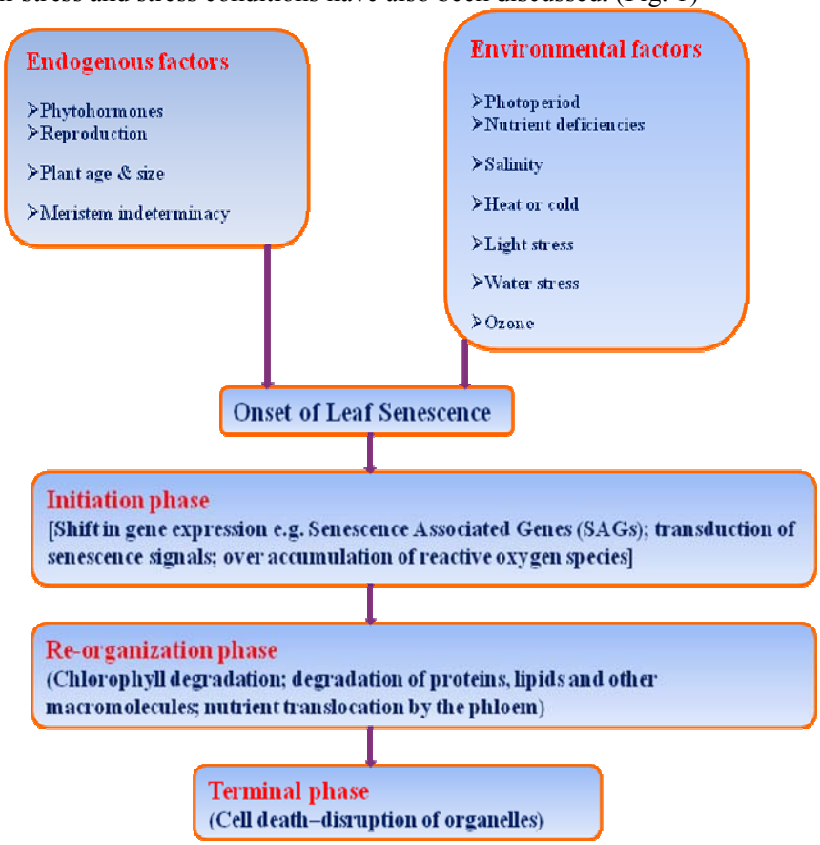

Fig. 1: Different factors involved in senescence

Biochemical changes during senescence:

(a) Photosynthetic pigments:

Photosynthetic pigments such as Chl *a*, Chl *b*, carotenoids and xanthophylls decreased during leaf senescence. In higher plants, studies reveal that a greater loss of chlorophylls than carotenoids, that results in yellow colouration of senescing leaves.

(i) Chlorophyll degradation:

The chloroplast is the first organelle in the green cell to show signs of decay, that is characterized by the dissociation of grana, increase in size and number of plastaglobuli and disruption of the chloroplast envelope [16]. Some reasearchers termed the degrading chloroplast as gerontoplast [17]. Chloroplast degradation includes the progressive loss of proteins in the chloroplast, such as ribulose biphosphate carboxylase (Rubisco) and Chl *a/b* binding protein (CAB) which is caused by the hydrolysis of proteins to free amino acids by the action of several endo and exopeptidases [18]. The Chloroplast degeneration is accompanied by chlorophyll degradation which is one of the most earliest symptoms of senescence.

The Chl degradation is one of the potential indicators of leaf senescence, any effect in degradation of chlorophyll can be directly related to leaf senescence. The breakdown of chlorophyll leads to the yellowing of blade, this is the most visible symptom of chlorophyll breakdown during senescence [19]. There is a pathway for chlorophyll degradation which consists of several reaction steps catalysed by enzymes [20].

In the process of chlorophyll degradation, removal of phytol by chlorophyllase enzyme is the first step involved and is the first enzyme in this pathway [21]. The second step involved in this process is the removal of magnesium from chlorophyllide by Mg-dechelatase. Oxygenolytic opening of porphyrin macrocycle in phaeophorbide, that produces fluorescenct chlorophyll catabolites (FCCs). This step is catalysed by an enzyme phaeophorbide a oxygenase which required oxygen, ATP, ferrodoxin, NADPH, iron, and thylakoid and stromal proteins [22]. The last step involves the catabolism of FCCs to non-fluorescent chlorophyll catabolites (NCCs) and the disposal of NCCs in vacuoles. During the degradation of chlorophyll the breakdown products of both chl *a* as well as chl *b* are derived as the final catabolites[23]. And these degraded products and the chlorophyll are transferred to the cell vacuole [24].

(ii) Carotenoid catabolism:

Carotenoids play a significant role in the assembly of light-harvesting complex as antenna pigments, and protect the photosynthetic apparatus from photo-oxidative damage. The carotenoid catabolism has been studied during fruit ripening and in flowers [25]. There is a slower degradation of carotenoids than Chls, which results in unmasked carotenoids. In gymnosperms and angiospermic plants a relative stability of carotenoids has been demonstrated during leaf senescence. A decline has shown in the rates of both the chlorophyll and carotenoid pigments was observed in ferns, tobacco and barley leaves during senescence. During naturally occurring and artificially-induced senescence, formation of plastoglobuli has been observed and the structures of these are demonstrated to contain thylakoid breakdown products which includes carotenoid esters and free carotenoids such as β-carotene and xanthophylls [26]. There is an increased free fatty acids which are released from triacyl glycerols by the action of hydrolysing lipases, and free carotenoids released from thylakoids that are esterified by these fatty acids, and their deposition in plastoglobuli has been observed during the advanced stages of senescence [27].

(b) Alterations in electron transport chain:

During leaf senescence many alterations in the structure and function of chloroplasts has occured [28,29]. The alterations include the degradation of photosynthetic pigments and proteins, inactivation of both photosystems (PS) I and II, and down regulation of enzyme activities associated with Calvin-Benson cycle [30]. Reports demonstrating the damage of PS II, with oxygen evolving complex (OEC) as one of the initial events during leaf senescence [31]. During senescence the disassembly of photosynthetic complexes which results in the differentiation of chloroplasts into gerontoplasts that causes the changes in stoichometries of photosynthetic complexes [32]. This changes may lead to an imbalances in the photosynthetic electron chain responsible for an enhanced production of ROS and the enhanced risk of photoinhibition. There will be the reduction in ability of photosynthetic CO2 fixation have been reported due to the loss in the total chlorophyll and protein content as well as the disorganization of the structure of chloroplast membranes during senescence. This loss of function in the structure of chloroplast may be associated with the decline in the photochemical activities of PS I and PS II there is more susceptible PS II to senescence than PS I and a greater decrease in PS II activity has been observed during senescence of leaves. The cause for this decreased PS II electron transport is not clear but studies have shown that the decrease in PS II activity is due to an inactivation of the oxygen evolution system, or a loss of PS II reaction centre complexes [33], or an inhibition of excitation energy transfer from carotenoids to chlorophyll. Thus during senescence, several studies have investigated the changes in the maximal efficiency of PS II photochemistry *(Fv/Fm) in vivo* on whole leaves.

(c) Lipid peroxidaition during senescence

Advanced senescence causes a notable increase in lipid peroxidation [34] which leads to the generation of free radicals, that can lead to the promotion of senescence [35]. Lipid peroxidation is an indicator of prevalence of free radicals in tissues. The senescence causes an enhancement in the lipid perixidation in chloroplast [36] which clearly indicates the production of ROS inside the organelle is enhanced. During senescence lipid peroxidation causes the production of variety of toxic aldehydes and ketones [37]. Such products of lipid peroxidation are malondialdehyde and 4-hydroxynonenal which cause protein damage by means of reactions with lysine amino groups, cysteine sulfhydryl groups and histidine imidazole groups [38]. The lipid peroxidation causes a chemical change in the membrane lipids which results in an increase in the ratio of sterol/phospholipid however, the sterol content declines with physiological aging.

Production of ROS and protection against reactive oxygen species during senescence

The initiation of senescence involved in the production of ROS [39]. The enzymes for ROS-production were identified, such as NADPH oxidases, aminooxidases and cell wall-bound peroxidises. During senescence the levels of ROS are increased that have been reported to coincide with increased levels of lipid peroxides [40] and oxidized proteins [41].

The photosynthetic electron transport chain might become another major source for ROS such as singlet oxygen ($^1O_2$), superoxide anion radicals ($O2^-$) and also hydrogen peroxide [42] during senescence. These are called reactive oxygen species (ROS) which are extremely reactive and are able to oxidize biological molecules, such as DNA, proteins or lipids. Several studies have reported an increase in the production of $O^{\bullet}{}_2^-$ during the natural and artificially induced senescence [43].

(i) Singlet oxygen ($^1O_2$)

Singlet oxygen is the highly reactive and is generated when chlorophyll is excited and form triplet state (^{3}Chl) which reacts with the molecular oxygen that is a triplet in its ground state ($^3O^2$). The triplet state of chlorophyll is generated when there is damage in the reaction centre of photosystem II [44] by charge recombination reactions [45] and in the light harvesting complex II (LHC II). The generation of $^1O_2$ during photosynthesis is caused by the photoactivation of photosensitizers, mainly chlorophylls and their precursors [46] and also during senescence [47] and under abiotic stresses [48]. Singlet oxygen has dual effect which act as an oxidising agent and can react with various biological molecules, causing damage and leading to cell death [49].

(ii) Superoxide Anion and Hydrogen peroxide:

Superoxide anions $O^{\bullet}2^-$ are generated in different plant cell organelles which include chloroplasts, peroxisomes, apoplast, the mitochondrial electron transport chain, and the plasma membrane [50]. In peroxisomes, there are two different sources for the generation of superoxide anion: one is by electron transport chain (ETC) in peroxisomal membrane [51] and in peroxisomal matrix via action of enzyme xanthine oxidase [52]. This generated superoxide anion ($O^{\bullet}2^-$) is converted to hydrogen peroxide (H2O2), by the action of CuZn-superoxide dismutase [53].

Hydrogen peroxide plays an important role under stress conditions as a signaling molecule which mediates between different physiological processes [54] i.e., involved in the regulation of the senescence process [55] protection against pathogen attack [56]. When compared with other ROS, H_2O_2 is the most stable and least reactive ROS which can easily cross the membrane [57] and hence acts as a good signaling molecule. The production of H_2O_2 in plants involved via two possible pathways: dismutation of $O^{\bullet}{}_2^-$ with the involvement of SOD [53], and via oxidases such as amino and oxalate oxidases. In plants, H_2O_2 by acting as a signaling molecule which is involved in the regulation of various abiotic and biotic stresses [54] and has a role in cell death which contributes to cell degradation [58] during the final stages of senescence.

Protection against ROS by antioxidant enzymes:

Plants have evolvled some defense mechanisms against ROS which include superoxide dismutase, ascorbate peroxidase (APX) and catalase [59]. The induction of these cellular antioxidant machinery is important for protection against various stresses [60]. The antioxidant defense system include both enzymatic and non-enzymatic antioxidants. The SOD and CAT are included under enzymatic antioxidants and GSH, AA (both water soluble), carotenoids and tocopherols (lipid soluble) [61] are included under non-enzymatic antioxidants.

(i) Catalase

CATs are tetrameric heme containing enzymes which directly involves the dismutation of H_2O_2 into H_2O and O_2 and also involves in the detoxification of ROS during stress conditions. In higher plants, the isozymes of CAT have been extensively studied. Maize has three isoforms (CAT 1, CAT 2 and CAT 3), found on separate chromosomes and are differentially expressed and independently regulated [62]. Catalase regulates the intracellular H_2O_2 [63]. The down-regulation of CAT 2 expression during leaf senescence has occurred and an increased level of H_2O_2 has been postulated [64].

(ii) SOD:

Superoxide dismutases (SOD; EC 1.15.1.1) are a class of metalloenzymes and ubiquitous in all oxygen-consuming organisms which catalyze the dismutation of superoxide radicals (O_2^-) to molecular oxygen and hydrogen peroxide, the substrate of catalases and peroxidises.

$$2\ O_2- + 2\ H^+ \rightarrow H_2O_2 + O_2$$

Superoxide dismutases can also be divided into three classes [65] in which the first class of SODs are located in the cytosol, chloroplasts and mitochondria of eukaryotic cells and having Cu^{2+} and Zn^{2+} at the active binding site. The class II SODs uses Mn^{2+} as a cofactor. The manganese-dependent SODs are found in prokaryotes and in the mitochondria of eukaryotes and have a speculated peroxisomal and cytosolic location for MnSODs [66]. The third class of SODs have Fe^{3+} at the active site and are found in prokaryotes and mainly in the chloroplasts of some plants.

Involvement of phytohormones in the regulation of senescence

The senescence programme is the final developmental phase of a leaf which can be influenced by several phytohormones. These are also called plant hormones which are chemically plant growth substances that regulate various aspects of plant growth & development. The hormones include auxins, gibberellins (GAs), cytokinins, abscisic acid, brassinosteroids & jasmonic acid(JA). Plant hormones such as ethylene, abscisic acid, salicylic acid, and jasmonic acid promote senescence, whereas auxin, gibberellic acids (GAs), and cytokinins retard it [67].

(i) Auxins

Auxins are a group of phytohormones which play a significant role in the plant growth and development. These are involved in various developmental processes such as apical dominance, embryonic and postembryonic patterning, vascular differentiation, root and shoot development, branching, tropisms and flowering. Auxins mainly represented by Indole Acetic Acid (IAA) and indole butyric acid, which are synthesized from tryptophan via two different pathways [68]. A distinct feature of auxin is the polar auxin transport by auxin efflux carriers with polar distribution in cells. Auxins referred to as plant developmental hormone which play a significant role in senescence [69]. Several studies have suggested the correlation between auxin levels with senescence and abscission. In plants, auxin may retard [70] or accelerate senescence. The levels of auxins were declined as leaf aged and senescence occurred when the levels of auxins between the leaf and stalk were approximately equal [70]. Exogenous application of auxins were reported to cause down-regulation of some SAGs [71]. Thus, auxins are contemplated as negative regulators of leaf senescence. Genetic mutants which are defective in auxin signalling may reinforces their role in leaf senescence [72]. For example, a transcription repressor of auxin signaling pathway, auxin response factor 2 (ARF2), is up-regulated during leaf senescence [70]. It has been reported the T-DNA mutants of ARF2 cause delay in leaf senescence, suggesting their role as positive regulators in the process [73]. Because of these reports auxins are effective in regulation of leaf senescence.

(ii) Gibberillins

Gibberellic acid is a pentacyclic diterpene which is involved in plant development processes such as cell elongation, seed germination, dormancy, reproductive growth, senescence and tolerance against various environmental stresses [74]. Gibberillins play a significant role in senescence by acting as a senescence-retarding hormone [75] whose active form declines in leaves as they age. The potential indicator of leaf senescence is the chlorophyll degradation, so effect on chlorophyll degradation can be directly related to leaf senescence that was considered to be associated with the endogeneous endogenous gibberellin content in shoots of *Paris polyphylla* during senescence[76]. It has been reported that the exogenous application of gibberillinsin in various plant species may result in delayed senescence. For example, plant species in which GA3 delays senescence include *lettuce*, *Rumex crispus* and *R. obtusifolius*, *Catharanthus roseus* [77], *nasturtium* (*Tropaeolum majus*) [78], and *Dioscorea rotundata* [79]. When a study with perennial plant P. polyphylla [76] it has shown that exogenous application with gibberellic acid (GA3) caused an enhancement in the level of internal GAs (GA4+GA7) which results in retardation of shoot senescence, that was attributed primarily to the suppression of degradation of proteins and chlorophyll as well as lipoxygenase activity [76]. It was proposed that GA antagonizes the effects of ABA by inhibiting leaf senescence in *Paris polyphylla* [80,]. Senescence process is inhibited by the availability of free GA (GA4 and GA7) [81]. Mutations in the F-box protein (SLY1), result in a block of GA-responsive genes, delay senescence when crossed to *abi1* [82].

(iii) Cytokinins

Cytokinins (CKs) are well-studied group of plant hormones, which are adenine derivatives with isoprenoid or aromatic side chains. In higher plants, isopentenyladenine and zeatin are the most abundant CKs. Cytokinins are involved in regulating various plant growth and developmental processes [83,84] and also function in adaptation to stress [85]. They play major roles in diverse developmental processes including shoot meristem initiation, leaf and root differentiation, vascular patterning, seed development, photomorphogenesis and gravitropism.

Like other plant hormones have their role in senescence, cytokinins receive the maximum attention in delaying leaf senescence [86].

The high levels of cellular cytokinins may delay leaf senescence [86]. During leaf senescence, certain studies [87] have demonstrated the down-regulation of cytokinin biosynthesis genes such as adenosine phosphate isopentenyl-transferase (IPT) and cytokinin synthase as well as up-regulation of cytokinin oxidation genes (cytokinin oxidase). During leaf senescence, the expression of cytokinin oxidase, an enzyme involved in CK degradation, is increased which results in the repression of signaling and transcription of genes involved in CK biosynthesis takes place [88]. IPT (isopentyl-transferase), an enzyme that catalyzes the rate-limiting step in CK biosynthesis, this is an evidence provided for a function of CKs in senescence. Cytokinins regulate senescence by controlling the activity of extracellular invertase [89].

The content of cytokinin decreases in senescing leaves and this has been proposed as one of the key signals for initiating senescence. Hexokinase (HXK), is an enzyme which controls the cytokinin-mediated senescence which induces senescence when over expressed [90]. The inhibition of leaf senescence by cytokinin has been observed when its biosynthesis genes are driven by the SAG 12 promoter. Cytokinins also mediate their action by modulating a number of transcription factor genes like GATA 22, HAT 4, HAT 22 and bHLH 64 [91,92]. For example, over expression of HAT 22 has been shown to cause reduction in chlorophyll content of seedlings and an early onset of leaf senescence in these plants [91].

Future prospects

Hormonal regulation of senescence is very important as it involves a complex interplay of signaling molecules at various stages of senescence. Recent progress in elucidating molecular events of phytohormone action in senescence just begun to clarify the composition of the regulatory pathways involved. However, the exact mechanism how hormones delay or hasten the process of plant senescence is not yet well known. Therefore, there is still a need to study the role of hormones in senescence at the physiological and molecular levels. It could be helpful in preventing loss during the storage of fruits, legumes, flowers and crops. Certainly future research on senescence will be an amalgamation of bioinformatical predictions, hormonal regulation of senescence that will unravel the mechanism underlying leaf aging.

REFERENCES

[1] Lobell, D. B., Sibley, A., & Ortiz-Monasterio, J. I. 2012. Extreme heat effects on wheat senescence in India. Nature Climate Change, 2(3), 186-189.

[2] Gan, S., & Amasino, R. M. 1997. Making sense of senescence (molecular genetic regulation and manipulation of leaf senescence). Plant Physiology, 113(2), 313.

[3] Wang, W. 2012. Regulatory RNA-binding proteins in senescence. *Ageing research reviews*, 11(4), 485-490.

[4] Diaz, C., Purdy, S., Christ, A., Morot-Gaudry, J. F., Wingler, A., & Masclaux-Daubresse, C. 2005. Characterization of markers to determine the extent and variability of leaf senescence in Arabidopsis. A metabolic profiling approach. Plant Physiology, 138(2), 898-908.

[5] Khanna-Chopra, R. 2012. Leaf senescence and abiotic stresses share reactive oxygen species-mediated chloroplast degradation. Protoplasma, 249(3), 469-481.

[6] Qian, C. L., Zhao, Y. Y., Mi, H. B., Chen, X. H., Guo, L. J., & Mao, L. C. 2012. Role of antioxidative system during the development and senescence of cucumber fruit. Biologia Plantarum, 56(4), 793-797.

[7] Zentgraf, U., & Hemleben, V. 2008. Molecular cell biology: are reactive oxygen species regulators of leaf senescence? In *Progress in botany* (pp. 117-138). Springer Berlin Heidelberg.

[8] Lee, I. C., Hong, S. W., Whang, S. S., Lim, P. O., Nam, H. G., & Koo, J. C. 2011. Age-dependent action of an ABA-inducible receptor kinase, RPK1, as a positive regulator of senescence in Arabidopsis leaves. Plant and Cell physiology, 52(4), 651-662.

[9] Ahmad, P., Sarwat, M., & Sharma, S. 2008. Reactive oxygen species, antioxidants and signaling in plants. Journal of Plant Biology, 51(3), 167-173.

[10] Ramel, F., Birtic, S., Ginies, C., Soubigou-Taconnat, L., Triantaphylidès, C., & Havaux, M. 2012. Carotenoid oxidation products are stress signals that mediate gene responses to singlet oxygen in plants. Proceedings of the National Academy of Sciences, 109(14), 5535-5540.

[11] Khanna-Chopra, R., & Selote, D. S. 2007. Acclimation to drought stress generates oxidative stress tolerance in drought-resistant than-susceptible wheat cultivar under field conditions. Environmental and Experimental Botany, 60(2), 276-283.

[12] Wang, L., Su, H., Han, L., Wang, C., Sun, Y., & Liu, F. 2014. Differential expression profiles of poplar MAP kinase kinases in response to abiotic stresses and plant hormones, and over expression of PtMKK4 improves the drought tolerance of poplar. Gene, 545(1), 141-148.

[13] Suzuki, N., Koussevitzky, S., Mittler, R. O. N., & Miller, G. A. D. 2012. ROS and redox signalling in the response of plants to abiotic stress. Plant, Cell & Environment, 35(2), 259-270.
[14] Schippers, J. H., Jing, H. C., Hille, J., & Dijkwel, P. P. 2007. Developmental and hormonal control of leaf senescence. Senescence processes in plants, 145-170.
[15] Häffner, E., Konietzki, S., & Diederichsen, E. 2015. Keeping Control: The Role of Senescence and Development in Plant Pathogenesis and Defense. Plants, 4(3), 449-488.
[16] Zavaleta-Mancera, H. A., Franklin, K. A., Ougham, H. J., Thomas, H., & Scott, I. M. 1999. Regreening of senescent Nicotiana leaves I. Reappearance of NADPH-protochlorophyllide oxidoreductase and light-harvesting chlorophyll a/b-binding protein. Journal of Experimental Botany, 50(340), 1677-1682.
[17] Van Doorn, W. G., & Yoshimoto, K. 2010. Role of chloroplasts and other plastids in ageing and death of plants and animals: A tale of Vishnu and Shiva. Ageing research reviews, 9(2), 117-130.
[18] Xie, Q., Michaeli, S., Peled-Zehavi, H., & Galili, G. 2015. Chloroplast degradation: one organelle, multiple degradation pathways. Trends in plant science, 20(5), 264-265.
[19] Sakuraba, Y., Han, S. H., Lee, S. H., Hörtensteiner, S., & Paek, N. C. 2015. Arabidopsis NAC016 promotes chlorophyll breakdown by directly upregulating STAYGREEN1 transcription. Plant cell reports, 1-12.
[20] Takamiya, K. I., Tsuchiya, T., & Ohta, H. 2000. Degradation pathway (s) of chlorophyll: what has gene cloning revealed? Trends in plant science, 5(10), 426-431.
[21] Matile, P., Schellenberg, M., & Vicentini, F. 1997. Localization of chlorophyllase in the chloroplast envelope. Planta, 201(1), 96-99.
[22] Hörtensteiner, S., & Kräutler, B. 2011. Chlorophyll breakdown in higher plants.Biochimica et Biophysica Acta (BBA)-Bioenergetics, 1807(8), 977-988.
[23] Christ, B., & Hörtensteiner, S. 2014. Mechanism and significance of chlorophyll breakdown. Journal of plant growth regulation, 33(1), 4-20.
[24] Sarwat, M., Naqvi, A. R., Ahmad, P., Ashraf, M., & Akram, N. A. 2013. Phytohormones and microRNAs as sensors and regulators of leaf senescence: assigning macro roles to small molecules. Biotechnology advances, 31(8), 1153-1171.
[25] Hirschberg, J. 2001. Carotenoid biosynthesis in flowering plants. Current opinion in plant biology, 4(3), 210-218.
[26] Biswal, U. C., & Biswal, B. 1988. Ultrastructural modifications and biochemical changes during senescence of chloroplasts. Int. Rev. Cytol, 113, 271-321.
[27] Tevini, M., & Steinmüller, D. 1985. Composition and function of plastoglobuli. Planta, 163(1), 91-96.
[28] Biswal, B., Mohapatra, P. K., Biswal, U. C., & Raval, M. K. 2012. Leaf senescence and transformation of chloroplasts to gerontoplasts. In Photosynthesis (pp. 217-230). Springer Netherlands.
[29] Khanna-Chopra, R. 2012. Leaf senescence and abiotic stresses share reactive oxygen species-mediated chloroplast degradation. Protoplasma, 249(3), 469-481.
[30] Martínez, D. E., Costa, M. L., & Guiamet, J. J. 2008. Senescence-associated degradation of chloroplast proteins inside and outside the organelle. Plant Biology, 10(s1), 15-22.
[31] Prakash, J. S., Baig, M. A., & Mohanty, P. 2001. Differential changes in the steady state levels of thylakoid membrane proteins during senescence in Cucumis sativus cotyledons. Zeitschrift für Naturforschung C, 56(7-8), 585-592.
[32] Mulisch, M., & Krupinska, K. 2013. Ultrastructural analyses of senescence associated dismantling of chloroplasts revisited. In *Plastid Development In Leaves During Growth and Senescence* (pp. 307-335). Springer Netherlands.
[33] Kura-Hotta, M., Satoh, K., & Katoh, S. 1987. Relationship between photosynthesis and chlorophyll content during leaf senescence of rice seedlings. Plant and Cell Physiology, 28(7), 1321-1329.
[34] Zhang, M. P., Zhang, C. J., Yu, G. H., Jiang, Y. Z., Strasser, R. J., Yuan, Z. Y., & Chen, G. X. 2010. Changes in chloroplast ultrastructure, fatty acid components of thylakoid membrane and chlorophyll a fluorescence transient in flag leaves of asuper-high-yield hybrid rice and its parents during the reproductive stage. Journal of plant physiology, 167(4), 277-285.
[35] Arora, A., Sairam, R. K., & Srivastava, G. C. 2002. Oxidative stress and antioxidative system in plants. Current science-Banglore-, 82(10), 1227-1238.
[36] Corpas, F. J., Gupta, D. K., & Palma, J. M. 2015. Production sites of reactive oxygen species (ROS) in organelles from plant cells. In *Reactive Oxygen Species and Oxidative Damage in Plants Under Stress* (pp. 1-22). Springer International Publishing.
[37] Wilhelmova, N., Domingues, P. M. D. N., Srbova, M., Fuksova, H., & Wilhelm, J. 2006. Changes in nonpolar aldehydes in bean cotyledons during ageing. Biologia plantarum, 50(4), 559-564.

[38] Refsgaard, H. H., Tsai, L., & Stadtman, E. R. 2000. Modifications of proteins by polyunsaturated fatty acid peroxidation products. Proceedings of the National Academy of Sciences, 97(2), 611-616.
[39] Juvany, M., Müller, M., & Munné-Bosch, S. 2013. Photo-oxidative stress in emerging and senescing leaves: a mirror image? Journal of experimental botany, ert174.
[40] Dhindsa, R. S., Plumb-Dhindsa, P. L., & Reid, D. M. 1982. Leaf senescence and lipid peroxidation: Effects of some phytohormones, and scavengers of free radicals and singlet oxygen. Physiologia Plantarum, 56(4), 453-457.
[41] Vanacker, H., Sandalio, L. M., Jiménez, A., Palma, J. M., Corpas, F. J., Meseguer, V., & Del Río, L. A. 2006. Roles for redox regulation in leaf senescence of pea plants grown on different sources of nitrogen nutrition. Journal of Experimental Botany, 57(8), 1735-1745.
[42] Apel, K., & Hirt, H. 2004. Reactive oxygen species: metabolism, oxidative stress, and signal transduction. Annu. Rev. Plant Biol, 55, 373-399.
[43] Krieger-Liszkay, A., Trösch, M., & Krupinska, K. 2015. Generation of reactive oxygen species in thylakoids from senescing flag leaves of the barley varieties Lomerit and Carina. Planta, 241(6), 1497-1508.
[44] Fischer, B. B., Hideg, É., & Krieger-Liszkay, A. 2013. Production, detection, and signaling of singlet oxygen in photosynthetic organisms. Antioxidants & redox signaling, 18(16), 2145-2162.
[45] Rutherford, A. W., & Krieger-Liszkay, A. 2001. Herbicide-induced oxidative stress in photosystem II. Trends in biochemical sciences, 26(11), 648-653.
[46] Krieger-Liszkay, A. 2005. Singlet oxygen production in photosynthesis. Journal of Experimental Botany, 56(411), 337-346.
[17] Springer, A., Acker, G, Bartsch, S., Bauerschmitt, H., Reinbothe, S., & Reinbothe, C. 2015. Differences in gene expression between natural and artificially induced leaf senescence in barley. Journal of plant physiology, 176, 180-191.
[48] Alboresi, A., Dall'Osto, L., Aprile, A, Carillo, P., Roncaglia, E., Cattivelli, L., & Bassi, R. 2011. Reactive oxygen species and transcript analysis upon excess light treatment in wild-type Arabidopsis thaliana vs a photosensitive mutant lacking zeaxanthin and lutein. BMC plant biology, 11(1), 62.
[49] Op den Camp, R. G., Przybyla, D., Ochsenbein, C., Laloi, C., Kim, C., Danon, A., & Nater, M. 2003. Rapid induction of distinct stress responses after the release of singlet oxygen in Arabidopsis. The Plant Cell, 15(10), 2320-2332.
[50] Rosenwasser, S., Rot, I., Sollner, E., Meyer, A. J., Smith, Y., Leviatan, N., & Friedman, H. 2011. Organelles contribute differentially to reactive oxygen species-related events during extended darkness. Plant physiology, 156(1), 185-201.
[51] Jajic, I., Sarna, T., & Strzalka, K. 2015. Senescence, Stress, and Reactive Oxygen Species. Plants, 4(3), 393-411.
[52] Corpas, F. J., Palma, J. M., Sandalio, L. M., Valderrama, R., Barroso, J. B., & Luis, A. 2008. Peroxisomal xanthine oxidoreductase: characterization of the enzyme from pea (Pisum sativum L.) leaves. Journal of plant physiology, 165(13), 1319-1330.
[53] Asada, K. 2006. Production and scavenging of reactive oxygen species in chloroplasts and their functions. Plant physiology, 141(2), 391-396.
[54] Quan, L. J., Zhang, B., Shi, W. W., & Li, H. Y. 2008. Hydrogen peroxide in plants: a versatile molecule of the reactive oxygen species network. Journal of Integrative Plant Biology, 50(1), 2-18.
[55] Peng, L. T., Jiang, Y. M., Yang, S. Z., & Pan, S. Y. 2005. [Accelerated senescence of fresh-cut Chinese water chestnut tissues in relation to hydrogen peroxide accumulation]. *Zhi wu sheng li yu fen zi sheng wu xue xue bao*= Journal of plant physiology and molecular biology, 31(5), 527-532.
[56] Kumar, N., Ebel, R. C., & Roberts, P. D. 2011. Antioxidant metabolism of grapefruit infected with Xanthomonas axonopodis pv. citri. Environmental and Experimental Botany, 71(1), 41-49.
[57] Yang, T., & Poovaiah, B. W. 2002. Hydrogen peroxide homeostasis: activation of plant catalase by calcium/calmodulin. *Proceedings of the National Academy of Sciences*, 99(6), 4097-4102.
[58] Zimmermann, P., Heinlein, C., Orendi, G., & Zentgraf, U. 2006. Senescence-specific regulation of catalases in Arabidopsis thaliana (L.) Heynh. Plant, cell & environment, 29(6), 1049-1060.
[59] Singh, S., Anjum, N. A., Khan, N. A., & Nazar, R. 2008. Metal-binding peptides and antioxidant defence system in plants: significance in cadmium tolerance. *Abiotic stress and plant responses. IK International, New Delhi*, 159-189.
[60] Mittler, R., Vanderauwera, S., Gollery, M., & Van Breusegem, F. 2004. Reactive oxygen gene network of plants. Trends in plant science, 9(10), 490-498.
[61] Garg, N., & Manchanda, G. 2009. ROS generation in plants: boon or bane?.Plant Biosystems, 143(1), 81-96.

[62] Koyro, H. W., Ahmad, P., & Geissler, N. 2012. Abiotic stress responses in plants: an overview. In *Environmental adaptations and stress tolerance of plants in the era of climate change* (pp. 1-28). Springer New York.

[63] Smykowski, A., Zimmermann, P., & Zentgraf, U. 2010. G-Box binding factor1 reduces CATALASE2 expression and regulates the onset of leaf senescence in Arabidopsis. Plant physiology, 153(3), 1321-1331.

[64] Causin, H. F., Marchetti, C. F., Pena, L. B., Gallego, S. M., & Barneix, A. J. 2015. Down-regulation of catalase activity contributes to senescence induction in wheat leaves exposed to shading stress. Biologia Plantarum, 59(1), 154-162.

[65] Gill, S. S., Anjum, N. A., Gill, R., Yadav, S., Hasanuzzaman, M., Fujita, M., & Tuteja, N. 2015. Superoxide dismutase—mentor of abiotic stress tolerance in crop plants. Environmental Science and Pollution Research, 1-20.

[66] Corpas, F. J., Gupta, D. K., & Palma, J. M. 2015. Production sites of reactive oxygen species (ROS) in organelles from plant cells. In *Reactive Oxygen Species and Oxidative Damage in Plants Under Stress* (pp. 1-22). Springer International Publishing.

[67] Sarwat, M., Naqvi, A. R., Ahmad, P., Ashraf, M., & Akram, N. A. 2013. Phytohormones and microRNAs as sensors and regulators of leaf senescence: assigning macro roles to small molecules. Biotechnology advances, 31(8), 1153-1171.

[68] Cohen, J. D., Slovin, J. P., & Hendrickson, A. M. 2003. Two genetically discrete pathways convert tryptophan to auxin: more redundancy in auxin biosynthesis. Trends in plant science, 8(5), 197-199.

[69] Im Kim, J., Murphy, A. S., Baek, D., Lee, S. W., Yun, D. J., Bressan, R. A., & Narasimhan, M. L. 2011. YUCCA6 over-expression demonstrates auxin function in delaying leaf senescence in Arabidopsis thaliana. Journal of experimental botany, err094.

[70] Ellis, C. M., Nagpal, P., Young, J. C., Hagen, G., Guilfoyle, T. J., & Reed, J. W. 2005. AUXIN RESPONSE FACTOR1 and AUXIN RESPONSE FACTOR2 regulate senescence and floral organ abscission in Arabidopsis thaliana. Development, 132(20), 4563-4574.

[71] Jones, B., Gunneras, S. A., Petersson, S. V., Tarkowski, P., Graham, N., May, S., & Ljung, K. 2010. Cytokinin regulation of auxin synthesis in Arabidopsis involves a homeostatic feedback loop regulated via auxin and cytokinin signal transduction. The Plant Cell, 22(9), 2956-2969.

[72] Lim PO, Lee IC, Kim J, Kim HJ, Ryu JS, Woo HR.2010. Auxin response factor 2 (ARF2) plays a major role in regulating auxin-mediated leaf longevity. J Exp Bot ;61:1419-30.

[73] Xu, F., Meng, T., Li, P., Yu, Y., Cui, Y., Wang, Y., & Wang, N. N. 2011. A soybean dual-specificity kinase, GmSARK, and its Arabidopsis homolog, AtSARK, regulate leaf senescence through synergistic actions of auxin and ethylene. Plant physiology, 157(4), 2131-2153.

[74] Rodrigues, C., Vandenberghe, L. P. D. S., de Oliveira, J., & Soccol, C. R. 2012. New perspectives of gibberellic acid production: a review. Critical reviews in biotechnology, 32(3), 263-273.

[75] Schippers, J. H., Jing, H. C., Hille, J., & Dijkwel, P. P. 2007. Developmental and hormonal control of leaf senescence. Senescence processes in plants, 145-170.

[76] Li, J. R., Yu, K., Wei, J. R., Ma, Q., Wang, B. Q., & Yu, D. 2010. Gibberellin retards chlorophyll degradation during senescence of Paris polyphylla. Biologia plantarum, 54(2), 395-399.

[77] Pandey, S., Gupta, K., & Mukherjee, A. K. 2007. Impact of cadmium and lead on Catharanthus roseus-A phytoremediation study. Journal of Environmental Biology, 28(3), 655.

[78] Beevers, L. 1966. Effect of gibberellic acid on the senescence of leaf discs of nasturtium (Tropaeolum majus). Plant physiology, 41(6), 1074.

[79] Ile, E. I., Craufurd, P. Q., Battey, N. H., & Asiedu, R. 2006. Phases of dormancy in yam tubers (Dioscorea rotundata). Annals of botany, 97(4), 497-504.

[80] Yu, K., Wei, J., Ma, Q., Yu, D., & Li, J. 2009. Senescence of aerial parts is impeded by exogenous gibberellic acid in herbaceous perennial Paris polyphylla. Journal of plant physiology, 166(8), 819-830.

[81] Yu, K., Wang, Y., Wei, J., Ma, Q., Yu, D., & Li, J. 2009. Improving rhizome yield and quality of Paris polyphylla through gibberellic acid-induced retardation of senescence of aerial parts. Plant signaling & behavior, 4(5), 413-415.

[82] Richards, D. E., King, K. E., Ait-ali, T., & Harberd, N. P. 2001. How gibberellin regulates plant growth and development: a molecular genetic analysis of gibberellin signaling. Annual review of plant biology, 52(1), 67-88.

[83] Argueso, C. T., Raines, T., & Kieber, J. J. 2010. Cytokinin signaling and transcriptional networks. Current opinion in plant biology, 13(5), 533-539.

[84] Perilli, S., Moubayidin, L., & Sabatini, S. 2010. The molecular basis of cytokinin function. Current opinion in plant biology, 13(1), 21-26.

[85] He, Y., Fukushige, H., Hildebrand, D. F., & Gan, S. 2002. Evidence supporting a role of jasmonic acid in Arabidopsis leaf senescence. Plant physiology, 128(3), 876-884.

[86] Kim, H. J., Ryu, H., Hong, S. H., Woo, H. R., Lim, P. O., Lee, I. C., & Hwang, I. 2006. Cytokinin-mediated control of leaf longevity by AHK3 through phosphorylation of ARR2 in Arabidopsis. *Proceedings of the National Academy of Sciences of the United States of America*, 103(3), 814-819.

[87] Frébort, I., Kowalska, M., Hluska, T., Frébortová, J., & Galuszka, P. 2011. Evolution of cytokinin biosynthesis and degradation. Journal of Experimental Botany, 62(8), 2431-2452.

[88] Buchanan-Wollaston, V., Page, T., Harrison, E., Breeze, E., Lim, P. O., Nam, H. G., & Leaver, C. J. 2005. Comparative transcriptome analysis reveals significant differences in gene expression and signalling pathways between developmental and dark/starvation-induced senescence in Arabidopsis. The Plant Journal, 42(4), 567-585.

[89] Lara, M. E. B., Garcia, M. C. G., Fatima, T., Ehneß, R., Lee, T. K., Proels, R., & Roitsch, T. 2004. Extracellular invertase is an essential component of cytokinin-mediated delay of senescence. The Plant Cell, 16(5), 1276-1287.

[90] Dai, N., Schaffer, A., Petreikov, M., Shahak, Y., Giller, Y., Ratner, K., & Granot, D. 1999. Overexpression of Arabidopsis hexokinase in tomato plants inhibits growth, reduces photosynthesis, and induces rapid senescence. The Plant Cell, 11(7), 1253-1266.

[91] Köllmer, I., Werner, T., & Schmülling, T. 2011. Ectopic expression of different cytokinin-regulated transcription factor genes of Arabidopsis thaliana alters plant growth and development. Journal of plant physiology, 168(12), 1320-1327.

[92] Wang, W. 2012. Regulatory RNA-binding proteins in senescence. Ageing research reviews, 11(4), 485-490.

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