Senescence: Regulation and Signalling

13

Riyaz Ahmad Dar, Inayatullah Tahir, and Syed Sabhi Ahmad

Abstract

Senescence is a multifaceted, genetically regulated programme, in which cascade of physiological and biochemical changes occur which bring about the deprivation of macromolecules and the recycling of their components to different parts of the plant. Senescence culminates in death of the plant organ as it necessitates cell viability and is often reversible until the late stages of development. The environmental stress factors such as drought, water logging, high or low solar radiation, extreme temperatures, ozone and other air pollutants, and excessive soil salinity, besides inadequate mineral nutrition in soil, negatively influence the senescence. These stress factors disturb the endogenously regulated system of the plant tissue which may result in promoting the process of the senescence. Despite the initiation by environmental factors, the process of senescence is coordinated through a common signalling network by endogenous and exogenous signals involving the signalling molecules ethylene, abscisic acid (ABA), salicylic acid (SA) and jasmonic acid (JA).

Keywords

Senescence • Ethylene • Abscisic acid (ABA) • Salicylic acid (SA) • Jasmonic acid (JA)

13.1 Introduction

Senescence in a broader sense refers to the process of growing old and consists of those changes that are part of genetically programmed events direct to death of a cell which in plants is often referred to as PCD (programmed cell death) (Lerslerwong et al. 2009; Yamada et al. 2009; Shahri and Tahir 2011; Shibuya 2012). Senescence is an unavoidable process that can be seen in particular at the final stage of ontogenesis,

R.A. Dar • I. Tahir (✉) • S.S. Ahmad
Plant Physiology Lab, Department of Botany, University of Kashmir, Srinagar 190006, Jammu and Kashmir, India
e-mail: reyazadar@gmail.com; inayatullahtahir@gmail.com; syedsabhiindrabi@gmail.com

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during which irreversible changes are initiated leading to gradual cell destruction and death of the organism. This process leads to the modification and degradation of the cell components; both at morphological and metabolic level (Fischer 2012). Senescence can be referred to those events that impart endogenous modulation of death or the process that leads to the death of individuals and/or organs. While as PCD can be defined as the programme in which a cell dynamically executes itself and whereby environmental or developmental stimuli trigger explicit cascade of events that terminate in cell death, it also refers to the process by which cells endorse their own death through the instigation of self-destruction systems. In the context of petal senescence, it seems appropriate to use these words almost interchangeably (Rogers 2006; van Doorn and Woltering 2008). Additionally, as senescence generally leads to death, it should be viewed as a programmed cell death (PCD) process or apoptosis (Vicencio et al. 2008). Apoptosis is a Greek term that generally refers to loss of petal or leaf tissues in plant kingdom (Vicencio et al. 2008; Narcin et al. 2005). Despite this, apoptosis is usually confined to animals which refers to morphological transformations such as nucleosome condensation, shrinkage, nuclear blebbing, decreased cell size and above all cell surface changes that lead to their phagocytosis.

13.2 Structural and Biochemical Changes in Senescing Tissues

The senescing cells demonstrate the characteristic biochemical and structural modifications during the process of senescence. In the case of leaf-senescing cells, the notable among structural changes is the disintegration of intracellular organelles such as chloroplasts (Noodén 1988). In chloroplasts, alterations in the structure of grana, its content and lipid droplet establishment are labelled as plastoglobuli. This leads to chloroplasts shrinkage and their conversion to gerontoplasts which is characterised by the crumbling of thylakoid membranes and deposit of the plastoglobulin. Noodén et al. (1997) proposed that chlorophyll breakdown with the concomitant leaf yellowing are used as markers of senescence. After chlorophyll breakdown lipid, protein and nucleic acid dilapidation takes place. Membrane reliability and cellular compartmentalisation are sustained till the last stage of leaf senescence (Pruzinska et al. 2005). A drop in photosynthates during senescence may lead to sugar starvation proceeding to conversion of lipids to sugars. On the contrary, the mitochondrion and nucleus that are essential for the energy formation and gene expression, respectively, remain integral till the completion of senescence (Lim et al. 2007). The premature and hallmark of flower senescence is the membrane relapse which ultimately leads to structural and functional changes in the senescing tissues. The structural change in the petals of senescing *Dianthus* is vesiculation of cytosolic and vacuolar compartments, whereas in the epidermal cells of daylily, the important change was the degradation of vacuolar membrane. The structural changes include the dehydration of senescing tissues, ion seepage, unusual metabolite convey and liberation of cytochrome C from the mitochondrion. The important biochemical changes were the production of reactive oxygen species (ROS); amplification in lipid peroxidation and membrane fluidity; protein hydrolysis followed by hydrolysis of nucleic acids, lipids and sugars; and ultimately reduction in anabolism (Fig. 13.1). Chloroplast deterioration followed by chlorophyll deprivation and the improved shortfall of important proteins such as ribulose bisphosphate carboxylase (Rubisco) and chlorophyll *a/b*-binding protein (CAB). Depending upon the action of several endo- and exopeptidases hydrolysis of proteins to free amino acids (Otegui et al. 2005; Lim et al. 2007). Senescence-related cysteine proteases play an important role in protein deterioration in the vacuole. Hydrolysis and metabolism of the membrane lipids are brought about by lytic acid hydrolase, lipoxygenase, phospholipase D and phosphatidic acid phosphatase in senescing leaves (Thomas et al. 2003). By performing an important part in flower senescence, numerous cysteine proteases have been exposed to be upregulated and further cloned from petals of

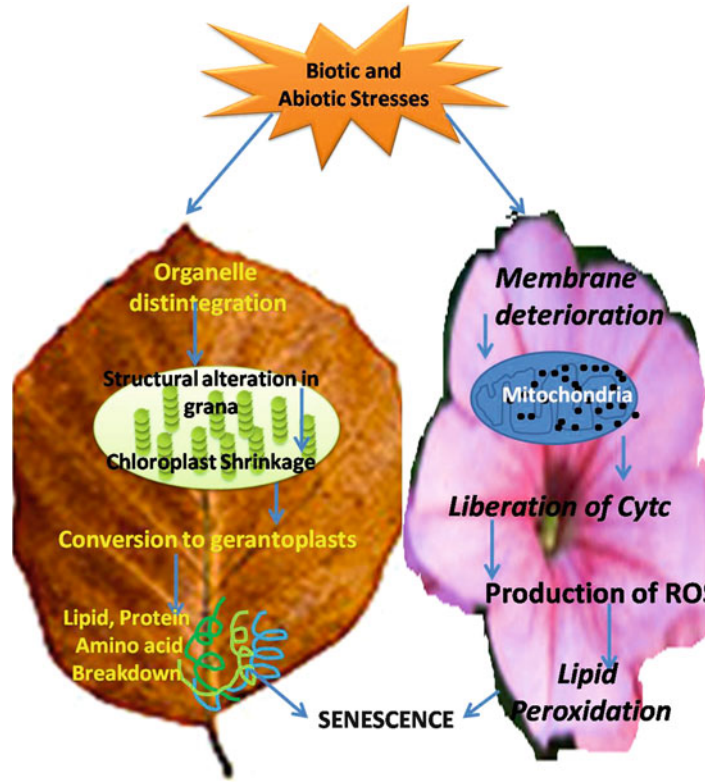

Fig. 13.1 Biochemical changes in senescing tissues

Narcissus, Alstroemeria, Sandersonia and *Petunia*. It has been reported that following the ethylene treatment, most senescence-associated cysteine protease genes increase in abundance. Out of nine cysteine protease genes analysed, six show increased profusion in ethylene-sensitive corollas of *Petunia hybrida* in the course ethylene pursued of petal senescence (Jones et al. 2005). After 3 h of ethylene treatment of *Dianthus caryophyllus*, the expression of cysteine protease *DCCP1* augmented multifold during the petal senescence. After cloning a gene for cysteine protease inhibitor that is expressed profusely in the petals of *Dianthus* during full-opening stage and temporally reduced expression with the progression in flower development that is at senescent stage. It may be concluded that this cysteine protease inhibitor might have an imperative function in the regulation petal senescence by controlling the expression of diverse cysteine proteases. Reactive oxygen species (ROS) generated during the various oxidative reactions is identified to be implicated in natural death of plant tissue together with petals. Hydrogen peroxide is the precursor of these reactive oxygen species; thus, the levels of hydrogen peroxide-regulating enzymes illustrate differential expression during senescence as observed in daylily that augmented activity of superoxide dismutase (SOD) and reduced activity of catalase directs to increased levels of ROS. However, during the senescence of *Dianthus* petals, the increased performance of both ascorbate peroxidase (APX) and catalase, raise in peroxisome number, decrease in the quantity of antioxidants, decrease in membrane transport proteins and redox reactions were reported in several flowers (van Doorn and Woltering 2008).

13.3 Hormonal Regulation of Senescence

The trigger of flower senescence is a very composite phenomenon and administered by the endogenous degree and sensitivity of hormones. The study of plant hormone regulation

on senescence has been reported since a long time back (Tripathi and Tuteja 2007; Fischer 2012). With respect to the trigger of senescence, the role of cytokinins and ethylene has long been implicated and perhaps best understood. But other phytohormones (Schippers et al. 2007; Trobacher 2009) and these phytohormones act as the signalling compound during the process of senescence.

13.3.1 Cytokinins

The role of cytokinins in delaying senescence has long been recognised. The regreening of tobacco leaves was observed after cytokinin treatment, and transgenic plants overexpressing IPT (isopentyl transferase) gene (coding for the enzyme catalysing the rate-limiting step in cytokinin biosynthesis) underneath the SAG_{12} promoter exhibited momentous delay in flower senescence. Based on the experimental findings, the levels of exogenous cytokinins drop during senescence and exogenous application or endogenous enhancement of cytokinin content using the senescence-specific SAG_{12} promoter delaying senescence. The endogenous levels of cytokinins diminish in the leaf-senescing cells, and the genes of cytokinin synthase and adenosine phosphate isopentenyl-transferase (*IPT*) are downregulated and cytokinin oxidase is upregulated during cytokinin synthesis and degradation, respectively (Buchanan-Wollaston et al. 2005).

The strong association between the principal metabolism and anti-senescence effect of cytokinins has been recommended. It has been demonstrated that extracellular invertase is necessary for cytokinin signalling, as cytokinin is no longer able to delay senescence in presence of an invertase inhibitor. Recently, using double-transgenic tomato lines overexpressing both *AtHXK1* (under control of the CaMV 35S promoter) and *IPT* (under control of the SAG_{12} and SAG_{13} promoters), Swartzberg et al. (2011) investigated the interaction of intracellular sugar sensing (by hexokinase) and enhanced cytokinin levels in senescence regulation. Based on visual observations of leaves and on leaf chlorophyll levels, it has been concluded that intracellular sugar sensing by hexokinase is dominant over extracellular sugar sensing (sensing of products of the apoplastic invertase involved in cytokinin signalling), as double-transgenic plants behaved like single-transgenic constitutive *AtHXK1* overexpressors. These experiments also suggest that apoplastic hexoses, in contrast to intracellular sugars, inhibit senescence.

The effect of cytokinins to reduce the onset ethylene biosynthesis and decreased sensitivity to ethylene has been well documented in both petals and leaf tissues. However, similar results were obtained by the exogenous application of kinetin. The interaction between CK and senescence in ethylene-sensitive flowers was gracefully confirmed by Chang et al. (2003), who altered *Petunia* with a SAG12-IPT construct intended to boost CK production at the onset of senescence in leaves. The transformed plants contain more CK content after pollination with the concomitant delay in ethylene synthesis and delay in senescence. The transformed plants were more resistant to exogenously applied ethylene and need longer pulse treatment to induce the endogenous ethylene production and the symptoms of floral senescence. In *Dianthus* petals BA acts as a retarder of the flow of ethylene biosynthesis, involved in natural senescence of *Dianthus caryophyllus*.

13.3.2 Ethylene

The endorsed function of ethylene in senescence has long been confirmed. Ethylene is a gaseous phytohormone formed by plants, and contact of plants to this hormone leads to early senescence in both flowers/petals and leaves (Tripathi and Tuteja 2007). The endogenous levels of ethylene increase during the process of senescence in both senescing leaves and petals with the upregulation of ethylene biosynthetic genes encoding ACC-synthase, ACC-oxidase and nitrilase in many plant species including *Arabidopsis* (Van der Graaff et al. 2006; Lim et al. 2007). Consistent with the notion that the significant delay in leaf senescence in two *Arabidopsis* mutants – ethylene

resistant (etr1) and ethylene insensitive 2 (ein2) – that are scarce in ethylene sensitivity and signal transduction, correspondingly, discloses the importance of endogenous ethylene signalling pathway as a positive regulator of leaf senescence. Delayed senescence was also observed in tomato after the antisense inhibitor of 1-aminocyclopropane-1-carboxylic acid (ACC) oxidase. A MAPK6 is a member of the MAPK pathway that reins plant ethylene signalling during the Arabidopsis senescence regulation. This MAPK is also upregulated by various abiotic and biotic factors and may therefore integrate signals from different pathways influencing plant senescence. Interestingly, the influence of ethylene on senescence is leaf age dependent: Short (1–3 days) ethylene treatments do not induce senescence in young leaves. In older leaves, ethylene effect increases with leaf age, and finally, beyond a certain age, leaf senescence starts even without ethylene as demonstrated by mutant analysis (Buchanan-Wollaston et al. 2005).

Like fruits, ethylene-sensitive flowers also reveal a climacteric rise in ethylene, subsequent pollination and their senescence is triggered by the hormonal changes. In ethylene-sensitive flowers, the senescence-associated changes in petals are observed only after compatible pollination as in the case of *Petunia* which is self-incompatible. The involvement of ethylene in flower development has been studied in great detail in ethylene-sensitive flower systems like *Dianthus, Geranium, Petunia* and orchids. In *Petunia*, *ACO1* is expressed explicitly in senescing corollas and in other floral organs' subsequent ethylene contact, out of four ACC-oxidase (*ACO*) gene members, while *ACO3* and *ACO4* were expressed in developing pistil tissue. The instant and tissue specificity of the amplified expression of *ACO* transcripts concurrent with pollination pursued ethylene production in styles and stigma pursued afterwards by corollas resulting in senescence. Increase in *ACO* transcript in stigma and petals of orchids were allied with pollination. Differential expression of ethylene biosynthetic genes was observed during carnation flower senescence. The genes encoding ethylene receptors show varying expression patterns during flower senescence among species. In carnation, for example, three ethylene-receptor genes, DcERS1, DcERS2 and DcETR1, were identified. DcERS2 and DcETR1 transcripts existed in substantial amounts in petals at the stage of flower development when flowers were fully open. The levels of transcripts for DcERS2 and DcETR1 did not show clear changes in the petals during flower senescence, although the DcERS2 transcript displayed a declining inclination when approaching senescent stage of flower. DcERS1 mRNA was not detected in petals at any stage. Onozaki et al. (2004) reported that ethylene sensitivity is high in the full-opening stage and decreases in the senescing petals of carnation; however, as ethylene receptors are negative regulators of ethylene signalling, it is thought that exhaustion directs to exponential enhancement in sensitiveness.

13.3.3 Abscisic Acid (ABA)

Besides ethylene, ABA is a phytohormone that helps the plant to respond the environmental strains. However, ABA is a key regulator for growth and development of the plant. It has been well documented by several authors (Lim et al. 2007; Tripathi and Tuteja 2007) that the ABA treatment endorses leaf abscission and hastens senescence in certain flowers. After the exogenous application of ABA, unusual early bud abscission and flower senescence was reported in certain cultivars. Being a primary regulator of flower senescence, it also brings about many senescence-associated changes in senescing tissues. These changes comprise ion seepage, enhanced protease activity, lipid peroxidation and expression of novel RNases and DNases. Hunter et al. (2004) proposed that the ABA concentration improved in petal/tepal tissue of senescent flowers as increased content of ABA corresponded with the manifestation of visible signs of senescence in petal/tepal tissue. ABA-mediated senescence initiation in an age-dependent manner was reported quite recently by a membrane-bound receptor kinase (RPK1) rich in leucine content.

This receptor kinase integrates ABA-mediated signals during stress responses, plant growth, stomatal closure and seed germination.

13.3.4 Polyamines (PAs)

PAs play an important role in many biological functions including plant growth and development and during the environmental stresses. Their function in regulation of growth and development in plants has been reviewed by several authors (Kumar et al. 1997; Pandey et al. 2000). There are many forms of PAs, but the most readily available forms of PAs are spermine (Spm), spermidine (Spd) and putrescine (Put) that occur in every plant cell. However, PAs have a well-reputable role in senescence (Tripathi and Tuteja 2007). By acting as anti-senescence agents, PAs have been reported to retard membrane deterioration and chlorophyll loss and increase in protease and RNase activity which untimely helps to retard or slow down the senescence process. By binding to DNA and proteins, PAs stabilise cell membranes. However, they are effective in millimolar concentrations in non-senescent tissues, and their concentration decreases with the age and senescence. Spermine inhibits the senescence in *Dianthus caryophyllus* due to corresponding inhibition of ethylene biosynthesis. This effect of PAs may be due to inhibition of the conversion of SAM to ACC.

13.3.5 Auxins, Gibberellic Acids and Jasmonates

It is very difficult to assign the specific role to auxin in the process of leaf senescence, because of its participation in most of plant developmental phases. However, the endogenous levels of auxin increase during the senescence which suggests its implication in the process of senescence (Sexton and Roberts). During age-dependent leaf senescence, the IAA biosynthetic genes encoding IAA1d oxidase (AO1), tryptophan synthase (TSA1) and nitrilases (NIT1-3) are upregulated (Van der Graaff et al. 2006).

The role of auxins and gibberellic acids in flower senescence has not been well established. However, the exogenous application of auxins in some ethylene-sensitive flowers enhances their senescence. Jones and Woodson (1999) convey that 2,4-D, a synthetic auxin, persuade the expression of ACC synthetase genes in the styles, ovaries and petals. A transitory raise has been reported in the mRNA of an Aux/IAA gene following the application of auxins in the petals of carnation (Hoeberichts et al. 2007). Acting as an antagonist to ethylene, GA application delays the onset of senescence in carnation cut flowers with reduced ethylene production.

Jasmonate has a well-characterised role in the process of senescence and regulates the senescence in isolated oat (*Avena sativa*) leaves. Methyl jasmonates (MeJA) are negative regulators of senescence as exogenous application of MeJA to isolated *Arabidopsis* leaves escorts to a hasty loss of chlorophyll content and decreased photochemical efficiency of photosystem II (PSII) and augmented expression of *SAG*s such as *SEN4, SEN5* and *γ VPE*. The observation that JA-dependent senescence is faulty in the JA-insensitive mutant *coronatine-insensitive 1* (*coi1*). This study provides a realistic approach for the role of JA signalling pathway in sustaining leaf senescence. Jasmonates have an inspiring consequence on flower senescence. Jasmonic acid besides numerous other metabolites rouses the ethylene production by enriching the levels of ACC and promotes the flower senescence of orchid species. However, after a 50h pollination provoked senescence, neither lipoxygenase activity nor jasmonic acid content changed in orchid petals.

13.4 Nonhormonal Regulation of Senescence

Leaf senescence is a dynamic process that can be activated by a multitude of internal and external cues. It diminishes photosynthetic carbon fixation but is important for nutrient recycling particularly nitrogen (Uauy et al. 2006). Accumulation of hexose in the ageing

leaves acts a signal for acceleration or initiation of senescence in annuals (Masclaux-Daubresse et al. 2005; Parrott et al. 2005, 2007; Pourtau et al. 2006; Wingler and Roitsch 2008; Wingler et al. 2009). The role of sugars in leaf senescence was elucidated by various workers. Some researchers believed that sugars act as signal molecules for regulation of senescence, but van Doorn was of the opinion that sugars might not always be a direct cause of senescence but may act via a number of other signals (Wingler and Roitsch 2008; Wingler et al. 2009). Exogenous application of sucrose promotes opening of cut flowers, and delayed senescence, and having no influence on the abscission of petals (Arrom and Munne-Bosch 2012), however changing the hormonal balance in floral tissues. A reasonable good amount of sugar is required for floral bud opening, as substrates for respiration and maintaining osmotic balance of cells. As cut flowers are devoid of any sugar source, the exogenous supply of sugars such as sucrose, glucose and trehalose is essential in promoting their opening.

The stress-generated factors and reactive oxygen species (ROS) containing unpaired electrons including singlet and triplet oxygen, superoxide, nitric oxide and hydroxyl radicals affect cells either through damage (by reacting with DNA, proteins, lipids, etc.) or by acting as signalling molecules, e.g. in hormonal signalling or in response to abiotic and biotic factors (Foyer and Noctor 2005; Pitzschke et al. 2006; Møller et al. 2007; Kazemi and Ameri 2012). Of particular importance to this, the number of evidences indicate that ROS contribute in senescence initiation and signalling (Zimmermann et al. 2006).

SA is a phenol that acts as a plant growth regulator, regulating a number of plant physiological processes including photosynthesis (Sawada et al. 2006; Munne-Bosch 2007). SA is an impending nonenzymatic antioxidant that increases stress tolerance during abiotic and biotic stresses. Sawada et al. (2006) and Kazemi et al. (2011b) showed that SA can prevent ACC-oxidase activity (Ansari and Misra 2007; Mba et al. 2007; Mahdavian et al. 2007; Canakci 2008; Kazemi et al. 2012); also SA appears to act as a germicide that decreases the concentration of bacterial species which occlude the xylem vessels and block them and obstruct the normal flux of water via the stem in cut *Dianthus* flowers. Increased antioxidant activity of enzymes delays the onset of hydrolysis of structural components of cells thereby diminishing the production ROS and a concomitant decrease in ACC oxidase sensitivity and activity. A role for salicylic acid in the regulation of developmental senescence was first demonstrated by Morris et al. (2000) using mutant's defective in SA signalling (*npr1* and *pad4*) as well as *NahG* transgenic plants. In these experiments, induction of the *SAG12* protease gene was dependent on the presence of SA, and mutants defective in SA signalling showed delayed yellowing.

Most of the genes encoding hydrolytic enzymes get expressed during the senescence and bring about the structural disassembly of cell components including macromolecules. Further, genes encoding nucleases, stress responsive enzymes, nitrogen mobilising enzymes and carbohydrates including many transcription factors showed increased expressions during the senescing leaves.

13.5 Reactive Oxygen Species and Nitrogen Species (ROS and RNS) in Senescence Signalling

Molecules having oxygen with unpaired electrons (reactive oxygen species) and nitrogen species (NO) play a critical role by acting as signalling molecules, for example, in hormonal signalling or in biotic and abiotic stresses. Among the ROS species, stress has been laid on H_2O_2 as it is relatively small, uncharged with longer half-life (~1ms) and thus can easily pass the membranes. Because of these reasons, the central position has been attributed to this molecule in signalling pathways. However, nitric oxide (NO) has been revealed to be implicated in several H_2O_2-mediated pathways either an antagonistic or synergistic mode of action. During the ABA-intervened drought- induced leaf senescence,

the ROS complex upstream regulator has been identified. The ROS production by the drought-responsive NAC transcription factor AtNTL4 (ANAC053) directly enhances the activity of gene promoters coding for enzymes involved in ROS biosynthesis (Lee et al. 2011). It has been proposed that the ABA-H_2O2-NO signalling cascade induces stomatal closure. The generation of NO by H_2O_2 induction has been reported in several plants like Mung bean, Arabidopsis and other plant species (Lum et al. 2002; He et al. 2005; Bright et al. 2006). The link between the NO generation and H_2O_2 can be analysed by the exclusion of H_2O_2 in addition to jamming of calcium channels (Neill et al. 2008). The cytoplasmic H_2O_2 can also unswervingly trigger explicitly Arabidopsis MAP triple kinase, AtANP1, which instigate a phosphorylation cascade concerning two stress AtMAPKs, AtMPK3 and AtMPK6. During this discourse MPK6 phosphorylates and thus trigger NR2 ensuing in improved NO assembly (Wang et al. 2010). An additional point of crosstalk among the NO and H_2O_2 signalling pathways has been revealed by positional cloning of rice gene (*NOE1*) coding for catalase, the elimination of which escorts to augmented H_2O_2 contents which in turn increases the activity of NR and leads to eminent NO concentrations. The exclusion of excess NO rearranges the cell death symptoms of the *noe1* mutants revealing an obliging function of H_2O_2 and NO during induction of PCD. Here, exclusively S-nitrosylated proteins were recognised, and overexpression of a rice S-nitrosoglutathione reductase could also raise the cell death signs (Aihong et al. 2012).

13.6 Conclusions

Senescence is not a simple wear and tear mechanism, but is a multifaceted, genetically regulated programme, in which series of biochemical and physiological changes occur which brings about the degradation of macromolecules and the recycling of their components to different parts of the plant. The changes are brought about by plant hormones. Nowadays senescence is a talk of the town as per flower horticulture is concerned. Tremendous work is being carried out with regard to the flower senescence. This review tries to bring the latest information regarding the senescence regulation, mechanism and signalling. We also tried to elucidate the role of growth regulators, reactive oxygen species and nitrogen species in senescence regulation and signalling.

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