International Journal of Biochemistry Research & Review

Volume 32, Issue 5, Page 40-56, 2023; Article no.IJBCRR.102812
ISSN: 2231-086X, NLM ID: 101654445

Plant Senescence: How Plants Choose, How and When to Die

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Author's contribution

The sole author designed, analyzed, interpreted and prepared the manuscript.

Article Information

DOI: 10.9734/IJBCRR/2023/v32i5815

Open Peer Review History:
This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: https://www.sdiarticle5.com/review-history/102812

Review Article

Received: 22/05/2023
Accepted: 28/07/2023
Published: 01/08/2023

ABSTRACT

The word "senescence in plants" refers to the natural ageing process that takes place in plant tissues, organs, and cells and ultimately causes plant death. It is an intricate, tightly controlled process that is affected by both external and internal variables. The internal biological clock of the plant, which is controlled by many external conditions like temperature, light, and nutrition availability, is one of the primary causes of senescence. The internal clock alerts the plant to begin senescing when it reaches a specific age or developmental stage. Several biochemical and physiological changes that the plant experiences during senescence cause the disintegration of cellular elements such as nucleic acids, proteins, chlorophyll and other macromolecules. The plant may continue to grow and develop because these breakdown products are recycled and utilised to create new tissues and organs.

In addition, the plant generates and stores a variety of signalling chemicals, including reactive oxygen species (ROS) and hormones, that control the senescence process. The different physiological and biochemical changes that take place during senescence are coordinated by these signalling molecules, which serve as messengers. Senescence is an ordinary and necessary component of the life cycle of plants, but it can also be brought on early by many environmental challenges, including disease, nutrient inadequacy, and drought. The relevance of comprehending the processes that regulate the senescence procedure is emphasized by the fact that premature senescence can have a major influence on plant growth and productivity.

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The genetically controlled degenerative process of leaf/plant senescence includes nutrient remobilization before the death of leaf/plant tissues. Together with other senescence-causing elements, age plays a significant developmental role in the process. Senescence is regulated at the cellular level by a variety of signalling molecules, hormones, and transcription factors. The complexity of the senescence process, as well as the perception and transmission of senescence signals, as well as subsequent regulatory events, are briefly reviewed in this overview, which also covers current advancements in this area. It will be explored where this field is headed in the future and how related techniques might be used to improve crops.

Keywords: Leaf senescence; age; map kinase cascade; signal transduction; LRR-RLKS; receptor-like kinase; transcription factor.

1. INTRODUCTION

The Senescence of the leaves is the result of a variety of events acting at the end of the plant's life cycle. Senescence begins when a cell loses its ability to produce chlorophyll and progresses to the breakdown of macromolecules such as proteins, carbohydrates, lipids, and nucleic acids. Depending on the stage of a plant's life cycle, degraded molecules are also recycled and transferred to organs and tissues used for storage. Plants with short lifespans restore the translocated nutrients into their fruits and seeds, unlike perennial plants that utilise their root and stem as storage organs to be used later for the growth of new leaves and blooms in the following season.

The gradual degeneration of chloroplasts and lack of photosynthetic activity is what causes the yellow imprint on the leaf that extends from the tip to the base [1,2]. As the grain ripens before harvest, this preferential breakdown of chlorophyll causes monocarpic plants to shift from green to golden yellow. Additionally, the autumn foliage of deciduous trees displays an impressive array of colours due to the breakdown of chlorophyll and the production of new photosynthetic pigments like anthocyanin etc as a result of the breakdown of macromolecules [3, 4]. Although the ageing and death of cells in leaves are frequently thought of as a process, leaf senescence is a well-organized mechanism that functions under strict genetic surveillance to ensure the continued existence of the plant and a supply of essential nutrients for the next generation of cells/ plants.

The signs of a genetically controlled programme include the beginning of senescence, genetic variation in senescence signs, and the mobilization of nutrients in terms of time, space, and quantity. Many hormones, receptors, and transcription factors play a significant role in the control and execution of senescence at the cellular and subcellular levels. Studies at the genetic and molecular levels reveal a wide range of intricate and well-organized ageing syndrome. Genetic approaches focusing on mutants with altered leaf expression of phenotype during senescence have indicated significant involvement of regulatory elements, either positive or negative, for switching on and off the mechanism of senescence [5]. An inclusive molecular understanding of the senescence pattern can be gained by investigating and characterising intracellular and extracellular signalling networks, regulatory elements, and differential gene expression profiles. Signal transduction is an essential part of leaf senescence, even though many external or internal cues/signals have a direct impact on the process's commencement or induction. Environmental signals that cause a plant to age prematurely include drought, salinity, extremes in temperature, a lack of nutrients, low light levels, and pathogen infection. In addition to environmental variables, endogenous hormones play a role in the senescence signalling cascades. A complex system of pathways that are activated as a result of internal and external signalling cues is formed to regulate leaf senescence.

2. SENESCENCE-RELATED SIGNALLING IN PLANTS

Plants naturally go through a process called senescence in which their tissues and organs gradually deteriorate to the point where the plant dies. A sophisticated system of signalling pathways that involve the observation and transmission of numerous internal and external signals controls it. The perception and transmission of these signals are highlighted in the following summary of senescence-related signalling in plants.

a) **Internal Signals:** Plant age, hormone changes, and cellular damage are some of

the internal signs that cause senescence. Certain receptors and sensors that recognise changes in the concentrations of hormones, nutrients, and other signalling molecules are involved in the perception of these signals.

b) **External Signals:** Pathogen infection, nutritional deprivation, and drought are biotic and abiotic stressors that can cause senescence [6]. Certain receptors and sensors that detect chemicals brought on by stress and start the subsequent signalling cascade are also required for the perception of these signals.

c) **Senescence-Related Genes (SAGs):** The detection of senescence-related signals results in SAG activation [7]. These genes create proteins that are involved in several cellular processes, such as the recapture of essential nutrients, the degradation of chlorophyll, and programmed cell death. These genes are only expressed during senescence.

d) **Hormonal Regulation:** Key senescence regulators include the plant hormones like ethylene, ABA, and some organic chemicals like Jasmonic acid, and salicylic acid. These hormones and chemicals are perceived by certain receptors, which then activate downstream signalling pathways involving transcription factors and SAGs [8].

e) **Transcription Factors (TFs):** Several TFs, including NAC, WRKY, and MYB, are activated as part of signalling pathways relevant to senescence. These TFs bind to specific cis-elements in the promoters of SAGs to regulate their expression [9-11].

f) **Protein Kinases and Phosphatases:** Important participants in the signalling associated with senescence include enzymes like protein kinases and phosphatases [12,13]. The activity of TFs and other proteins involved in the senescence pathway is modulated by these enzymes.

g) **Crosstalk among Signalling Pathways:** Signalling pathways associated with senescence are linked and frequently interact with one another. For instance, the hormonal control of senescence may be influenced by the observation and distribution of stress signals, which would then fine-tune the senescence response.

Senescence-related signalling in plants entails the sensing and transmission of a variety of internal and external cues that activate SAGs, TFs, and other proteins implicated in the senescence pathway. It is essential to comprehend the molecular processes underlying these signalling pathways to increase plant tolerance to environmental challenges and increase agricultural yields. Several hormones' effects on the model plant *Arabidopsis* leaf senescence process, as well as the recognition of regulatory genes, notably those encoding transcription factors, have been the focus of much research over the past two decades [9,10,11]. It is still unclear, though, how senescence stimuli and signals are identified, comprehended, and translated into cells, leading to a change in the expression of genes and the beginning of leaf senescence. Additionally, to age, which is regarded to be the primary cue in plants, senescence is hypothesised to be brought on by a variety of environmental conditions. Jasmonic acid, ethylene, salicylic acid, and abscisic acid, as well as other environmental conditions such as nutrition scarcity, darkness, extreme heat, pathogen infection, and drought, are among them. The observation and transduction of diverse stimuli and signals are what ultimately lead to the "senescence syndrome," thus it is to be anticipated that there will be significant overlap and interactions between these communication pathways.

A number of these stimuli and signals are recognised by cell surface receptor proteins, which subsequently transport the information across the plasma membrane to start signal transduction inside the cell and turn on the senescence gene regulatory network. Senescence's complicated regulatory network cannot be fully understood from the little information available on the age- and environment-mediated signal pathways involved in the process [14,15]. Nevertheless, a sizable number of genes that could be involved in signal transduction have been discovered by transcriptome research. Two of these genes' principal categories are (RLKs) receptor-like kinases and (MAPK) mitogen-activated protein kinases.

3. SENESCENCE-REGULATING HORMONES

All living things experience senescence, a natural ageing process that can be controlled by many hormones. Among the hormones that regulate senescence are the following:

a) **Cytokinins:** These hormones encourage plant cell division and postpone senescence. They also play a role in maintaining plant growth and controlling leaf ageing. Cytokinins (CKs), a class of phytohormones, are essential for the developmental process and growth of plants. CKs, referred to as senescence retarding hormones, prevent chloroplast degradation whether they are supplied exogenously or endogenously [16]. The transcriptome analysis revealed that during senescence, the transcript accumulation for the genes encoding the enzymes that break down cytokinin, such as cytokinin oxidase and cytokinin inactivating N- and O-Glycosylases, decreased while it increased for the genes encoding the enzymes that produce it, such as isopentyl phosphotransferase (IPT) and cytokinin synthase. In a variety of plant species, it has been demonstrated that senescence-specific SAG12 promoter-mediated activation of the CK synthesis gene IPT considerably delays leaf senescence [17]. Because IPT was carefully tuned to express itself only during senescence in these trials, senescence-induced boosts in endogenous CK levels prevented or reversed leaf senescence once it had started. Increasing CK levels commonly resulted in increased stress tolerance and delayed stress-induced senescence in the SAG12-IPT-expressing plants, in addition to normal senescence [7]. With enhanced IPT expression, antioxidant enzymes like peroxidase, superoxide dismutase, and catalase were activated, which prevented drought-induced leaf senescence in *Agrostis stolonifera.* In plants overexpressing cytokinin synthesis genes, maintenance of photosynthetic activities and extension in root development under moisture challenges have been found. His protein kinases (AHKs) as the CK receptor, B-response regulators, and A-type nuclear response regulators (ARRs) [18], which act downstream from the receptors by regulating leaf senescence, have all been identified as components of the CK signalling cascade in *Arabidopsis*. A loss-of-function mutation in the protein decreased sensitivity to a cytokinin-dependent delay of leaf senescence, whereas a gain-of-function mutation in the CK receptor AHK3 produced delayed leaf senescence. Furthermore, CK-induced senescence signalling may be mediated by 71 upregulated and 11 downregulated immediate CK-responsive genes in *Arabidopsis* that were identified in a genome-wide expression profile [18].

b) **Gibberellins:** These hormones encourage plant stem elongation and postpone senescence. Moreover, they control how seeds germinate and how fruits develop. Gibberellic acid (GA) encourages the lengthening of stems and leaves, seed germination, flowering, fruit and seed development, and plant responses to various stressors. Since the level of GA's active form declines as embryonic senescence progresses, it has been suggested that GA is a phytohormone that delays senescence. The effect of GA on *Taraxacum officinale* detached leaf tissues in postponing senescence was first demonstrated. Much research shows that the presence of free GA (GA4 and GA7) inhibits the ageing of leaves; nevertheless, one study hypothesised that the reason GA delayed leaf senescence in *Paris polyphylla* was because of the stimulating effects of ABA [19]. Senescence increased the expression of the GA 2-oxidase2 enzyme, which deactivates GA, by 18-fold and is GA-inducible AtGA2OX2. GA operates as an antagonist of leaf senescence, which is supported by the delayed senescence features shown in *Arabidopsis* mutants missing in GA transmission or synthesis [20].

c) **Abscisic Acid (ABA):** This hormone helps plants go into dormancy and promotes the ageing of their leaves. Moreover, it controls how seeds germinate and how organisms react to environmental challenges. Abscisic acid (ABA) is a crucial phytohormone that controls a variety of growth and development processes, such as leaf senescence, abscission, fruit ripening, stomatal closure, and stress reactions to salinity, drought, and low temperatures. Several signal gears, such as the G-Protein, ROS, PP2C, SnRK2, Ca2+, and MAPK pathways, regulate the mechanisms underlying these functions. Much research on ABA unequivocally shows that it plays a favourable regulatory effect in the senescence of leaves [21]. In *Arabidopsis*, senescence is accompanied by increased expression of genes involved in ABA production and signalling responses, according to microarray

studies. Studies have also shown that during senescence, various plants, including *Zea mays, Nicotiana tabacum, Oryza sativa, Avena sativa*, and *Arabidopsis*, exhibit higher levels of endogenous ABA and activation of genes linked to ABA signalling. In *Arabidopsis*, it has been demonstrated that the ABA receptor PYL9 encourages both drought tolerance and leaf senescence. When a plant is dehydrated, ABA avoids early senescence by controlling water loss through stomatal closure. The senescence-associated gene 113 (SAG113), which encodes a protein phosphatase 2C (PP2C), acts as a negative regulator of ABA signalling by preventing stomatal closure, which results in the desiccation of the leaves [22].

d) Auxins: These hormones encourage plant cell elongation and postpone senescence. Also, they play a role in controlling how plants grow and develop. According to certain theories, auxins are essential for plant growth and development and act as an adverse regulator of leaf senescence. Auxin application to *Arabidopsis* detached leaves caused SAG12, a gene that signals the beginning of developmental senescence, to express less. In *Arabidopsis* plants overexpressing the gene for the auxin biosynthetic protein YUCCA6, leaf senescence was delayed and the expression of SAGs was decreased. [23]. Another study discovered that when the short auxin-up RNA gene 39 (SAUR39) was overexpressed in rice, transgenic plants with early senescence features displayed reduced auxin transport and availability [24]. The regulation of senescence and the abscission of *Arabidopsis* leaves has also been linked to the auxin response factor 1 and 2 repressors of auxin response genes. ORESARA14, an *Arabidopsis* gene that encodes ARF2, showed a delay in leaf senescence [25]. However, given that senescence leaves have been discovered to have higher levels of auxin (IAA) and IAA-induced ethylene synthesis in *Nicotiana tabacum* leaves can reverse IAA's senescence-delaying action, the role of auxin in senescence appears to be more convoluted.

e) Ethylene: This hormone encourages the ageing of leaves and the ripening of fruits in plants. Moreover, it controls how plants grow and react to environmental challenges. Ethylene plays some roles in several developmental stages, including senescence, fruit ripening, abscission, and stress reactions to biotic and abiotic stimuli. Since it promotes senescence, ethylene has been referred to as a senescence-promoting hormone. The receptors bound to the membrane at one end, some negative/ positive regulators, and the downstream transcription factors at the other will make up the ethylene signalling cascade, according to projections. Inhibiting the synthesis of ethylene causes a delay in senescence, whereas applying ethylene speeds up senescence in leaves and flowers. Studies on gene expression have shown that during age-dependent leaf senescence, transcripts involved in ethylene production and signalling are more abundant. The mutant ethylene-insensitive2 (ein2) plants displayed delayed embryonic leaf senescence as a result of decreased expression of 21 senescence-associated genes (SAGs), which generate the polygalacturonases and pectinesterases enzymes responsible for cell wall disintegration. By turning on the transcription factors ORE1 and AtNAP, it has been shown that EIN3, a signalling molecule upstream of EIN2, positively regulates leaf senescence [22,26], both of which promote senescence. Exogenous ethylene administration of mature and immature leaves resulted in early and ordinary senescence in the corresponding leaves, illuminating how ethylene signalling cascade-mediated senescence operates in an age-dependent manner [27].

f) Brassinosteroids (BR): Brassinosteroids are used by plants for several processes, such as germination, senescence, flowering, abscission, and in response to various stressors. To detect BR ligands in the BR signalling pathway, three BRI1-associated receptor kinase 1 (BAK1) homologs work as co-receptors of bound to the membrane receptor kinase 1 (BRI1) Protein BRASSINOSTEROID INSENSITIVE 1. Brassinosteroids have been shown to play a role in the positive regulation of leaf senescence through the externally induced acceleration of senescence and the delaying of senescence in BR mutant plants. When Epibrassinolide (eBL) was given to wheat

detached leaves, at low and high concentrations, respectively, both postponed and accelerated leaf senescence was observed. The levels of expression of genes encoding the BR signalling proteins BRI1 kinase inhibitor 1 (BKI1) and BZR1 in senescing leaves were found to be significantly reduced in the *Oryza sativa* early senescence mutant OSPLS1 [28]. In detached leaves of *Pisum sativum* exposed to 24-epibrassinolide (EBR), senescence increased and free fatty acid levels got higher, suggesting that BRs may govern leaf senescence via changing cell lipid composition [29].

g) **Jasmonic acid (JA) and methyl jasmonates (MeJA**): Are engaged in several abiotic and biotic stress responses as well as numerous senescence-related processes in plants. The first indication of jasmonate's role in controlling senescence was the accelerated senescence of MeJA-treated detached oat leaves [14]. Senescence-associated enhancer trap lines in 14 of the 125 *Arabidopsis* plants had reporter gene expression brought on by Jasmonic acid. Senescent leaves produced four times as much endogenous JA as non-senescent leaves, and JA treatments caused senescence to begin in both detached and attached leaves. During the age-dependent senescence of leaves, increased expression of JA synthesis genes (OPR3, AOC4, AOC1, and LOX3) and signalling genes (JAZ1, JAZ6, JAZ8 and MYC2) has been observed**.** After exogenous administration of JA and MeJA, the expression of genes associated with photosynthesis declined. MeJA treatment also raised the expression of genes including SEN4 [30,31], SAG21 and ERD1, that are linked to age-dependent senescence [32]. The regulation of JA-mediated activation for BrOPR3, BrAOC3 and BrLOX4, expression during JA-promoted leaf senescence in *Brassica rapa var. chinensis* has been attributed to the ethylene response factor BrERF72 [32, 33].

h) **Salicylic acid (SA):** For influencing developmental processes such as seed germination, senescence, flowering, and responses to biotic/abiotic stimuli, salicylic acid works as a signalling molecule. As a pro-senescence regulator of embryonic leaf senescence, SA has been hypothesised to influence both the onset and progression of leaf senescence. According to transcriptome and microarray studies, many SA production, signalling, and response genes were shown to be enhanced during the early stages of leaf senescence. Numerous SAGs are thought to be dependent on the SA signalling pathways, according to an examination of the gene expression profiles of *Arabidopsis* NahG in its wild-type and mutant forms. According to an additional study, the transcription factor WRKY75 positively influences the senescence of leaves by increasing the production of SA and reducing H2O2 scavenging, in part via limiting the transcription of CATALASE2 [34].

Every time a cell recognises the cues or signs of senescence, a series of events related to signal transduction involving several signalling elements at various levels of regulation is triggered. The numerous signalling components interact significantly as a result of these occurrences. Hormones play a crucial role in the control of both age-dependent and stress-induced leaf senescence. It has been observed that almost all of the major phytohormones participate in the signalling pathways involved in senescence. In numerous investigations, it has been discovered that ethylene, abscisic acid, brassinosteroids, jasmonic acid, salicylic acid, cytokinins, auxin, and gibberellic acid govern leaf senescence. Controlling senescence is a difficult process involving a variety of hormones and signalling pathways in interaction with one another.

4. PATHWAY FOR SENESCENCE SIGNAL TRANSDUCTION: TRANSCRIPTION FACTORS

Changes in gene expression, nutrition remobilization, metabolism, cellular structure, and cell death are all part of the complicated biological process known as plant senescence. By binding to certain DNA sequences and regulating gene expression and the signalling pathways that cause senescence, transcription factors (TFs) play a significant role in regulating these events. The mechanism for transducing plant senescence signals through transcription factors is summarised as follows:

a) **Initiation of Senescence:** Age, stress, or developmental cues are only a few examples of the internal or environmental

triggers that can start the senescence process. These triggers cause the production of genes linked to senescence through the activation of particular signalling pathways (SAGs).

b) **Activation of TFs:** A variety of transcription factors that function as leading regulators of the senescence process control SAGs. WRKYs, which are named after the conserved WRKY domain, MYBs, and NACs (NAM/ATAF/CUC) are some of the significant TFs implicated in senescence (MYB-like DNA-binding proteins) [34].

c) **TF Regulation of Gene Expression:** By attaching to certain cis-elements in the promoter regions of SAGs after activation, these TFs control how they are expressed. For instance, SAG promoters contain NAC recognition sequences (NACRS) that NAC transcription factors bind to activate the expression of SAGs.

d) **Crosstalk among TFs:** Moreover, the interaction between TFs affects how active they are. For instance, WRKY TFs can fine-tune the senescence process by activating or repressing the expression of NAC TFs.

e) **Feedback Regulation:** TFs themselves are vulnerable to feedback control as senescence develops, which can either augment or reduce their activity. For instance, whereas some NAC TFs suppress their genes, others promote their expression.

f) **Downstream Events:** Many subsequent events, such as chlorophyll degradation, nutrient remobilization, and automated cell death, are brought on by the expression of SAGs that are controlled by TFs.

A wide range of TFs is activated and regulated as part of the complicated and tightly controlled route for plant senescence signal transduction through transcription factors. It is essential to comprehend the molecular processes behind this pathway to increase plant tolerance to environmental challenges and increase crop yields.

The two phases of the senescence signal transduction pathway that involves transcription factors are called initiation and maintenance.

4.1 Initiation Phase

Many stresses, including oxidative stress, oncogene activation, telomere shortening, nutritional deprivation, and DNA damage cause the initiation phase to occur [35]. These stressors trigger several signalling cascades, such as the p53, p16INK4a/pRB, and p38MAPK pathways, which in turn trigger transcription factors involved in senescence.

a) **p53 pathway:** As a result of DNA damage, the cancer suppressor protein p53 is activated, which turns on the transcription of targeted genes responsible for cell cycle arrest, apoptosis, and senescence. The downstream transcription factors, such as p21 and PUMA, which suppress CDK activity and promote the production of pro-apoptotic genes, are the main regulators of p53-mediated senescence.

b) **p16INK4a/pRB pathway:** The cyclin-dependent kinase inhibitor p16INK4a protein stops the activation of CDKs necessary for cell cycle progression. Upstream transcription factors, like E2F, which is released from the pRB protein upon DNA damage, control the activation of p16INK4a. Senescence is brought on by the p16INK4a/pRB pathway, which prevents cell cycle progression.

c) **p38MAPK pathway:** Many stresses, such as oxidative stress and DNA damage, activate the p38MAPK pathway. Senescence-related genes, such as p16INK4a and p21, are expressed as a result of the active p38MAPK pathway activating transcription factors like ATF2 [36,37].

4.2 Maintenance Phase

Senescence has been maintained throughout the maintenance phase thanks to the persistence of senescence-related genes' expression. Several transcription factors, including NF-B, C/EBPβ, and HMGA2, control this phase.

a) **NF-κB:** Many stressors and cytokines stimulate the NF-B transcription factor, which causes the prolonged production of senescence-related genes like IL-6 and IL-8, which support the secretory phenotype associated with senescence (SASP).

b) **C/EBPβ:** When different stresses activate the C/EBPβ transcription factor, senescence-related genes like p16INK4a and p21 are expressed continuously.

c) **HMGA2:** The HMGA2 transcription factor controls the expression of several genes

involved in cell cycle progression and senescence. It does this by modifying the chromatin. For the maintenance of senescence in different cell types, HMGA2 must be expressed over an extended period.

The control of genes relevant to senescence and the signalling pathways that cause senescence are greatly influenced by transcription factors. It may be possible to develop new therapeutic targets for the treatment of age-related disorders as a result of the identification and characterisation of transcription factors implicated in senescence.

5. PERCEPTION AND TRANSDUCTION

Senescence-inducing signals and stimuli are recognised, translated, and cause profound changes in gene expression that fuel the senescence syndrome and ultimately lead to cell death. Senescence-associated genes (SAGs) are genes with upregulated expression patterns, such as photosynthetic genes, whereas senescence-downregulated genes (SDGs) are genes with downregulated expression patterns. Transcriptional factors (TFs), which function as regulatory proteins and switches in the process of differential gene expression, have been shown to have the ability to either induce or inhibit plant senescence. Leaf senescence regulators have been shown to belong to numerous TF families. Based on DNA-binding domains, 2403 TF-encoding genes in the *Arabidopsis* genome have been found to encode at least 287 TFs in 34 gene families, including members of the HB, APE2, MYB, C2H2, bZIP, NAC, and WRKY gene families [34].

NAC is a huge TF family that has frequently been linked to controlling the senescence of leaves. Certain NAC TFs, including ORE1, ANAC016, ATAF1, ORS1, and AtNAP, are positive senescence controllers, and their overexpression frequently causes premature senescence in *Arabidopsis*, whereas their repression causes delayed senescence. Contrarily, the NAC family's negative senescence regulators, including VNI2 (VND- INTERACTING2) [38], and JUB1, have also been observed to cause a delayed senescence phenotype when overexpressed (JUNGBRUNNEN1). NAC TFs typically create intricate regulatory networks during senescence by directing other NACs and collaborating with other TFs or NACs to regulate the transcription and translation of the target genes.

Another important TF family that regulates leaf senescence is the WRKY TF family, which is involved in a variety of growth and growth-related processes, including senescence and stress responses. It is known how WRKY6, WRKY18, and WRKY22/WRKY29 affect defence responses and the control of senescence. A senescence-promoting transcription factor called WRKY53 controls the expression of many SAGs, some of which are connected to pathogen and stress responses. The WRKY53 transcription factor, which has a beneficial role in the regulation of dark-induced senescence and exhibits delayed leaf senescence in its knockout mutant, has been identified as the target of WRKY22 [34]. Additionally, it has been demonstrated that WRKY70 and WRKY54 have a detrimental function in the control of senescence. [36,37].

Positive regulators such as AUXIN RESPONSE FACTOR (ARF2), AP2, G-box binding factor GBF1, C-repeat binding factor CBF2, and negative regulators like the B-type cytokinin response regulator ARR2 are members of other transcription factor families that regulate senescence [25].

5.1 Senescence-Related Receptor-Like Kinases (RLKs) in Plants

Senescence, or the ageing and dying process in plants, is greatly influenced by Receptor-Like Kinases (RLKs). RLKs control a variety of biochemical and physiological events during senescence, including hormone signalling, nutrition remobilization, and cell death.

One example of an RLK linked to plant senescence that has been extensively researched is the Ethylene Response Factor (ERF) subfamily of RLKs. ERFs are responsible for controlling ethylene-mediated signalling, one of the primary regulators of senescence in many plant species. It has been established that ERFs regulate the expression of genes involved in leaf senescence, including those that code for chlorophyll-degrading enzymes. The Somatic Embryogenesis Receptor Kinase (SERK) family of RLKs, which has been connected to the regulation of programmed cell death during senescence, serves as another example [33]. The signalling pathways for abscisic acid (ABA), ethylene and brassinosteroids, all of which are important in controlling senescence, are all mediated by SERKs.

RLKs have generally been identified as significant regulators of plant senescence, and additional study into their functions and modes of action may have significant ramifications for agricultural operations including enhancing crop yields and extending the shelf life of harvested goods. Cell-surface receptors with distinct structural characteristics are called plant receptor-like kinases (RLKs). The normal structure of an RLK includes a transmembrane domain that spans the plasma membrane, an extracellular binding domain at the N-terminus for ligand binding, and a cytoplasmic kinase domain that typically participates in signal transduction by phosphorylating downstream components to activate the regulatory network. The extracellular domain is involved in the perception of ligands. After binding to a ligand, RLKs can auto-phosphorylate the intracellular component, activating the downstream regulatory network and changing gene expression [39]. RLKs, the biggest superfamily of proteins in plants with over 600 members encoded by the *Arabidopsis* genome, play a wide range of functional roles in growth, development, resistance to pathogens, self-incompatibility and hormone responses. Epidermal growth factor repeats (EGFR), self-incompatibility (S), leucine-rich repeats (LRR), and lectin domains are a few of the more than 20 distinct types of extracellular domains of RLKs that have been described. Leucine-rich repeat receptor-like protein kinase (LRR-RLK), which contains more than 200 members, is the biggest subfamily of RLKs in *Arabidopsis*. The extracellular binding domain of LRR-RLKs contains varying numbers of leucine-rich repeat units, which are generally 24 amino acids long. Although though numerous LRR-RLK proteins have been discovered in a wide range of plant species, including *Arabidopsis, Solanum tuberosum, Populus, Oryza sativa and Solanum lycopersicum* only a small number of LRR-RLKs have been proven to be involved in plant growth and stress responses thus far [40]. RLKs are appropriate components for receptors for signals that cause senescence because they function by detecting signals at the cell surface and translating those signals across the plasma membrane to start signal transduction inside the cell. Several LRR-RLK family members have been identified as leaf senescence regulators. A senescence-associated receptor-like kinase (SARK), an LRR-RLK gene with increased levels of transcript and protein accumulation during leaf senescence, has been discovered in the *Phaseolus vulgaris cv. bulgarian*. To increase IPT expression and plant stress tolerance, the PSARK promoter has been utilised [41]. SARK's role in regulating senescence, however, has not been documented. Later, the *Glycine max* was used to isolate another senescence-upregulated LRR-RLK gene known as GmSARK, which was discovered to be important in regulating leaf senescence. It has been shown that the *Arabidopsis* AtSARK and the *Glycine max* GmSARK both acts as positive regulators of leaf senescence. Induced overexpression of the genes GmSARK or AtSARK caused precocious senescence, but leaf senescence was delayed in plants with lower levels of these genes' expression [42]. Recent research has shown that the bean-like LRR-RLK protein PpSARK from the *Physcomitrella patens* functions as a negative regulator of moss senescence. Even though all of the aforementioned SARKs were found to be senescence-upregulated, it is important to underline that only PpSARK was isolated based on sequence similarity and that it shares a lot of similarities with the bean SARK [43,44].

In addition to the SARKs, other LRR-RLK proteins have also been investigated for their roles in *Arabidopsis* leaf senescence. The RECEPTOR PROTEIN KINASE 1 (RPK1) a membrane-bound receptor kinase, favourably controls both age-dependent and ABA-mediated leaf senescence [45]. Senescence was significantly delayed in RPK1 knockout mutants and was induced by ABA as well as ageing processes. More recently, it was shown that SERK4, a somatic embryogenesis receptor-like kinase member belonging to the LRR-RLK subfamily, acts as an antagonist in the signalling pathways responsible for leaf senescence. It has been demonstrated that the five members of the *Arabidopsis* SERK family (SERK1-5) function as co-receptors in a variety of signalling pathways, regulating a variety of stress- and development-related activities. Unexpectedly, it was found that AtSARK is also CLAVATA3 INSENSITIVE RECEPTOR KINASE 3 (CIK3), acting as a co-receptor in the CLAVATA pathway for regulating stem cell homeostasis in *Arabidopsis* with LRR II-RLKs CIK1, CIK2, and CIK4 in the same subgroup. [45]. It has been shown that one co-receptor interacts with many receptors in different signalling pathways. Along with other LRR-RLKs, AtSARK and SERK4 are likely a part of receptor complexes that regulate senescence. The LRR-RLK senescence-induced receptor-like kinase (SIRK), whose expression pattern was characterised, is specific to leaf senescence [39]. More than 40 receptor-like kinase genes have

been connected to the senescence of *Arabidopsis* leaves. A recent study found that controlling leaf senescence and reactions to salt stress depends on the rice receptor kinase OSBBS1/OsRLCK109, a member of the RLCK subfamily but lacking an external domain. Early leaf senescence and sensitivity to salt stress were both traits of the bbs1 mutant. The roles of other LRR-RLKs and RLK subtypes remain to be defined [39].

6. MITOGEN-ACTIVATED PROTEIN KINASE IN SENESCENCE SIGNALLING

RLKs typically initiate phosphorelay-based signal transduction within the cell. The mitogen-activated protein kinase (MAPK) cascade (MAPKKK-MAPKK-MAPK) is one of the most important signalling routes in plants. The MAPK cascade pathways are involved in several physiological processes, including the synthesis of hormones, cell differentiation, and reactions to abiotic and biotic stimuli. In a prior investigation, the senescence of *Arabidopsis* leaves was associated with nine MAPKKK, three MAPKK, and three MAPK genes. In maize, ZmMPK5 is connected to the recovery from low-temperature stress and leaf senescence. The kinase activity of MAPKKK18 and ABA signalling were both necessary for its senescence-regulating function in *Arabidopsis*. It was discovered that MAPKKK18 is a helpful controller of leaf senescence. The ABA signalling pathway was also used by SPOTTED LEAF3 (SPL3), also known as OsMAPKKK1 to favourably regulate leaf senescence in *Oryza sativa* [36,37]. It has been established that MAPKK9 (MKK9) significantly regulates the senescence of *Arabidopsis* leaves. When MKK9 was overexpressed, the transgenic plants displayed an early leaf senescence phenotype but not the kinase-inactive mutant MKK9KR. The detached leaves of mkk9 null mutant plants, on the other hand, exhibited a postponed beginning of senescence. It was found that the *Arabidopsis* MAPKK EDR1 acts as a contraceptive, an antagonist of ethylene-induced leaf senescence, and a negative regulator of defensive responses. It was also discovered that the maize MAPKK ZmMEK1 is a bad senescence regulator. The creation of a dominant-negative mutant of ZmMEK caused SA-dependent leaf senescence in *Arabidopsis*, while ZmMEK1 transcripts accumulated throughout the senescence of dark-induced *Zea mays* leaves. Signals are frequently phosphorylated first and then passed on to MAPKs before they reach the transcription factors, which change the expression of genes. It was discovered that MAPK6 (MPK6) is MKK9's target in the *Arabidopsis* MAPK cascade. In *Arabidopsis*, it has been shown that MKK9 can phosphorylate MPK6 when both MKK9 and MPK6 are expressed in the protoplasts of the mkk9 null mutant. The kinase activity (MKK9EE) was boosted by the presence of a constitutively active form of MPKK9. Most importantly, MKK9's ability to promote senescence was heavily dependent on MPK6, and mutants lacking MPK6 exhibited delayed leaf senescence similar to MKK9. In maize, it was discovered that the MAPKK ZmMEK1 directly targets the MAPK ZmSIMK1 and that the ZmMEK1-ZmSIMK1 cascade's SA level-modulating activities are essential for regulating leaf senescence [46,47, 48].

Transcription factor activity ultimately modifies gene expression as a result of the MAP kinase signalling cascade. It has been discovered that *Arabidopsis* MPK6 works by encouraging the cleavage and nuclear translocation of the protein ORESARA3 (ORE3)/ETHYLENE INSENSITIVE2 (EIN2), which is involved in leaf senescence caused by a variety of stimuli such as MeJA, age, darkness, ABA, and ethylene. The transcription factor EIN3, which hastens MeJA-induced leaf senescence, was maintained by the released C-terminal end of ORE3/EIN2 (CEND). Intriguingly, it was discovered that the DNA-binding protein *Arabidopsis* MAPKKK MEKK1 directly controls the transcription factor WRKY53 which promotes senescence [36,37].

7. OTHER COMPONENTS IN SENESCENCE SIGNALLING

Other signalling systems, including Calcium (Ca2+)-related signalling, also play a significant role in regulating leaf senescence in addition to the MAP kinase cascade. By preventing the (Ca2+) cyt-dependent activation of MPK6, it has been shown that the *Arabidopsis* Bax inhibitor-1 (AtBI1) inhibits MeJA-induced leaf senescence. The Calcineurin B-like-Interacting Protein Kinase 14 (CIPK14) was involved in the interaction and phosphorylation of the ssDNA binding protein WHIRLY1 in *Arabidopsis*. A protein that is known as WHIRLY1 which is found in the nucleus of plastid and regulates the ageing of leaves has been identified. WHIRLY1 accumulated more in the nucleus and showed a stronger affinity for the promoter of the transcription factor WRKY53, which regulates senescence; when it was

phosphorylated by CIPK14 [49]. Senescence-Suppressed Protein Phosphatase (SSPP), a phosphatase 2C-type protein phosphatase, was discovered to be a signalling element working downstream of the LRR-RLK AtSARK. The cytoplasmic domain of AtSARK was discovered to be able to interact with and be dephosphorylated by SSPP. By overexpressing SSPP, AtSARK-induced premature leaf senescence and changes in hormonal responses may be reversed [8]. Another protein phosphatase 2C gene, SAG113, was discovered to be an AtNAP transcription factor direct target gene. SAG113 was reported as adversely modulating ABA signalling, which is significant in controlling the loss of water during the mechanism of leaf senescence [7].

8. MOLECULAR PATHWAYS OF PLANT SENESCENCE

Numerous molecular mechanisms control plant senescence. The most well-known pathway is the leaf senescence pathway, which is characterised by the overexpression of genes related to senescence and the downregulation of genes related to cell division and photosynthesis. A network of transcription factors, including WRKY, NAC, and MYB, controls this pathway by promoting the expression of SAGs [7]. Hormone-mediated pathways, which control plant hormones including ethylene, abscisic acid, and jasmonic acid, are another significant system that controls plant senescence. Ethylene is produced as a response to environmental and developmental signals and is a key regulator of leaf senescence. Abscisic acid, whose concentrations rise as senescence develops, also has an impact on the regulation of leaf senescence. The senescence of both flowers and leaves is controlled by jasmonic acid, which is produced in response to pathogen infection and stress.

The autophagy process, which involves the destruction of cellular components, and the reactive oxygen species (ROS) pathway, which is characterised by the build-up of ROS during senescence, are two other pathways that control plant senescence. The complicated interactions between these pathways might change based on the type of plant and the surrounding surroundings. Plants' senescence is a tightly controlled process that is governed by an intricate web of biochemical pathways. The following are a few of the major molecular processes involved in plant senescence:

a) **Leaf Senescence Pathway:** The leaf senescence process results in an upregulation of senescence-associated genes (SAGs), while genes necessary for cell division and photosynthesis are downregulated. A network of transcription factors, such as WRKY, NAC, and MYB, controls this pathway by promoting the expression of SAGs. As a result of SAG activation, nutrients are recycled to other areas of the plant and cellular components are broken down [37].
b) **Hormone-Mediated Pathway:** Hormones including abscisic acid, ethylene, and jasmonic acid control plant senescence. Ethylene is produced in response to environmental and developmental signals and is a key regulator of leaf senescence. Abscisic acid, whose concentrations rise as senescence develops, also has an impact on the control of leaf senescence. The senescence of both leaves and flowers is controlled by jasmonic acid, which is produced in response to stress and pathogen infection.
c) **Autophagy Pathway:** The autophagy route entails the breakdown of organelles, proteins, lipids, and other cellular constituents to release nutrients that the plant can utilise during senescence. Autophagy-related genes (ATGs), which are involved in the creation of autophagosomes and the transfer of cellular components to the vacuole for destruction, are one of a complex network of proteins that regulate this pathway [37].
d) **Reactive Oxygen Species (ROS) Pathway:** Senescence-related ROS build-up is what distinguishes the ROS pathway. As a by-product of metabolic processes, ROS have the potential to oxidatively harm biological components [15]. Low ROS concentrations can, however, also function as signalling molecules that control gene expression during senescence.
e) **Senescence-Associated Proteases (SAPs) Pathway:** A family of proteases known as SAPs is involved in the cellular component breakdown process during senescence. They are a key component in the disintegration of proteins and other macromolecules and are activated by SAGs.
f) **Epigenetic Pathway:** Via alterations to DNA and histone proteins, the epigenetic pathway controls the expression of genes. These changes may affect how

senescence-related genes like SAGs and others are expressed.

To control plant senescence, various molecular pathways interact with one another in intricate ways. Understanding these pathways is essential for increasing agricultural productivity and longevity as well as reducing the detrimental effects of environmental stress on the development and growth of plants.

9. ENVIRONMENTAL FACTORS AFFECTING PLANT SENESCENCE

Light, water availability, temperature, and nutrient availability are a few environmental elements that might influence how quickly plants age. While low light intensity can postpone senescence by lowering levels of ethylene and other plant hormones, high light intensity can speed up senescence by increasing the generation of ROS.

Senescence in plants is also greatly influenced by temperature, with high temperatures hastening the process and cold temperatures delaying it. Another crucial component is the availability of water; whereas high soil moisture can delay senescence, drought stress frequently speeds it up. Senescence can also be influenced by nutrient availability, with dietary adequacy delaying it and nutrient shortfall increasing it.

10. CONCLUSIONS AND PERSPECTIVES

Senescence is a complex process in plants that is regulated by a variety of molecular pathways and environmental factors. To increase crop productivity and longevity and to create plans to lessen the effects of environmental stress on plant growth and development, it is critical to comprehend the mechanisms underlying plant senescence. To completely understand the molecular mechanisms and environmental elements that control plant senescence and to create practical methods for enhancing plant performance under stressful situations, more study is required.

Plant's senescence is believed to be a complex process in which various environmental cues are absorbed into developmental pathways connected to maturation. As a result, systems that detect environmental stimuli as well as the ageing of cells, organs, and the entire plant are required. To carry out the ageing process, these systems must also transduce, integrate, and choose the appropriate routes. The molecular mechanisms underlying these critical phases have been studied, and this will continue to be an important area of research. It is more challenging to determine how plants coordinate these processes during senescence, both temporally and geographically. New technology and innovative systems biology-based approaches will be needed to fully understand how environmental signals are absorbed into data on developmental age. This choice-making process might be better understood by examining the evolutionary causes of plant senescence. It is still unknown how plants create a special and tightly regulated degeneration mechanism by fusing environmental inputs with developmental signals. One of the key technological challenges to solve these issues would be to set up a phenome laboratory and combine phenomic information with proteomic, genomic, and metabolomic data. This would combine the understanding of the regulatory processes from conception to death with the hereditary and environmental factors that contribute to plant growth and development.

Senescence in plants is a result of the whole developmental process, which involves integrating a variety of internal and external signals into knowledge about developmental age through intricate regulatory pathways. Integrative techniques that enable an assessment of the dynamic changes that occur in physiological, biochemical, and molecular phenotypes are required for a complete understanding of the underlying mechanistic principles of the complicated senescence process in plants. Genomes, transcriptomics, metabolomics, proteomics, epigenomics, and phenomics are just a few of the multi-omics datasets that systems biology approaches combine. These methods should provide a more precise picture of the regulatory networks underlying plant senescence.

The crucial stage of the leaf senescence pathway, signal transduction determines whether the senescence regulatory network downstream is activated [50,51]. The understanding of how leaf senescence is regulated has advanced significantly over the past 20 years, particularly in the areas of hormone regulation and transcriptional regulation [52]. Many signalling components have been uncovered thanks to extensive research into signal perception and transduction. A few of the signalling cascades that have been found are the AtSARK-SSPP cascade, the MKK9-MPK6 cascade in

Arabidopsis, and the ZmMEK1-ZmSIMK1 cascade in *Zea mays*. Much work is still needed to understand the complexity and specifics of the regulatory framework for leaf senescence that connects signals and transcription factors. This network will be clarified through the identification of additional signalling proteins and, perhaps more importantly, by establishing connections between the different parts that have already been identified. In the signal transduction of leaf senescence, there are still several significant problems that need to be solved. For instance, how plant cells understand or detect the cue or signal of ageing and how this information is transmitted to transcription factors to change gene expression.

a) **Changes in the chloroplasts during plant senescence:** The physiological and biochemical changes brought on by senescence in leaf cells include a considerable metabolic shift from anabolism to catabolism that redistributes nutrients to developing organs. The transition from carbon absorption to nutrient remobilization requires the breakdown of cellular components, including chloroplasts. The two reviews in this issue that talk about changes to chloroplasts demonstrate how swiftly this area of research is progressing. The coordinated regulation of chloroplast degradation involves interactions between intra- and extra-plastid degradative mechanisms. The massive loss of chlorophyll stored in chloroplasts results in leaf yellowing, one of the most strikingly evident indicators of plant senescence. With an emphasis on molecular information and biochemical, evolutionary concerns, and transcriptional regulation of the process, the multi-step pheophorbide a PPh/phyllobilin route for the degradation of chlorophyll is being explored. *Arabidopsis* pheophytinase (PPH), is the main enzyme responsible for breaking down chlorophyll, as well as its biochemical and structural properties. The amino acid residues that make up the catalytic triad of PPH are also discovered through site-directed mutagenesis in conjunction with in silico modelling of the 3D structure [53].

b) **Regulatory mechanisms underlying leaf senescence:** Although there are still a variety of physiological and molecular evaluation approaches, the study of leaf senescence has developed dramatically and evolved greatly over the past 20 years. Recent molecular genetic studies and omics analysis have considerably improved our knowledge of the underlying biological mechanisms of leaf senescence. One of the most intriguing advances in this area has been the identification of multiple transcription factors that are crucial for controlling leaf senescence. Transcription factors are an essential mechanism that controls changes in the transcriptome of the genome, allowing the genome to continuously incorporate developmental and environmental inputs.

c) **Prospects for crop improvement:** An important trait of commercial plants is the senescence of the leaves, which lowers yield and biomass and changes nutritional value. Determining the biological principles underlying the process, including regulation, has thus been a focus of research projects. This is because programmes for plant breeding are interested in manipulating it. The most well-known biotech use of senescence modification technology for plant productivity and quality involves increasing IPT expression under the direction of senescence-associated promoters in senescing leaves to increase cytokinin production in senescing leaves.

d) **Signalling components identified in the process of leaf senescence:** To design new systems or strategies to manage leaf senescence for agricultural productivity, the previously identified signal transducing elements that are now on hand could make for a solid starting point. Based on the well-researched hormonal regulation and transcriptional control of leaf senescence, senescence-manipulating technologies have been created to delay leaf senescence, improve stress tolerance, and increase crop output [54,55]. In contrast to the NAP transcription factor-based technology, which has been employed successfully by more than a dozen plant species, the SAG12-IPT system has been used by more than 20 plant species to delay leaf senescence. Senescence-regulating proteins of the MAP kinase cascade have been identified to take part in stress reactions. Through genetic modification of the signalling components at the intersections of the senescence and stress responses, it may be possible to increase stress tolerance

and delay senescence. As additional signalling elements of the leaf senescence regulatory network are discovered and additional complete signalling pathways are unravelled, it is envisaged that senescence modification strategies with enhanced specificity and efficiency would be created for agricultural development.

ACKNOWLEDGEMENT

The author would like to thank Department of Botany, J V College, Baraut, for providing the necessary facilities. The author is also grateful to the department's personnel for assisting time to time.

COMPETING INTERESTS

Author has declared that no competing interests exist.

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