

# The Role of Hormones in the Aging of Plants – A Mini-Review

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## Key Words

Arabidopsis · Phytohormones · Senescence · Signaling

## Abstract

**Background:** In plants, the final stage of organ development is termed senescence. This is a deterioration process that leads to the decay of tissues and organs, and that, in the case of annual, biennial and/or monocarpic plants, leads to the death of the plant itself. The main function of leaf senescence is nutrient recycle and, since this confers an adaptive advantage, it can be considered an evolutionary selected process. Multiple developmental and environmental signals control senescence, and among them plant hormones are understood to play important roles. In particular, the function of cytokinins and ethylene in senescence has been studied for decades, but it is only since *Arabidopsis thaliana* was established as a model organism for molecular genetic studies that the underlying molecular and biochemical events have begun to be elucidated. **Methods:** In this review, we summarize the present understanding of the role of hormones in the developmental control of leaf senescence in plants and in particular highlight recent studies which address its molecular control. **Results:** Important findings which connect hormone action to developmental senescence were made in the past few years. For example, it was shown that ethylene activity in natural, age-dependent leaf senescence is conferred by the regulatory function of EIN2, an ethylene-signaling component, in the control of the tran-

scription factor *oresara 1* (ORE1), which regulates a large set of senescence-associated genes in their expression. ORE1 mRNA abundance is regulated by the microRNA *miR164*, which in aging plants is degraded in an EIN2-dependent manner, and it is interesting that another microRNA also governs the hormonal control of senescence. *miR319* regulates mRNA abundance of a class of transcription factors which control the expression of LOX2 (lipoxygenase 2), a key enzyme in the JA biosynthetic pathway, and thereby regulates JA homeostasis in senescing leaves. **Conclusion:** Reverse and forward genetics have facilitated the elucidation of molecular mechanisms involved in the control of leaf senescence by phytohormones. Studies initiated on the interactions between the different hormonal pathways that control leaf senescence should improve our knowledge in the future.

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## Introduction

Like other organisms, also plants age as their development progresses. However, depending on reproductive strategies and life form (whether annual, biannual or perennial), plant life spans can vary from a few days to hundreds, and in some cases even thousands of years [1].

The life cycle of flowering plants is a succession of distinct growth phases starting with germination, followed by a juvenile and adult vegetative phase, succeeded by

flowering and reproduction and eventually resulting in senescence of the whole plant, which leads to its death. In plants, in addition to age-dependent, developmental senescence, senescence can also be induced by adverse environmental conditions such as darkness or drought. Moreover, in response to pathogen attack a hypersensitive response (HR) can be initiated, that is a localized self-destruction process, which acts at a cellular level to restrict pathogen spread [2].

During their life cycle, plants continuously produce new organs that are formed and develop using nutrients, part of which are recycled and mobilized from senescing organs. In annual, biennials and other monocarpic plant species, leaf senescence is tightly associated with whole-plant senescence and also with flowering and seed production. A delay of flowering can delay senescence and thereby prolong the leaf and plant life span. This was first shown in classical studies in which flowers or fruit of plants were removed, and vegetative growth, which in monocarpic plants declines when flowering is initiated, was prolonged. However, while many late-flowering or sterile mutants show extended life spans, not all do. In fact, studies with the model plant *Arabidopsis thaliana* have shown that, while whole plant senescence is genetically linked to the control of flowering, flowering-independent pathways also participate [1].

Although senescence is a destructive process, it progresses orderly, in a highly regulated manner, and importantly in plants it can be utilized for the benefit of the organism. At the organ level, leaf senescence is employed to relocate resources in harsh environmental conditions from the leaves to sinks such as perennial structures including stems, over-wintering buds, bulbs or tubers, which contain at least one indeterminate meristem for the outgrowth in the next season. Moreover, when flowering is initiated, a relocation of nutrients from leaves to reproductive organs improves reproductive success [2].

Leaf senescence is a spectacular phenomenon in nature (fig. 1a) and a process well studied since in agriculture and horticulture it significantly limits yields and contributes to large post-harvest losses. Moreover, it is an accessible system, which, in particular in work with *Arabidopsis*, has enabled researchers to elucidate regulatory molecular and biochemical events. Thus, our understanding of leaf senescence is advanced, and it is known that both in the developmental and in the stress-induced control of senescence plant hormones play important regulatory roles.

Hormones are small-molecular-weight compounds that act as chemical messengers to transport signals from

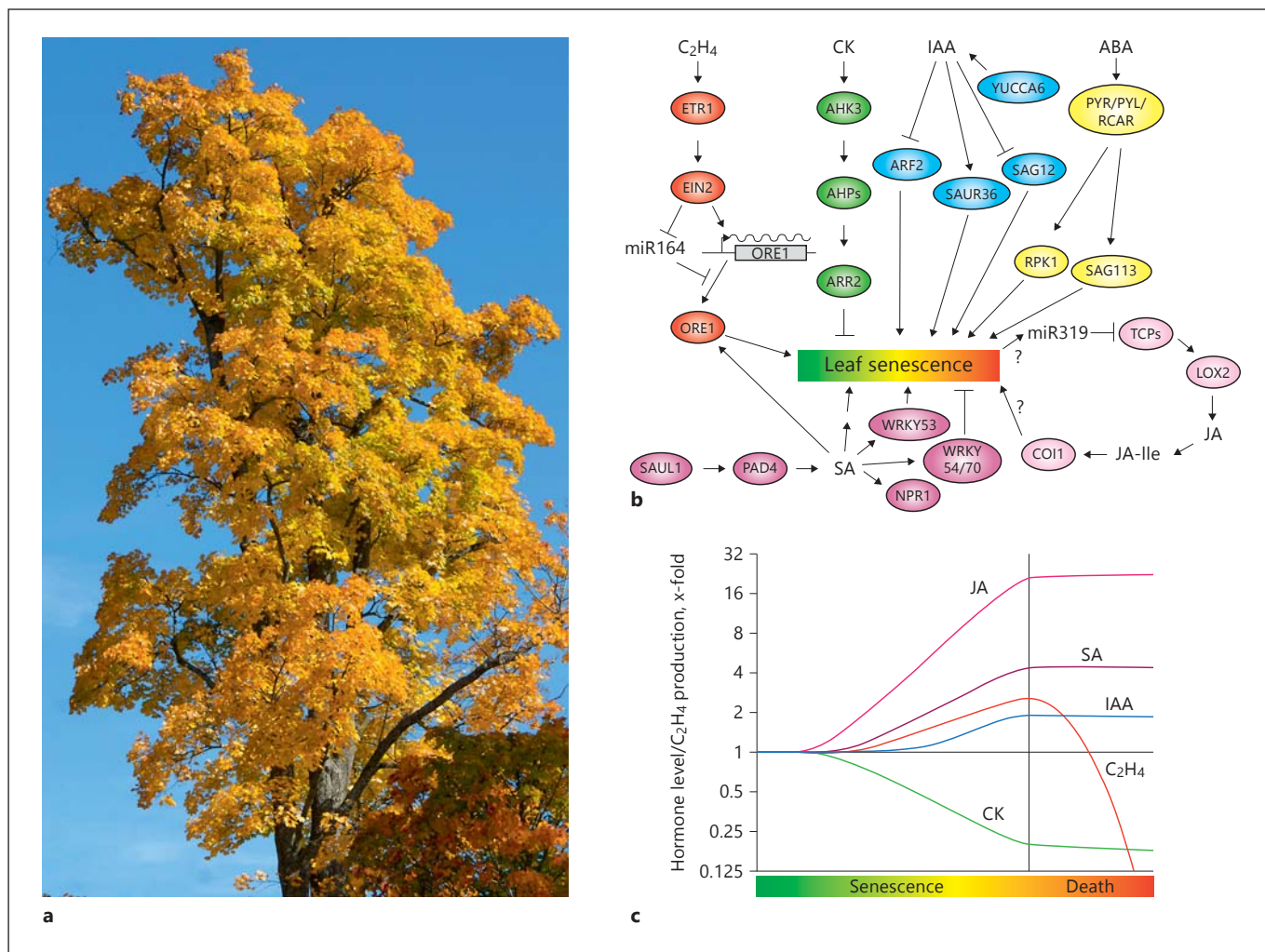
sites of synthesis to sites of action, where they alter gene expression and/or protein activities. In plants, a large number of different classes of hormones exist that act in concert to allow for an integration of environmental signals, such as day length, water supply or perception of stressful growth conditions, into endogenous developmental programs. Therefore, plant hormones are essential to incorporate information about prevailing environmental conditions into important developmental decisions, such as when to germinate, when to flower or also when to initiate senescence [3] (fig. 1b).

In this review, we will briefly summarize our understanding of the role of different classes of plant hormones in developmental and in some instances also dark-controlled senescence and in particular highlight recent advances in this field.

### Cytokinins

A well-studied group of plant hormones are the cytokinins (CKs), which are adenine derivatives with isoprenoid or aromatic side chains. The most abundant CKs in higher plants are isopentenyladenine and zeatin [2]. They act as master regulators of plant growth and play major roles in diverse developmental processes including shoot meristem initiation, leaf and root differentiation, vascular patterning, seed development, photomorphogenesis and gravitropism. In addition, the role of CKs as major senescence-delaying hormone has been known for decades [2].

The idea for a function of CKs in senescence came from the observation that endogenous CK levels declined during leaf senescence [2] (fig. 1c), while exogenous application of CK delayed senescence in a large variety of monocotyledonous and dicotyledonous species [4]. Consistent with these results, the transcription of genes involved in CK biosynthesis and signaling is repressed, while the expression of CK oxidase, an enzyme involved in CK degradation, is induced during leaf senescence [5]. Direct evidence for a function of CKs in senescence was provided when IPT (isopentenyl-transferase), an enzyme that catalyzes the rate-limiting step in CK biosynthesis, was expressed under control of a senescence-inducible promoter in tobacco (*Nicotiana tabacum*). These plants displayed significantly prolonged leaf longevity and all aspects of senescence were remarkably repressed in the adult stage [2, 4]. The approach also yielded similar results for a variety of crop plants including lettuce (*Lactuca sativa*), broccoli (*Brassica oleracea* var. *italica*) and bok choy (*Brassica rapa chinensis*), supporting the idea that



**Fig. 1.** The role of hormones in developmental senescence of plants. **a** Leaf senescence of a maple tree in autumn. **b** Hormone signaling pathways and their contribution to leaf senescence in arabidopsis. Arrows indicate genetic evidence for positive regula-

tory roles. Bars indicate genetic evidence for negative regulatory roles. AHPs = Histidine phosphotransfer proteins. **c** Changes in  $C_2H_4$  production and levels of JA, SA, CK and the auxin IAA during leaf senescence in arabidopsis.

CK-controlled plant senescence is conserved across plant species [2].

In arabidopsis, CKs initiate signaling utilizing three histidine protein kinases, AHK2, AHK3, and AHK4, which act as CK receptors. Upon CK binding, the receptors autophosphorylate and transfer CK signals via histidine phosphotransfer proteins to nuclear localized arabidopsis response regulators (ARRs), which regulate transcription of CK target genes [4]. Importantly, one of the CK receptors, AHK3, also acts as a key component of CK-mediated leaf longevity. A gain-of-function mutation of AHK3 delays leaf senescence, while a loss-of-function mutation in AHK3, but not in the other two CK receptors,

reduces sensitivity to CK in leaf senescence assays. AHK3-mediated regulation of senescence depends on its phosphorylation activity against its downstream target ARR2, a transcription factor of the B type ARRs [6] (fig. 1b).

It is thought that CK targets in senescence are factors which regulate source to sink nutrient transfer since inducing the expression of extracellular invertase, an enzyme involved in the apoplastic phloem unloading pathway, was sufficient to mediate CK-induced leaf longevity. Moreover, inhibition of this extracellular invertase repressed senescence in the presence of CK, supporting the notion that CKs may act by modulating the activity of this enzyme [2].

## Ethylene

In addition to CKs, also ethylene (C<sub>2</sub>H<sub>4</sub>) plays a prominent role in senescence. Ethylene is the smallest plant hormone known and the only one that is a gas under ambient conditions. As such it is highly volatile and is considered to be utilized to synchronize processes in plant populations. Ethylene regulates various developmental processes including seed germination, root hair development, stress responses, flowering, fruit ripening and abscission. In addition, ethylene can act as a strong promoter of senescence, and in many plant species ethylene biosynthesis is induced in senescing leaves and in ripening fruits [2, 7] (fig. 1c).

Although the observation that ethylene-containing illumination gas induced leaf abscission and yellowing was first made more than 100 years ago, the molecular mechanisms underlying ethylene action are only now beginning to be revealed [7]. Ethylene is synthesized from the amino acid methionine in a complex biosynthetic pathway, in which the rate-limiting step, the conversion of S-adenosyl-L-methionine to 1-aminocyclopropane-1-carboxylic acid (ACC), is catalyzed by isoforms of ACC synthase. If ACC synthase expression is reduced, senescence in several plant species is delayed, speaking for a promotive role of ethylene in senescence. However, mutants constitutively overproducing ethylene undergo senescence like wild-type plants, which shows that other factors including age likely also determine ethylene-induced senescence [2].

Ethylene is soluble in lipid membranes and is bound by the ethylene receptors ETR1 (ethylene responsive 1) and homologous proteins. Upon ethylene binding, ETR1 initiates ethylene signaling in which CTR1 (constitutive triple response 1), a negative regulator of the pathway, controls phosphorylation-dependent cleavage of EIN2 (ethylene insensitive 2), a NRAMP-like integral membrane protein that regulates the activity of downstream transcription factors [8].

Several mutants defective in ethylene signaling show malfunctioning senescence including plants deficient in ETR1 and EIN2. A model for how ethylene promotes senescence was proposed by Kim et al. [9], and revolves around the function of the NAC family (for NAM, ATAF1, 2, and CUC2) transcription factor ORE1 (ore-sara 1) that regulates the expression of a large set of senescence-associated genes (SAGs). In this very interesting study, the authors showed that the expression level of ORE1 increases in an age-dependent mode and is under control of EIN2 in a dual manner. In young leaves, ORE1 mRNA abundance is regulated by the microRNA *miR164*,

which in aging plants is degraded by EIN2 releasing ORE1 repression (fig. 1b). In addition, also ORE1 transcription is enhanced by EIN2 [9]. However, how this induction is executed at the molecular level and, importantly, whether ethylene is necessary and sufficient to activate EIN2 in an age-dependent process, remains to be shown.

## Auxins

Auxins are a group of phytohormones which are indispensable for the growth and development of plants and direct a wide range of developmental processes such as apical dominance, embryonic and postembryonic patterning, vascular differentiation, root and shoot development, branching, tropisms and flowering. Biologically active auxins are indole acetic acid (IAA) and indole butyric acid, which are synthesized from tryptophan and exist largely in bound forms [10].

Auxin measurements in senescing leaves have shown that albeit total IAA levels declined, the abundance of free, bioactive IAA increased 2-fold (fig. 1b), which correlates with an increased expression of key enzymes involved in IAA biosynthesis during age-dependent leaf senescence [2]. Therefore, a promotive role of auxins in natural senescence was suspected. A very recent study supports this role since it identified a gene known to be strongly auxin inducible, SAUR36, as highly upregulated in its expression in senescing leaves. The study provides evidence that loss of SAUR36 function results in delayed senescence and that gain of SAUR36 activity leads to premature leaf senescence, showing that SAUR36 is a positive regulator of senescence [11].

Recently, senescence-associated receptor-like kinase from soybean (*GmSARK*), a leucine-rich repeat-receptor-like kinase was identified as another positive regulator of leaf senescence. Arabidopsis plants expressing *GmSARK* under control of a glucocorticoid-inducible promoter showed accelerated leaf senescence, while in plants impaired in the expression of a *GmSARK* orthologue, leaf senescence was significantly delayed. Since *GmSARK* expression was found to be auxin-inducible and the early senescence phenotypes of *GmSARK* overexpressing plants were abolished in an auxin-deficient mutant background, it is thought that auxin regulates senescence through *GmSARK* [12].

In a different recent study, a negative role for auxin in dark-induced leaf senescence was proposed. Flavin-containing mono-oxygenases are a family of enzymes that catalyze the rate-limiting step in auxin biosynthesis and are

encoded by the *YUCCA* gene family in arabidopsis. Interestingly, an overexpression of *YUCCA6* in the dominant *yuc6-1D* mutant led to increased levels of free IAA, but also caused delayed dark-induced senescence. In detached leaf assays performed in the dark, it was found that *yuc6-1D* conferred delayed dark-induced senescence, which correlated with elevated auxin levels and a reduced expression of the senescence marker gene *SAG12* [13]. Therefore, it appears that auxin delays dark-induced leaf senescence, and another recent study supports this hypothesis. This work showed that mutation of *ARF2*, an auxin-responsive transcription factor and repressor of auxin signaling conferred an increased sensitivity to auxin, which correlated with delayed senescence in two allelic mutants, *ore14-1* and *ore14-2* [14]. Similar senescence-related phenotypes were also described in a study conducted with T-DNA insertional mutants of *ARF1* and *ARF2* [15]. Further support for a model in which auxin represses senescence comes from the finding that exogenous application of auxin represses transcription of a subfamily of SAGs [2] (fig. 1b). In summary, the role of auxin in leaf senescence appears to be complex and requires further investigation.

### Salicylic Acid

The phenolic compound salicylic acid (SA) is in particular known for triggering defense reactions against biotrophic pathogens such as the HR and the systemic-acquired resistance, but also appears to play roles in leaf senescence. Mutants with elevated SA levels are severely dwarfed and exhibit necrotic lesions caused by excessive HR induction. Plants with impaired SA biosynthesis or signaling are hypersensitive to biotrophic pathogens but otherwise show normal development, except that leaf senescence is delayed, the expression of several SAGs is decreased and signs of necrosis are repressed during aging. In addition, it was reported that SA levels increase approximately 4-fold in senescing leaves [16] (fig. 1c), and a recent study showed that SA controls the expression of the WRKY transcription factors WRKY53, -54, and -70 [17], transcription factors that are regulated in a senescence-dependent manner [2, 17].

Plants synthesize SA from chorismate or cinnamate, and recently it was shown that the *PAD4* (phytoalexin-deficient4)-dependent SA biosynthetic pathway is crucial for low-light-induced senescence in *saul1* mutants. *SAUL1* (senescence-associated ubiquitin ligase 1) is an E3 ubiquitin ligase important for suppressing pro-death events, and in low-light conditions *saul1* plants show typical

symptoms of senescence including chlorophyll loss, cell death and SA accumulation. One of the earliest events after transfer of *saul1* mutants to low light is the induction of expression of senescence-promoting transcription factors including WRKY53, WRKY6 and AtNAP (NAC-like activated by AP3/PI). Later, also increased transcript levels of *ORE1*, *SAG12* and genes involved in SA biosynthesis and signaling were detected [18]. Interestingly, a treatment with SA was able to induce senescence in *saul1* mutants even in high-light conditions, and this depended on *PAD4* activity (fig. 1b). These data provide clear evidence for a role of *PAD4* in dark-induced senescence. Currently, the biochemical function of *PAD4* is unknown, but it is thought to be involved in an amplification loop that induces SA production in the HR [19]. In summary, several lines of evidence argue for a role of SA in leaf senescence, although the underlying molecular mechanisms are still ill characterized.

### Jasmonic Acid

Oxylipins are important signaling molecules in plants and animals. In plants, the oxylipin jasmonic acid (JA) impacts on a number of processes including response to biotic and abiotic stress, seed germination, flower development, fruit ripening and embryogenesis. JA is synthesized from linolenic acid and is activated by conjugation with isoleucine, which enables binding to COI1 (coronatine insensitive 1), an F-box protein that acts as a JA receptor. COI1 initiates JA signaling by ubiquitinating and thereby stimulating proteasome-dependent degradation of JAZ (jasmonate zim domain) proteins, which repress expression of JA-responsive genes [10].

First evidence for a senescence-promoting role of JA came from the observation that a compound isolated from wormwood (*Artemisia absinthum*) caused rapid chlorophyll loss in oat (*Avena sativa*). This compound was identified as methyljasmonate (MeJA), a volatile JA derivative, and further research showed unambiguously that application of external MeJA induces leaf senescence in a number of plant species. Importantly, the expression of several key enzymes involved in chlorophyll breakdown is induced by JA [20], and recently it was found that JA triggers repression and degradation of rubisco activase, an enzyme which allows for rapid carbamate formation during carbon fixation, in a COI1-dependent manner [21]. In addition, it was shown that JA biosynthesis is upregulated in senescing leaves [22] (fig. 1c). This may be mediated by the action of microRNA *miR319* that targets

a clade of TCP (teosinte branched/cycloidea/PCF) transcription factors which control the expression of *LOX2* (*lipoxygenase 2*), a gene encoding a key enzyme in the JA biosynthetic pathway [23] (fig. 1b).

However, while it is evident that application of JA can promote leaf senescence, compelling evidence for an essential function of JA in leaf senescence is still lacking. In most JA biosynthesis and signaling mutants, initiation and progression of leaf senescence are indistinguishable from wild-type plants. JA mutants that show delayed senescence display pleiotropic defects including infertility, which may explain the observed senescence phenotypes. Recently, the impact of altered JA biosynthesis on developmental as well as on stress-induced leaf senescence was investigated [22]. For this purpose, plants with reduced expression of *LOX2* were generated and assessed for changes in JA levels in developmental and stress-induced senescence. Whereas in wild-type arabidopsis plants JA concentrations increased significantly in all types of senescence, assessed JA levels did not increase in the *LOX2*-silenced plants. Still, these lines showed normal onset and progression of developmental senescence, and only a minor delay of osmotic stress-induced senescence was detected. This suggests that JA may not be essential for these processes [22].

## Absciscic Acid

Absciscic acid (ABA) is formed from three isoprene subunits and is a key hormone in plant adaptation to environmental stresses. ABA is perceived by receptors of the PYR/PYL/RCAR (pyrabactin resistance/PYR1-like or regulatory component of ABA receptor) protein family, which interact with protein phosphatase 2C (PP2C) type phosphatases to repress dephosphorylation of SNF1-related protein kinase 2 (SnRK2), kinases that regulate transcription factor activities and other ABA responses [10].

In addition to its important role in environmental stress responses, ABA also regulates development processes including the induction of seed dormancy, the synthesis of seed storage proteins and lipids and the inhibition of the transition from embryonic to vegetative growth. A promotive role of ABA in senescence was indicated by early studies on ABA function, which have demonstrated that exogenous application of ABA promotes leaf senescence [2]. Also whole-genome expression analyses using microarrays have revealed that genes involved in the key steps of ABA biosynthesis and signaling are upregulated during leaf senescence in arabidopsis [5], which is correlated with an increase in endogenous ABA

levels in many plant species, including tobacco, maize, rice, oat and arabidopsis [2, 24].

Mechanistic evidence for a positive regulatory role of ABA in senescence comes from two studies. One elucidated the role of the receptor-like kinase 1 RPK1 in leaf senescence. RPK1 is a membrane-bound leucine-rich repeat receptor-like kinase that acts as an upstream component of ABA signaling, whose expression was found to increase in an ABA-dependent manner throughout the progression of leaf senescence. Moreover, leaf senescence was accelerated in transgenic plants overexpressing *RPK1*, and ABA-induced senescence was delayed in *rpk1* mutant plants [25], speaking for a promotive role of RPK1 in the pathway (fig. 1b).

A second work dealt with the function of senescence associated gene 113 (*SAG113*), a PP2C protein phosphatase that acts as a negative regulator of ABA signaling [20]. *SAG113* is induced by ABA in senescing leaves and is significantly reduced in its expression in the ABA biosynthesis and signaling mutants *aba2-1* and *abi4-1*. Since leaf senescence is delayed in a *sag113* knockout mutant line and inducible *SAG113* overexpression promotes senescence, these results indicate that *SAG113* is a positive regulator of senescence [24].

## Conclusion

It is evident that hormones play essential regulatory roles in plant senescence. However, from a cell signaling perspective we know little about the factors which interconnect hormone action with this physiological process. Despite recent progress in elucidating molecular events of phytohormone action in senescence, it is likely that we have only just begun to clarify the composition of the regulatory pathways involved. Moreover, in addition to members of the classical plant hormones, there is evidence that also hormones discovered more recently, such as the brassinosteroids and strigolactones, are implicated in senescence, and that phytohormone action is tightly interconnected with the action of other signals such as, for example, reactive oxygen species [17], leaving ample of room for further investigations.

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