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Hormonal regulation of leaf senescence through integration of developmental and stress signals

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Abstract Leaf senescence is a genetically controlled dismantling programme that enables plants to efficiently remobilise nutrients to new growing sinks. It involves substantial metabolic reprogramming whose timing is affected by developmental and environmental signals. Plant hormones have long been known to affect the timing of leaf senescence, but they also affect plant development and stress responses. It has therefore been difficult to tease apart how the different hormones regulate the onset and progression of leaf senescence, i.e., whether they directly affect leaf senescence or affect it indirectly by altering the developmental programme or by altering plants' response to stress. Here we review research on hormonal regulation of leaf senescence and propose that hormones affect senescence through differential responses to developmental and environmental signals. We suggest that leaf senescence strictly depends on developmental changes, after which senescence can be induced, depending on the type of hormonal and environmental cues.

Keywords Senescence · Hormones · Aging · Development · Stress · Age-related-changes

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Introduction

Regulation of senescence by development and environment

The influence of the environment on leaf senescence is visualised particularly well in deciduous trees during autumn. Perennial or polycarpic plants can grow and reproduce for many years and, while their whole plant senescence does not seem to be regulated by development, the senescence of their individual leaves is tightly controlled. Although induction of leaf senescence in deciduous trees appears primarily to be signalled by environmental factors such as day length, in aspen trees it is also strictly dependent on developmental signals (Fracheboud et al. 2009). Monocarpic plants have a different survival strategy and die after they reproduce, which can be after several years. Thus, whole plant senescence of monocarpic plants is under correlative control of reproduction (Davies and Gan 2012) and as such is critically dependent on the plant developmental programme. However, because flowering time is to some extent environmentally controlled (Koornneef et al. 1998), senescence in monocarpic plants also depends on the integration of developmental and environmental signals. Thus, the interaction between the developmental programme and environmental signals ultimately determines the onset of leaf senescence in a wide variety of plants.

Little is known about how plants integrate these signals to control senescence, but extensive research in the model monocarpic plant *Arabidopsis thaliana* (*Arabidopsis*) and other plant species is starting to provide sufficient information to build models that explain how leaves regulate senescence. Figure 1a illustrates a model built around two main findings: first, senescence critically depends on

development, i.e., senescence cannot be induced until a certain developmental stage is reached. Development coincides with cellular changes and here we refer to these changes as age-related-changes or ARCs. We define an ARC as any irreversible change that is strictly dependent on age and thus development. In leaves, the end of cell-division and end of leaf expansion are examples of ARCs. Not all ARCs are readily visible, and any physical, chemical and biochemical change that occurs as a result of differential regulation of developmental processes can be considered an ARC. Furthermore, because ARCs are irreversible, they can be viewed as accumulating, rather than transient (Fig. 1a). Thus ARCs are tangible events that cumulatively describe the process of ageing.

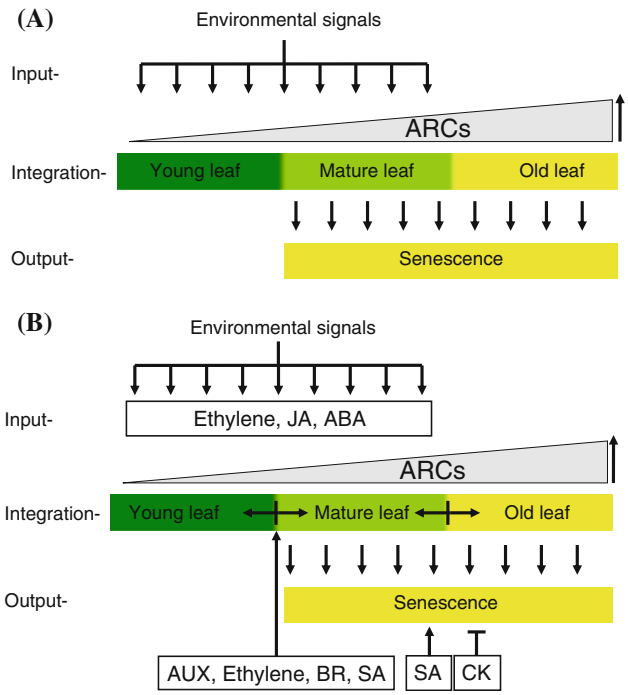

Fig. 1 A tentative model for the regulation of leaf senescence by integration of environmental and developmental signals. **a** Age-related changes (ARCs) take place and accumulate throughout plant development (*right-angled triangle*). Environment and ARCs represent input signals. These signals are integrated and, depending on the occurrence of certain ARCs, the output (senescence) is induced. Therefore, environmental signals cannot induce senescence in young leaves despite adverse environmental conditions, because of the absence of ARCs. As the leaf matures because of ARCs, it becomes competent to respond to the senescence-inducing signals. In old leaves, additional ARCs have occurred, and senescence is induced regardless of environmental cues. **b** The role of hormones in channelling the input (environmental signals and ARCs) and output (senescence). The model emphasises that different hormones have diverse roles throughout leaf development. Ethylene, JA, ABA and SA regulate leaf senescence by responding to environmental cues but its output depends on development, or ARCs. Aux and BR, but also ethylene, can alter the plant developmental programme to change the timing of the occurrence of ARCs (indicated by the *double-arrows*). By contrast, SA and CK regulate senescence by altering the senescence process

We propose that leaf senescence depends on ARCs and, accordingly, that young leaves are insensitive to senescence-inducing signals (Fig. 1a). In *Arabidopsis* this is supported by the observation that senescence cannot be induced until a certain leaf developmental age is reached (Grbic and Bleecker 1995; Weaver et al. 1998; Jing et al. 2002, 2005). Jing et al. (2002) have identified the timing of an ARC in cotyledons, where cotyledons from 19-day-old plants did not senesce in response to ethylene, whereas cotyledons from 23-day-old plants did, despite the cotyledons being visually indistinguishable. Thus, here an ARC occurred at ~21 days, although the nature of the change is unknown. In these cases, ethylene was the senescence-inducing signal, but young *Arabidopsis* leaves are similarly insensitive to jasmonate as a senescence-inducing signal (Fig. 2). The need for ARCs to occur before senescence can be induced may be universal among plants. For example, aspen leaves need to "acquire competence to respond to the photoperiodic trigger to undergo autumn senescence" (Fracheboud et al. 2009). After occurrence of the required ARCs, senescence does not automatically commence. However, at this time the leaf enters a developmental phase where it can integrate environmental signals, which if adverse will cause the leaf to senesce early. Upon further ARCs, senescence will be induced regardless of environmental signals. This ensures that senescence occurs even under environmentally favourable conditions (Fig. 1a). This model makes evolutionary sense, as senescence is a fundamentally destructive process and inducing senescence in young leaves may be too costly, even under the most adverse conditions.

The second finding that forms the basis for our model (Fig. 1a) is that the timing of ARCs is subject to hormonal control. Indeed, it seems logical that hormone treatments that alter the developmental programme are likely to affect the timing of ARCs. Various ethylene treatments were found to have different effects on the occurrence of ARCs (Jing et al. 2005) and this could be the result of a direct effect on development. However, since the environment can affect hormone concentrations, this opens the possibility that the environment can feed back to the developmental programme to change the timing of ARCs and therefore the competence to senesce. Thus, although an ARC is inevitable, the moment at which it occurs is not static, but subject to genetic and hormonal control.

Hormones play a crucial role in developmental processes as well as in the integration of environmental signals to plant development. Indeed, all classical plant hormones have been described as playing a role in the regulation of leaf senescence (Schippers et al. 2007). For example,

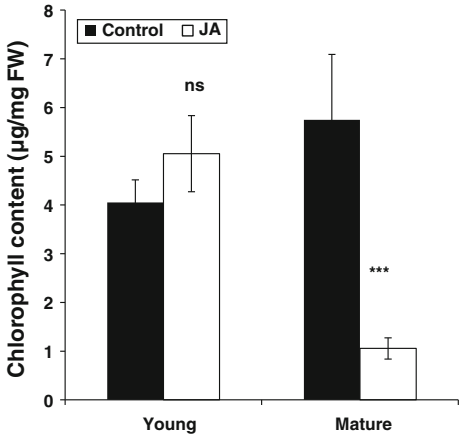

Fig. 2 Mature leaves degreen more rapidly than young leaves when treated with methyl jasmonate. Young and mature leaves of *Arabidopsis* plants were detached and placed in the dark for 3 days at 22 °C in 1 % ethanol (control) or 1 % ethanol containing 100 µM methyl jasmonate. Chlorophyll content was then determined by the method of (Wintermans and De Mots 1965). *Error bars* indicate the standard deviation of the mean of three biological replicates. *ns* Not significant, *** significant ($P < 0.001$)

senescence is accelerated by ethylene, jasmonic acid (JA), abscisic acid (ABA) and salicylic acid (SA), and delayed by auxin, gibberellic acid (GA) and cytokinins (CKs). Defence-related hormones such as ethylene, JA and SA increase during the later stages of leaf senescence (Breeze et al. 2011; Guo and Gan 2012), and were proposed to protect the plant from biotic infections because older leaves become more susceptible to biotic hazards (von Saint Paul et al. 2011). Hormones can therefore potentially affect leaf senescence during all three leaf developmental stages: (1) by affecting leaf development, and as such altering the timing of ARCs (2) by integrating environmental signals, and (3) by affecting the speed at which the senescence process occurs once the leaf has committed to senesce.

Taking the model described in Fig. 1a as a basis, we review the roles that hormones play in regulating the onset of senescence. We first describe the possible nature of the ARCs that occur during plant development and that may allow the leaf to respond to senescence-inducing signals. We then review the recent advancements in our understanding of how different plant hormones regulate senescence and speculate on the role of ARCs in this process. We chiefly discuss developmentally or naturally induced leaf senescence. By this, we mean leaf senescence induced in an intact plant by developmental and environmental processes, as opposed to senescence induced by detachment of tissues, continues darkness or by other treatments. By necessity, we had to make selections from the literature and we have chosen to put emphasis on data obtained from the monocarpic model plant *Arabidopsis*.

The nature of age-related signals

Senescence has evolved as a recycling process in both monocarpic and polycarpic plants, and the reclaimed nutrients are used for production of new photosynthetic tissues, reproductive organs and for seed maturation (Guiboileau et al. 2010). During leaf development, the leaf changes from being a net sink to net source of nutrients. Therefore nutrient balance can be considered an ARC and it can be speculated that the regulation of senescence is associated with changes in cellular or leaf nutrient status. Consistent with this, nutrient remobilisation has been found to correlate with senescence in many species. For example, whole plant senescence in dioecious staminate spinach plants is induced by the exhaustive reallocation of nutrients from the leaves to the small staminate flowers (Sklensky and Davies 2011). Senescence is delayed in pea plants that show a slower rate of flower development and consequently reduced import of nutrients from green leaves (Kelly and Davies 1986, 1988). Senescence is also delayed by crop manipulations such as inhibiting kernel set in maize and depodding in soybean (Wittenbach 1982; Miceli et al. 1995; Borras et al. 2003). However, in soybean the mechanical removal of the reproductive sink did not prevent the drop in photosynthetic efficiency of the leaves (Wittenbach 1982; Craftsbrandner et al. 1984). Although remobilisation of nutrients does occur in both perennials and annuals, it was proposed that in perennials nutrient remobilisation does not trigger whole plant death because of the lower proportion of nutrients remobilised (Davies and Gan 2012).

There are a number of other ARCs which appear important for senescence. Reactive oxygen species (ROS) have long been proposed to function as signals in senescence (Zentgraf 2007; Schippers et al. 2008). ROS accumulate in *Arabidopsis* as the leaf photosynthetic efficiency declines (Bowler et al. 1992; Hensel et al. 1993). Therefore, the age-associated increase in ROS accumulation can be considered an ARC. Changes in biochemical activity and structural conformation of various photosynthetic-related proteins, such as RuBisCO, is also controlled by age (Kato et al. 2004). The age-associated changes in hormone content and the decreases in membrane fluidity with age, due to alterations in membrane constituents such as phospholipids, long-chain fatty acids and sterols (Borochov and Halevy 1978; Itzhaki et al. 1990; Bejatal and Borochov 1994), can also be thought of as ARCs.

Thus, it is tempting to speculate that nutrient status or damage accumulation, as a result of metabolism, are the ARCs that mark the age of a leaf and allow senescence to be induced. However, even if those constitute the signals, it remains to be discovered how these signals are integrated

into the plant developmental programme to allow senescence to commence.

Ethylene

Ethylene is a simple gaseous phytohormone, which affects a wide variety of processes during plant development. The hormone regulates cell division, cell elongation, cell size, fruit ripening, abscission, senescence, and biotic and abiotic stress responses (Abeles 1986; Wang et al. 2002; Skirycz et al. 2011). Ethylene signalling regulates stress-related genes important for plant survival and growth (Achard et al. 2006; Cao et al. 2007). For example, over-expression of the ethylene-inducible transcription factor *AtEBP* in tobacco BY-2 cells inhibits cell death caused by exposure to H_2O_2, heat, and the proapoptotic mammalian protein Bax21 (Ogawa et al. 2005). Mutant plants such as *constitutive triple response 1* (*ctr1-1*), in which ethylene signalling is continuously switched on, show higher survival rates than wild type plants when exposed to salt stress (Cao et al. 2007). Furthermore, the ethylene-insensitive gain-of-function receptor mutants *ethylene response 1-1* (*etr1-1*), *ethylene-insensitive4* (*ein4-1*) and the ethylene-insensitive signalling mutant *ethylene-insensitive 2* (*ein2*), are less tolerant than wild type to salt stress (Cao et al. 2007). Ethylene signalling can, however, also reduce tolerance to other stresses. For example, freezing tolerance of *Arabidopsis* seedlings was reduced in the ethylene over-expressing mutant *ethylene overproducer 1* (*eto1*) and increased in *etr1-1*, *ein4-1* (Shi et al. 2012) and *C-REPEAT/DRE BINDING FACTOR2* (*CBF2*) over-expressing plants, which showed reduced sensitivity to ethylene (Sharabi-Schwager et al. 2010). The diverse role of ethylene throughout the plant life cycle suggests that it is involved in acclimatisation of plants to various stresses by modulating sensitivity and responsiveness of tissues (Ogawa et al. 2005; Achard et al. 2006; Cao et al. 2007; Xu et al. 2008).

Ethylene is widely acknowledged as a senescence-promoting hormone because ethylene treatment accelerates leaf and flower senescence and inhibitors of ethylene synthesis and action can delay senescence (Abeles et al. 1988; Woltering and Vandoorn 1988; Jing et al. 2005). Transcriptomic studies have highlighted the influence of ethylene-related processes during developmentally induced leaf senescence. Transcripts for ~25 % of the identified ethylene synthesis and signalling genes increase in abundance during developmental leaf senescence (van der Graaff et al. 2006). The transcript abundance of ethylene signalling genes was found to increase in concert with transcripts for transport, pectinesterase and lipid catabolic activity at the time chlorophyll concentrations and transcripts of photosynthetic genes declined (Breeze et al. 2011). Buchanan-Wollaston et al. (2005) found that the delayed developmental senescence of *ein2* plants was associated with lower transcript abundance of 21 senescence-associated genes (SAGs) encoding polygalacturonases and pectinesterases involved in cell wall degradation processes. Taken together, these studies suggest that ethylene signalling regulates the latter stages of leaf senescence around the time chlorophyll loss is manifesting, by controlling senescence-associated degenerative and transport activities.

Tight control of ethylene synthesis is also required for the normal timing of senescence. Jing et al. (2005) found that *Arabidopsis* plants treated with ethylene for 16 h had lower numbers of senescing leaves than plants treated for only 12 h. Van Zhong and Burns (2003) found that the number of ethylene-related transcripts altered by treating wild type plants with 20 µl/L ethylene for 24 h was greater than those observed in *ctr1-1* plants, and proposed that constitutive ethylene signalling negatively feeds back to suppress ethylene-regulated expression of genes. This dampening of ethylene response by the continuous presence of ethylene may help to explain why *ctr1* and wild type plants grown under continuous ethylene supply do not show early leaf senescence (Kieber et al. 1993).

The role of ethylene in controlling timing of developmental leaf senescence was further suggested by the delayed senescence of *etr1-1* and *ein2* (Grbic and Bleecker 1995; Oh et al. 1997). EIN2 is also important for senescence controlled by ABA and MeJA, illustrating the crosstalk that occurs between the hormones (Kim et al. 2011a). EIN2 controls the timing of senescence in part through regulating transcript abundance of the ethylene-inducible positive regulator of senescence *NAC DOMAIN CONTAINING PROTEIN 92/ORESARA1-1/ARABIDOPSIS NAC DOMAIN CONTAINING 2* (*ANAC092/ORE1/AtNAC2*) (Kim et al. 2009). Transcript accumulation of *ANAC092* increases during developmental senescence both because its promoter is more active (Balazadeh et al. 2010) and because EIN2 causes an age-related decline in MicroRNA (miR164), which targets *ANAC092* transcripts for degradation (Kim et al. 2009).

The ability of ethylene to accelerate senescence of leaves is dependent on leaf maturity. Ethylene supply to mature leaves causes early onset of leaf senescence, whereas its exposure to young leaves does not (Grbic and Bleecker 1995; Jing et al. 2002). This differential response of ethylene in old and young leaves suggested the requirement of ARCs in fully developed leaves in order for ethylene to alter the timing of senescence. In addition, ethylene has also been found to affect the timing of an ARC (Jing et al. 2005), suggesting that this hormone can affect the onset of senescence in different ways (Fig. 1b). The prerequisite of ARCs in determining the timing of ethylene-signalling-related senescence has also been

suggested by more recent studies. Balazadeh et al. (2010) found that *ANAC092* over-expression accelerated senescence of mature, but not young leaves. It was speculated that this was because *ANAC092* transcripts were degraded by the high levels of miR164 in the young leaves. Chen et al. (2011) identified a chloroplast-localised protein *Arabidopsis* A-fifteen (AAF) that, when over-expressed, caused precocious developmental- and dark-induced leaf senescence. The senescence caused by AAF required a functional EIN2 protein and involved increased production of ROS. Interestingly, although *AAF* transcript abundance is high in young expanding leaves as well as in early senescing leaves during their normal leaf development, it does not promote senescence in the young leaves, presumably because of the absence of ARCs. Lumba et al. (2012) reported that increased ethylene (and/or its signalling pathway) may have an important role in determining the transition of a leaf from the juvenile to adult state. These researchers proposed a model whereby decreased activity of FUSCA3 (FUS3) in the leaf primordium removes repression of ethylene-related signalling, thereby allowing early transition from juvenile to adult leaves. This, they suggested, was consistent with the juvenile leaf identity phenotype observed in *etr1* mutants.

Thus ethylene is a versatile hormone that plays an important role in development, stress response and senescence. Expanding on the model presented in Fig. 1a, we illustrate two proposed main functions of ethylene in the regulation of senescence in Fig. 1b. First, ethylene may alter the leaf developmental programme and, as such, the timing of ARCs. Secondly, in mature leaves that have already acquired the competency to senesce, ethylene may integrate environmental stress signals to regulate the timing of senescence.

Jasmonates

Jasmonates are oxylipins derived from the fatty acid α-linolenic acid, a component of chloroplast membranes. Jasmonates regulate a range of plant growth and development processes such as seed germination, root growth, fertility, anthocyanin production, senescence and plant defence against biotic and abiotic stresses (Wasternack 2007). JA is involved in the activation of both direct (production of glucosinolate, methyl nicotinate and cedrol) and indirect defence responses (by turning on a cascade of defence genes) against wounding and herbivore attacks (Dicke et al. 1999; Birkett et al. 2000; Mewis et al. 2005; Kang et al. 2006; Bruinsma et al. 2007).

Jasmonates have been linked with the senescence programme for many years. Ueda et al. (1981) first showed that exogenously supplied methyl jasmonate accelerated senescence of leaves. More recently JA was shown to activate senescence-associated promoters in 14 out of 125 senescence-enhancer trap lines in *Arabidopsis*, a number similar to that induced by ethylene (He et al. 2001). Methyl jasmonate application also increases transcript abundance of accepted genetic markers of developmental senescence such as *SEN4*, *ERD1*, and *SAG21* (Xiao et al. 2004; Jung et al. 2007). In fact, in *coronatine-insensitive 1* (*coi1*), which is defective in all jasmonate responses (Yan et al. 2009), ~12 % of the developmental SAGs are no longer up-regulated (Buchanan-Wollaston et al. 2005).

Transcript abundance of genes involved in JA synthesis (*LOX3*, *AOC1*, *AOC4*, and *OPR3*) and signalling (*MYC2,JAZ1*, *JAZ6*, and *JAZ8*) increases during developmental leaf senescence (van der Graaff et al. 2006; Breeze et al. 2011). Their increased transcript abundance occurred before chlorophyll was lost from the tissue (Breeze et al. 2011). Jasmonate content also increases in leaves as they senesce developmentally (He et al. 2002; Seltmann et al. 2010; Breeze et al. 2011). He et al. (2002) reported that senescing leaves of *Arabidopsis* had ~4-fold higher JA content than non-senescing leaves, whereas Seltmann et al. (2010) showed that 10-week-old *Arabidopsis* leaves had ~50-fold more JA and ~6-fold more OPDA, a precursor of jasmonic acid, than 6-week-old leaves.

Results obtained from the study of a range of mutants in JA biosynthesis and signalling have, however, been contradictory with respect to the importance of JA in dark-induced senescence. Some mutants impaired in JA signalling, e.g., *coi1-2*, or with reduced jasmonate content because of the silencing of the beta-oxidation gene *3-KETOACYL-COA THIOLASE 2* (*KAT2*), show delayed dark-induced leaf senescence (Castillo et al. 2004; Xiao et al. 2004; Castillo and Leon 2008). However, other mutants, e.g., *allene oxide synthase* (*aos*), which appear completely devoid of jasmonate (Park et al. 2002), and *oxophytodienoic acid reductase* (*opr3*), which accumulates OPDA, the precursor of JA (Stintzi et al. 2001), do not show delayed dark-induced leaf yellowing (Schommer et al. 2008).

The role of JA in regulating developmental senescence is also debatable. Knockout or knockdown lines of the *RuBisCO ACTIVASE* (*RCA*) gene caused chlorotic phenotypes and elevated transcript abundance of *SEN4*, *SAG13*, and *SAG21* in leaves of 3-week-old *Arabidopsis* plants (Shan et al. 2011). RCA protein and transcript abundance was reduced by JA in a COI1-dependent manner, which may therefore be a mechanism by which increased JA content promotes senescence. (Castillo et al. 2004) also found delayed developmental senescence in their *KAT2* antisense plants, but conversely there have been no reports that developmental senescence is altered in *aos* and *opr3* plants. He et al. (2002) noted that natural leaf senescence was not delayed in *coi1* plants compared with the background

Columbia-*glabrous* (Col-*gl1*), although by contrast, Xiao et al. (2004) reported that leaves of *coi1-2* plants showed a slight 10 % delay in chlorophyll loss and reduced transcript accumulation of *SEN4*. Seltmann et al. (2010) examined the importance of increased amounts of JA produced by dark- and developmentally senescing *Arabidopsis* leaves. They found that RNAi silencing of *LIPOXYGENASE 2* (*LOX2*) prevented the increase in the 13-LOX products (JA, OPDA, and arabidopsides) in the leaves. However, this did not delay chlorophyll loss in the leaf tissues. This indicated, first, that LOX2 activity was the predominant driver of jasmonate production and, secondly, that the increase in 13-LOX products was not involved in hastening natural or dark-induced senescence. Seltmann et al. (2010) cautioned that this did not, however, rule out the contribution of other oxidised lipids, because non-enzymatic lipid peroxidation also occurs and expression of a 9-LOX increases in leaves during ageing.

Despite the large body of research on JA and senescence, the role of jasmonates in the initiation of senescence and as a cellular protector during the senescence process remains unclear. However, the importance of JA in stress responses, the increased expression of JA biosynthetic genes before any visible sign of chlorophyll loss, and higher JA content in senescing leaves, all indicate a role for the hormone in leaf senescence. To demonstrate the relationship between ARCs and JA, we treated different-aged *Arabidopsis* leaves with 100 μM methyl jasmonate and found that older leaves degreened more rapidly than younger leaves (Fig. 2). Thus, as indicated in Fig. 1b, we propose that JA functions to integrate stress signals to induce the onset of senescence after ARCs have provided the leaves with the competence to senesce. Its increased concentrations at the last stages of senescence may induce increased cellular protection during this destructive process.

Salicylic acid

Salicylic acid is a phenolic phytohormone, which regulates many aspects of plant growth and development including seed germination, fruit ripening, flowering, senescence and defence response against various biotic and abiotic stresses (Vlot et al. 2009). SA application increases the plant tolerance to a range of abiotic stresses, including salinity, boron and cadmium toxicity, by regulating redox homeostasis (Choudhury and Panda 2004; Eraslan et al. 2007; Shakirova et al. 2007; Çanakci and Dursum 2012).

Salicylic acid positively regulates developmental leaf senescence. For example, *NAPHTHALENE OXYGENASE* (*NahG*)-expressing *Arabidopsis* plants (which have reduced SA concentrations), and *phytoalexin deficient 4* (*pad4*), and *nonexpresser of pathogen related genes* (*npr1*) plants (which are impaired in SA signalling) show delayed senescence compared with wild type (Morris et al. 2000). Furthermore, the SA content of *Arabidopsis* leaves increases at the time chlorophyll concentrations have just started to decline significantly (Breeze et al. 2011), which suggests that SA signalling is involved in controlling the latter part of the senescence programme.

van der Graaff et al. (2006) showed that 12 out of 18 genes putatively involved in SA biosynthesis and signalling were up-regulated in senescing leaves. Transcript profiling of wild type and *NahG* transgenic plants further revealed that ~20 % of SAGs were regulated by the SA-signalling pathway (Buchanan-Wollaston et al. 2005). SA treatment induces expression of many SAGs, including vacuolar processing enzymes (*αVPE* and *γVPE*) (Kinoshita et al. 2010), *SEN1* (Schenk et al. 2005) and WRKY transcription factors *WRKY6* (Robatzek and Somssich 2001) *WRKY53* (Miao et al. 2004), *WRKY54* (Besseau et al. 2012) and *WRKY70* (Ülker et al. 2007). *WRKY70* transcript abundance is abolished in *NahG* plants but unaffected in *aos* and *ein2* plants, indicating that its transcription is specific to the presence of SA (Ülker et al. 2007). Loss of *WRKY70* transcripts causes up-regulation of developmental SAGs and defence genes for both SA (*PR1* and *PR2*) and JA/ethylene (*COR1* and *PDF1.2*) signalling pathways, illustrating the link between plant defence and developmental leaf senescence programmes (Ülker et al. 2007). Besseau et al. (2012) found that the normal increase in *WRKY53*, *54*, *70* and *30* transcripts during senescence is inhibited by 25–55 % in the SA-deficient mutant *sid2*. *WRKY53* positively regulates and *WRKY54* and *WRKY70* negatively regulate developmental senescence (Besseau et al. 2012). Because of this, Besseau et al. (2012) proposed three different phases for the action of WRKY transcription factors in leaf development. First, there is expression of negative regulators prior to leaf senescence; secondly, co-induction of both positive and negative regulators at the onset of senescence; and finally, predominance of positive regulators during the progression of senescence. The study of Besseau et al. (2012) suggests that SA has a role not just in controlling senescence progression, but in controlling the onset as well, by sequentially inducing negative and positive regulators of the programme.

Another mechanism by which SA regulates leaf senescence is autophagy (ATG). SA induces ATG, an intracellular process for vacuolar degradation of cytoplasmic constituents (Yoshimoto et al. 2009). SA and ATG interact to regulate pathogen response and developmental- but not dark-induced leaf senescence (Yoshimoto et al. 2009; Xiao et al. 2010). The ATG mutants, *atg5* and *atg2*, which completely lack ATG, have increased SA and ROS contents and senesce early. Their precocious developmental

senescence is SA-dependent and JA/ethylene-independent, as the early senescence of *atg5* was suppressed in crosses with *NahG*, *salicylic acid induction deficient 2 (sid2)* and *npr1*, but not with crosses with *coi1* or *ein2* (Yoshimoto et al. 2009). Interestingly, the accelerated senescence of *atg5* in the dark was not due to SA, as it was not suppressed by over-expression of *NahG*. The *atg5* mutant also showed excessive immunity-related programmed cell death and spread of chlorotic death upon pathogen infection that was not seen in the wild type, a response dependent on SA production and signalling (Yoshimoto et al. 2009).

Acyl-CoA binding protein 3 (ACBP3) is thought to control senescence through its binding of phosphatidylethanolamine (PE) and its disruption of ATG by inhibiting conjugation of PE to ATG8 (Xiao et al. 2010). *ACBP3* overexpression causes precocious starvation- and developmentally -induced senescence, which is dependent upon the SA- but not the JA-signalling pathway (Xiao et al. 2010). Accelerated developmental leaf senescence was only seen in plants that were older than 3 weeks, despite transcript abundance of *ACBP3* being high in young leaves, suggesting a requirement for ARCs. ACBP3 over-expression in *Arabidopsis* also confers SA- but not JA-dependent plant resistance to *Pseudomonas syringae* pv. *tomato* (Xiao and Chye 2011).

Taken together, the studies suggest that SA has an important role in regulating stress tolerance during the senescence process by influencing lipid metabolism, ATG and production of ROS. We propose that SA is involved in the onset and progression of leaf senescence by controlling the relative abundance of negative and positive regulators of leaf senescence.

Abscisic acid

Abscisic acid is a sesquiterpenoid (15-carbon) hormone that regulates a myriad of plant growth and developmental processes including seed dormancy, seed germination, embryogenesis, stomatal closure, regulation of shoot and root growth, fruit ripening, leaf senescence, abscission and stress responses against various biotic and abiotic stress (Hirayama and Shinozaki 2010; Lee and Luan 2012). Its role in controlling pathogen-associated responses is complex (Mauch-Mani and Mauch 2005). Some studies suggest it increases susceptibility to plant pathogens, e.g., by suppressing defence responses orchestrated by ethylene, JA and SA (Mauch-Mani and Mauch 2005), whereas others suggest it induces tolerance against pathogens, e.g., by suppressing ethylene signalling (De Vleesschauwer et al. 2010), increasing JA production (Adie et al. 2007) and closing stomata to prevent infection of the leaf interior (Melotto et al. 2006). Stomatal closure also occurs in the tolerance response to drought and salinity, serving as a platform for cross-talk between biotic and abiotic stresses (Lee and Luan 2012).

Increasing evidence suggests that plant responsiveness to abiotic stress is intimately linked with leaf longevity (Yang et al. 2011; Zhang and Gan 2012). Yang et al. (2011) showed that the NAC transcription factor *VND-INTERACTING 2* (*VNI2*) is up-regulated during leaf senescence by ABA and by salt stress. Their findings suggest that *VNI2* serves to integrate ABA-mediated abiotic stress signals into the developmental senescence programme through controlling a set of *COLD-REGULATED* (*COR*) and *RESPONSIVE TO DEHYDRATION* (*RD*) genes. Both senescence-induced and salt-induced increases in transcript abundance of *COR15A*, *COR15B*, *RD29A* and *RD29* were dependent on *VNI2* expression. Constitutive over-expression of *VNI2* and these *COR* and *RD* genes resulted in delayed developmental leaf senescence. The increases in transcript abundance of *VNI2*, *COR15A*, *COR15B*, *RD29A* and *RD29* by high salt stress were found to be mediated in part by ABA, as the increases were inhibited in the ABA-deficient mutant *aba3-1*. Together these findings illustrate the close connectivity between ABA stress signalling and developmental senescence signalling pathways, and strengthens the view that enhanced stress tolerance accompanies increased life span.

A number of other studies suggest involvement of ABA in the regulation of developmental leaf senescence. Exogenously applied ABA promotes leaf senescence (Nooden 1988; Becker and Apel 1993; Yang et al. 2002). ABA content increases in many leaf tissues as they developmentally age, including rice (Philosoph-Hadas et al. 1993), maize (He et al. 2005) and *Arabidopsis* (Breeze et al. 2011), and leaf ageing is accompanied by increased transcript abundance of genes associated with ABA biosynthesis and signalling (van der Graaff et al. 2006; Breeze et al. 2011). Ageing leaves lose increased amounts of water because of the ABA-mediated up-regulation of *SAG113,* a gene that encodes protein phosphatase 2C, a negative regulatory component in the ABA signalling pathway that inhibits stomatal closure specifically in senescing leaves (Zhang and Gan 2012). Knockout of *SAG113* delays developmental senescence, whereas its over-expression accelerates the process. Zhang and Gan (2012) further showed that the ABA- and senescence-associated expression of *SAG113* was dependent upon the NAC transcription factor AtNAP, which positively regulates developmental leaf senescence (Guo and Gan 2006). In addition, they found that over-expression of *SAG113* brought the delayed senescence phenotype of *atnap* knockouts back to wild type (Zhang and Gan 2012). This illustrates a strong link between ABA and regulation of developmental senescence. Zhang et al. (2012) proposed that before leaf senescence,

ABA signalling induces stress tolerance processes such as stomatal closure to reduce water loss and suppress senescence, but as the leaf ages, ABA signalling changes to induce transcripts such as *SAG113* that negatively regulate stress tolerance responses to accelerate senescence. The influence of leaf age or maturity on ABA responses related to senescence was also seen for the senescence- and ABA-inducible receptor kinase RPK1 (Lee et al. 2011). These researchers found that *rpk1* knockout mutants showed delayed developmental senescence, whereas conditional over-expression of RPK1 in leaves of 3-week-old plants accelerated senescence. However, induction at an earlier developmental stage retarded growth without triggering senescence, suggesting the requirement of ARCs for its effects on senescence. They also showed RPK1 activity was specific for ABA response, as *rpk1* knockout mutants impaired the ability of exogenously supplied ABA, but not MeJA or ethylene, to cause senescence.

Abscisic acid is involved in various developmental processes and is intricately involved in stress responses. The effect of ABA on leaf senescence appears to depend strongly on ARCs. This therefore suggests that ABA is important for the integration of stress signals to regulate the induction of leaf senescence, but only after the occurrence of the necessary ARCs (Fig. 1b).

Brassinosteroids

Brassinosteroids are polyhydroxysteroids, which influence growth of epicotyls and hypocotyls, seed germination, rhizogenesis, flowering, abscission and senescence reviewed in (Rao et al. 2002). BRs also regulate responses to biotic and abiotic stress (Szekeres et al. 1996; Krishna 2003; Ali et al. 2008; Bajguz and Hayat 2009; Hayat et al. 2010; Hasan et al. 2011). They do so by modulating antioxidant systems, accelerating chlorophyll breakdown and inhibiting anthocyanin production (Ali et al. 2008; Bajguz and Hayat 2009; Hasan et al. 2011). Brassinolide, thought to be the most important BR, induced disease resistance in plants such as tobacco and rice (Nakashita et al. 2003). The resistance did not require SA biosynthesis and was different from that of systemic acquired resistance and wound-inducible disease resistance. In contrast, another study reported that BRs facilitated *Pythium graminicola* pathogen infection in rice by negatively interacting with SA and GA (De Vleesschauwer et al. 2012). BR application has also been shown to improve root nodulation and pod yield in drought-stressed *Phaseolus vulgaris* by increasing cytokinin, free protein and proline contents (Bajguz and Hayat 2009).

Brassinosteroids appear to regulate senescence positively, as BR application accelerates senescence and BR-deficient mutants show delayed senescence (Ding et al. 1995; He et al. 1996; Clouse and Sasse 1998; Yin et al. 2002; Saglam-Cag 2007). Moreover, exogenous application of epibrassinolide (eBL) altered leaf senescence in a dosage-dependent manner, with low eBL concentrations delaying and high concentrations accelerating leaf senescence of detached wheat leaves (Saglam-Cag 2007). The *bri1 (BR insensitive 1)* null mutants display a prolonged life span concomitant with a reduction in transcript levels of several SAGs (He et al. 2007) and the *bri1-EMS-suppressor* 1 exhibits accelerated senescence due to a constitutively active BR response pathway (Yin et al. 2002). A recent study found that $P450_{SUI}$, encoding CYP105A1 monooxygenase, may degrade BRs, and plants over-expressing this gene showed delayed senescence and phenotypes typical of BR-deficient plants (Dasgupta et al. 2011). Similarly, over-expression of *Arabidopsis UGT73C6*, encoding a UDP-glycosyltransferase, inactivates BRs and delays leaf senescence (Husar et al. 2011). Thus BRs have an important function in stress responses and plant development, including the regulation of developmental senescence. Delayed senescence in BR mutants, however, coincides with developmental alterations and therefore it is possible that the altered senescence phenotype is a secondary effect of the altered development. The results seem to suggest that BRs can affect the developmental programme of the leaf and, as such, alter the timing of ARCs and therefore competence to senesce (Fig. 1b).

Gibberellic acid

Gibberellic acid is a pentacyclic diterpene well known for its effect on cell elongation, seed germination, dormancy, reproductive growth, senescence and tolerance against various environmental stresses (Rodrigues et al. 2011). Increased GA content resulting from a mutation in the GA repressor SPINDLY induces salt and drought tolerance in *Arabidopsis* (Qin et al. 2011). Exogenously applying GA to *Catharanthus roseus* alleviated the toxicity symptoms caused by exposure of the plant to cadmium (Pandey et al. 2007) and applying GA to the deciduous pecan tree reduced chlorotic foliar injury caused by the black pecan aphid (Cottrell et al. 2010).

Gibberellic Acid is a senescence-retarding hormone (Schippers et al. 2007) whose active form declines in leaves as they age. For example, expression of the GA-inducible gene encoding GA 2-oxidase 2, which is involved in deactivation of GA, increased 18-fold during senescence, indicating that biologically active GA is removed during developmental leaf senescence (van der Graaff et al. 2006). Furthermore, GA concentrations in leaves of romaine lettuce declined with the progression of senescence because of conversion of GA to an inactive GA glucoside.

Leaf senescence is inhibited by the availability of free GA (GA_4 and GA_7) (Yu et al. 2009b; Li et al. 2010). In *Paris polyphylla*, exogenously applied GA inhibited leaf senescence whereas paclobutrazol, an inhibitor of GA-synthesis, accelerated the programme. Furthermore, it was proposed that GA inhibited leaf senescence through antagonising ABA effects in *Paris polyphylla* or, by preserving sugars due to decreased respiration in Easter lily (Franco and Han 1997; Yu et al. 2009a). Therefore we propose that GAs are not directly involved in the regulation of senescence, but rather they may work by antagonising the effects of ABA.

Auxins

Auxins function in cell growth and development, and much of their signalling components and role in organ architecture has been uncovered (Woodward and Bartel 2005; Kim et al. 2011b). Elucidating the role of auxin in the regulation of developmental leaf senescence has been complicated because of its importance in various aspects of plant growth and development (Lim et al. 2007). During senescence, endogenous indole acetic acid (IAA) concentrations increase twofold (Quirino et al. 1999), but the significance of this observation is unclear.

Several reports point to auxin as a negative regulator of developmental leaf senescence. *YUCCA6* encodes a flavin-containing monooxygenase, which catalyzes the rate-limiting step in auxin biosynthesis (Zhao et al. 2001). Both a YUCCA6 activation mutant *yuc6-1D* and a *35S:YUC6 Arabidopsis* plant showed delayed leaf senescence and decreased *SAG* expression as a result of elevated free IAA concentrations (Kim et al. 2011b). This is consistent with earlier results showing that exogenous auxin application leads to a transient decrease in the expression of many SAGs, including *SAG12* (Noh and Amasino 1999). Kant et al. (2009) furthermore found that increased expression of *SAUR39* (small auxin up RNA) caused lower free IAA concentrations, reduced auxin transport and early senescence. SAUR39 was found to express at higher levels in older leaves compared with younger ones, whereas auxin biosynthesis occurred in younger leaves in meristematic regions. The data suggest that SAUR39 might be involved in auxin signalling and affect developmental leaf senescence by suppressing auxin biosynthesis and its polar transport in an age-dependent manner. Further evidence for a role of auxin in senescence came from the finding that the delayed senescence mutant *oresara14* encodes AUXIN RESPONSE FACTOR 2 (ARF2), which is a repressor of auxin response genes (Lim et al. 2010). These results confirmed work from Ellis and co-workers who found that ARF2 promotes transitions between multiple stages of *Arabidopsis* development (Ellis et al. 2005). Therefore, consistent with its role in plant developmental processes, it is tempting to speculate that auxin regulates developmental leaf senescence by directly modifying the developmental programme and therefore timing of the occurrence of ARCs and consequently the competence to senesce.

Cytokinins

Cytokinins are adenine- or phenylurea-based chemicals widely known for their involvement in regulating various plant growth and developmental processes (Argueso et al. 2010; Perilli et al. 2010) and also for their function in adaptation to stress (Ha et al. 2012). For example, elevated CK content increases drought tolerance and suppresses drought-induced leaf senescence in creeping bentgrass due to higher activity of antioxidant enzymes such as superoxide dismutase, peroxidase, and catalase (Merewitz et al. 2011). Furthermore, higher CK content in transgenic tobacco and creeping bentgrass plants overexpressing CK biosynthesis genes under a stress-induced promotor resulted in drought tolerance. This was thought to result from extended maintenance of components of the photosynthetic machinery and increased root growth relative to shoot growth (Merewitz et al. 2010, 2011, 2012; Rivero et al. 2010).

The senescence-retarding role of CKs appears universal in plants and has been demonstrated in many studies by exogenously applying the hormone, endogenously enhancing its concentration by genetic modification, or in mutants impaired in cytokinin signalling (Gan and Amasino 1995; Ori et al. 1999; McCabe et al. 2001). For example, plants with a gain of function mutation in one of the CK receptors, AHK3, caused delayed leaf senescence, whereas plants with the gene knocked out showed a reduced CK-dependent delay in senescence (Kim et al. 2006).

Cytokinin content decreases in senescing leaves and this has been proposed as one of the key signals for initiating senescence (Gan and Amasino 1995). During senescence, transcript abundance of genes involved in CK biosynthesis such as *isopentyl phosphotransferase* and *cytokinin synthase* decline, whereas mRNA abundance of genes involved in degrading cytokinins increase, e.g., *cytokinin oxidase* and *cytokinin-inactivating N-AND O-GLUCOSYLASES* (Buchanan-Wollaston et al. 2005).

Cytokinins appear to regulate senescence by controlling the activity of extracellular invertase (Lara et al. 2004). Extracellular invertase functions to regulate source-sink relations by hydrolyzing sucrose to hexoses, which are then transported into the cell. Increased CK content results in elevated extracellular invertase activity in tobacco leaves and over-expression of extracellular invertase phenocopies a *SAG12:IPT* plant. Consistent with a fundamental function for

invertase in the delay of senescence, CK does not delay senescence when invertase activity is inhibited (Lara et al. 2004).

Cytokinin-mediated senescence is also controlled by hexokinase (HXK), which induces senescence when over-expressed (Dai et al. 1999). A *HXK Overexpressor/SAG12:IPT* double-transgenic line has the same accelerated senescence phenotype as the HXK over-expressor alone (Swartzberg et al. 2011). This suggests that HXK over-expression is epistatic over increased extracellular invertase activity (Swartzberg et al. 2011), consistent with a primary role for source-sink relations as a regulator of senescence. Furthermore, root-specific increased expression of the CK-degrading gene *cytokinin oxidase/dehydrogenase* (*AtCKX*) resulted in lower foliar CK content and delayed leaf senescence (Werner et al. 2003). The plant also had a greatly increased root:shoot ratio and it is possible that the increased root-sink strength alters the source-sink relations, resulting in delayed senescence despite the lower CK concentrations. Thus the effect of cytokinin on leaf senescence appears to be by altering source sink relations. While progress has been made, the molecular mechanism of CK remains unclear and confirmatory studies of its relationship with source-sink relations in other plant species would be useful.

Cytokinin can efficiently inhibit leaf senescence when its biosynthesis genes are driven by the *SAG12* promoter (Gan and Amasino 1995). *SAG12* is a frequently used marker for developmental leaf senescence that is expressed once the first signs of senescence become visible (Zhang et al. 2010). As such, it is a marker that indicates that the senescence process has already started. The fact that *SAG12*-driven IPT expression so efficiently delays senescence suggests that it can suppress or reverse the senescence process, once started. It has been hypothesised that declining cytokinin content may trigger the onset of senescence. However, leaf senescence did not occur earlier in plants with reduced leaf cytokinin content caused by constitutive over-expression of the cytokinin-degrading gene *AtCKX* (Schmulling et al. 2003), suggesting it regulates progression rather than onset. Thus, as indicated in Fig. 1b, the results suggest that CKs affect the senescence process and not the regulation of its onset.

Integration of hormonal signals

Leaf senescence is a genetically regulated and evolutionarily selected phenomenon that has become part of the plant developmental programme to ensure efficient remobilisation of nutrients from dying parts to reproductive parts (Lim et al. 2007). No mutation, treatment or environmental condition has been found to date that completely abolishes the process, suggesting that leaf senescence is governed by age or developmental default. Thus, ARCs as a result of default developmental processes define the age-window in which senescence can be induced and therefore may be considered master regulators of senescence (Fig. 1a) (Schippers et al. 2007). However, both the timing and progression of senescence are highly flexible and all hormones can either positively or negatively modulate senescence. Hormones integrate developmental and environmental cues, allowing senescence to be adapted to varying environmental conditions in order to extract the optimal amount of nutrients from the dying tissue. Although much of the work discussed above was aimed at isolation of the effect of single hormones, it is becoming increasingly clear that hormonal pathways communicate (Robert-Seilaniantz et al. 2011). In agreement with this, various studies have suggested that developmental- or induced-leaf senescence is regulated by antagonistic and cooperative actions of various hormones (He et al. 2001; Back and Richmond 2006; Depuydt and Hardtke 2011; Delatorre et al. 2012). Furthermore, JA-, ethylene-, and ABA-induced leaf senescence were all delayed in plants defective in the functions of EIN2, ORE9 or CBF2/CBF3 (Kim et al. 2009; Sharabi-Schwager et al. 2010; Woo et al. 2011). Thus, the functions of hormones overlap and they regulate leaf senescence through common genetic pathways. The regulation of leaf senescence by plant hormones therefore appears a complex process and this is consistent with it being a networked control mechanism (Breeze et al. 2011). So how do the hormones modulate senescence? Although the early signalling events after hormone detection have largely been identified, very little is known about the molecular mechanisms that cause changes in timing of developmental senescence. Nevertheless, the past years have seen important progress in the field and here we would like to extend current models (Fig. 1b). Much is based on speculation and we hope that this will be a basis of further discussion and research.

Hormones have a crucial function in development and stress adaptation, and senescence is a developmentally regulated process that can be modulated by stress. We propose that the hormones auxin and BRs affect senescence by modulating plant development. The hormones ethylene, JA SA and ABA, on the other hand, appear to modulate senescence primarily by responding to environmental cues and stress. The stress in itself, however, probably through hormone action, can interact with developmental age and cause alterations in timing of senescence (Jing et al. 2005; Yoshimoto et al. 2009; Chen et al. 2011). The hormonal pathways may work synergistically or antagonistically with one another and thus the integration of stress and developmental age to regulate the leaf senescence depends on the homeostasis of all the hormones.

Therefore there are close relationships among ARCs, stress, hormones and senescence regulation, where the outcome of the integration of developmental and environmental signals ultimately determines the timing of senescence. So what are these ARCs and how do they integrate environmental cues? Nutrient and/or oxidation status may contribute, although direct evidence is lacking. Furthermore, the teasing apart of the effects of individual hormones or their combinations on ARCs and stress should be revealing and help to extend or change current models. Identifying the molecular events leading to changes in ARCs and timing of senescence is another, perhaps more difficult question to answer. Hormones have multiple roles throughout plant growth, and by limiting the effect of hormones to regulate senescence only after the occurrence of certain ARCs, the inherently destructive process called senescence can remain a robust but flexible process that is beneficial to plant survival.

References

Abeles F (1986) Manipulation of plant growth by ethylene. Manip Ethyl Responses Hortic XXII IHC 201:11–20

Abeles FB, Dunn LJ, Morgens P, Callahan A, Dinterman RE, Schmidt J (1988) Induction of 33-Kd and 60-Kd Peroxidases during Ethylene-Induced Senescence of Cucumber Cotyledons. Plant Physiol 87:609–615

Achard P, Cheng H, De Grauwe L, Decat J, Schoutteten H, Moritz T, Van der Straeten D, Peng JR, Harberd NP (2006) Integration of plant responses to environmentally activated phytohormonal signals. Science 311:91–94

Adie BAT, Perez–Perez J, Perez–Perez MM, Godoy M, Sanchez-Serrano JJ, Schmelz EA, Solano R (2007) ABA is an essential signal for plant resistance to pathogens affecting JA biosynthesis and the activation of defenses in *Arabidopsis*. Plant Cell 19: 1665–1681

Ali Q, Athar HUR, Ashraf M (2008) Modulation of growth, photosynthetic capacity and water relations in salt stressed wheat plants by exogenously applied 24-epibrassinolide. Plant Growth Regul 56:107–116

Argueso CT, Raines T, Kieber JJ (2010) Cytokinin signalling and transcriptional networks. Curr Opin Plant Biol 13:533–539

Back A, Richmond AE (2006) Interrelations between gibberellic acid, cytokinins and abscisic acid in retarding leaf senescence. Physiol Plant 24:76–79

Bajguz A, Hayat S (2009) Effects of brassinosteroids on the plant responses to environmental stresses. Plant Physiol Biochem 47:1–8

Balazadeh S, Siddiqui H, Allu AD, Matallana-Ramirez LP, Caldana C, Mehrnia M, Zanor MI, Köhler B, Mueller-Roeber B (2010) A gene regulatory network controlled by the NAC transcription factor ANAC092/AtNAC2/ORE1 during salt-promoted senescence. Plant J 62:250–264

Becker W, Apel K (1993) Differences in gene-expression between natural and artificially induced leaf senescence. Planta 189: 74–79

Bejatal S, Borochov A (1994) Age-related-changes in biochemical and physical-properties of carnation petal plasma-membranes. J Plant Physiol 143:195–199

Besseau S, Li J, Palva ET (2012) WRKY54 and WRKY70 co-operate as negative regulators of leaf senescence in *Arabidopsis thaliana*. J Exp Bot 63:2667–2679

Birkett MA, Campbell CAM, Chamberlain K, Guerrieri E, Hick AJ, Martin JL, Matthes M, Napier JA, Pettersson J, Pickett JA, Poppy GM, Pow EM, Pye BJ, Smart LE, Wadhams GH, Wadhams LJ, Woodcock CM (2000) New roles for cis-jasmone as an insect semiochemical and in plant defense. Proc Natl Acad Sci USA 97:9329–9334

Borochov A, Halevy AH (1978) Microviscosity of plasmalemmas in rose petals as affected by age and environmental factors. Plant Physiol 61:812–815

Borras L, Maddonni GA, Otegui ME (2003) Leaf senescence in maize hybrids: plant population, row spacing and kernel set effects. Field Crops Res 82:13–26

Bowler C, Vanmontagu M, Inze D (1992) Superoxide-dismutase and stress tolerance. Annu Rev Plant Physiol Plant Mol Biol 43: 83–116

Breeze E, Harrison E, McHattie S, Hughes L, Hickman R, Hill C, Kiddle S, Kim YS, Penfold CA, Jenkins D, Zhang C, Morris K, Jenner C, Jackson S, Thomas B, Tabrett A, Legaie R, Moore JD, Wild DL, Ott S, Rand D, Beynon J, Denby K, Mead A, Buchanan-Wollaston V (2011) High-resolution temporal profiling of transcripts during *Arabidopsis* leaf senescence reveals a distinct chronology of processes and regulation. Plant Cell 23: 873–894

Bruinsma M, Van Dam NM, Van Loon JJA, Dicke M (2007) Jasmonic acid-induced changes in Brassica oleracea affect oviposition preference of two specialist herbivores. J Chem Ecol 33:655–668

Buchanan-Wollaston V, Page T, Harrison E, Breeze E, Lim PO, Nam HG, Lin JF, Wu SH, Swidzinski J, Ishizaki K, Leaver CJ (2005) Comparative transcriptome analysis reveals significant differences in gene expression and signalling pathways between developmental and dark/starvation-induced senescence in *Arabidopsis*. Plant J 42:567–585

Çanakci S, Dursum B (2012) The effect of pre-application of salicylic acid on some physiological and biochemical characteristics of tomato seedling (*Lycopersicon esculentum* L) growing in cadmium containing media. Afr J Biotechnol 11:3173–3178

Cao WH, Liu J, He XJ, Mu RL, Zhou HL, Chen SY, Zhang JS (2007) Modulation of ethylene responses affects plant salt-stress responses. Plant Physiol 143:707–719

Castillo MC, Leon J (2008) Expression of the beta-oxidation gene 3-ketoacyl-CoA thiolase 2(KAT2) is required for the timely onset of natural and dark-induced leaf senescence in *Arabidopsis*. J Exp Bot 59:2171–2179

Castillo MC, Martínez C, Buchala A, Métraux JP, León J (2004) Gene-specific involvement of β-oxidation in wound-activated responses in *Arabidopsis*. Plant Physiol 135:85–94

Chen GH, Liu CP, Chen SC, Wang LC (2011) Role of *ARABIDOPSIS* A-FIFTEEN in regulating leaf senescence involves response to reactive oxygen species and is dependent on ETHYLENE INSENSITIVE2. J Exp Bot 63:275–292

Choudhury S, Panda SK (2004) Role of salicylic acid in regulating cadmium induced oxidative stress in *Oryza sativa* L. roots. Bulg J Plant Physiol 30:95–110

Clouse SD, Sasse JM (1998) Brassinosteroids: essential regulators of plant growth and development. Annu Rev Plant Physiol Plant Mol Biol 49:427–451

Cottrell TE, Wood BW, Ni X (2010) Application of plant growth regulators mitigates chlorotic foliar injury by the black pecan aphid (Hemiptera: Aphididae). Pest Manag Sci 66:1236–1242

Craftsbrandner SJ, Below FE, Harper JE, Hageman RH (1984) Effects of Pod Removal on Metabolism and Senescence of Nodulating

and Nonnodulating Soybean Isolines.2. Enzymes and Chlorophyll. Plant Physiol 75:318–322

Dai N, Schaffer A, Petreikov M, Shahak Y, Giller Y, Ratner K, Levine A, Granot D (1999) Overexpression of *Arabidopsis hexokinase* in tomato plants inhibits growth, reduces photosynthesis, and induces rapid senescence. Plant Cell 11:1253–1266

Dasgupta K, Ganesan S, Manivasagam S, Ayre BG (2011) A cytochrome P450 monooxygenase commonly used for negative selection in transgenic plants causes growth anomalies by disrupting brassinosteroid signaling. BMC Plant Biol 11:67

Davies PJ, Gan S (2012) Towards an integrated view of monocarpic plant senescence. Russ J Plant Physiol 59:467–478

De Vleesschauwer D, Yang Y, Cruz CV, Hofte M (2010) Abscisic acid-induced resistance against the brown spot pathogen *Cochliobolus miyabeanus* in rice involves MAP kinase-mediated repression of ethylene signaling. Plant Physiol 152:2036–2052

De Vleesschauwer D, Van Buyten E, Satoh K, Balidion J, Mauleon R, Choi IR, Vera-Cruz C, Kikuchi S, Hofte M (2012) Brassinosteroids antagonize gibberellin- and salicylate-mediated root immunity in rice. Plant Physiol 158:1833–1846

Delatorre CA, Cohen Y, Liu L, Peleg Z, Blumwald E (2012) The regulation of the SARK promoter activity by hormones and environmental signals. Plant Sci 193:39–47

Depuydt S, Hardtke Christian S (2011) Hormone signalling crosstalk in plant growth regulation. Curr Biol 21:R365–R373

Dicke M, Gols R, Ludeking D, Posthumus MA (1999) Jasmonic acid and herbivory differentially induce carnivore-attracting plant volatiles in lima bean plants. J Chem Ecol 25:1907–1922

Ding W, Zhao Y, Ding WM, Zhao YJ (1995) Effect of epi-BR on activity of peroxidase and soluble protein content of cucumber cotyledons. Acta Phytophysiol Sinica 21:259–264

Ellis CM, Nagpal P, Young JC, Hagen G, Guilfoyle TJ, Reed JW (2005) AUXIN RESPONSE FACTOR1 and AUXIN RESPONSE FACTOR2 regulate senescence and floral organ abscission in *Arabidopsis thaliana*. Development 132: 4563–4574

Eraslan F, Inal A, Gunes A, Alpaslan M (2007) Impact of exogenous salicylic acid on the growth, antioxidant activity and physiology of carrot plants subjected to combined salinity and boron toxicity. Sci Hortic 113:120–128

Fracheboud Y, Luquez V, Bjorken L, Sjodin A, Tuominen H, Jansson S (2009) The control of autumn senescence in European aspen. Plant Physiol 149:1982–1991

Franco RE, Han SS (1997) Respiratory changes associated with growth-regulator-delayed leaf yellowing in Easter lily. J Am Soc Hort Sci 122:117–121

Gan S, Amasino RM (1995) Inhibition of leaf senescence by autoregulated production of cytokinin. Science 270:1986–1988

Grbic V, Bleecker AB (1995) Ethylene regulates the timing of leaf senescence in *Arabidopsis*. Plant J 8:595–602

Guiboileau A, Sormani R, Meyer C, Masclaux-Daubresse C (2010) Senescence and death of plant organs: nutrient recycling and developmental regulation. C R Biol 333:382–391

Guo Y, Gan S (2006) AtNAP, a NAC family transcription factor, has an important role in leaf senescence. Plant J 46:601–612

Guo YF, Gan SS (2012) Convergence and divergence in gene expression profiles induced by leaf senescence and 27 senescence-promoting hormonal, pathological and environmental stress treatments. Plant, Cell Environ 35:644–655

Ha S, Vankova R, Yamaguchi-Shinozaki K, Shinozaki K, Tran L-SP (2012) Cytokinins: metabolism and function in plant adaptation to environmental stresses. Trends Plant Sci 17:172–179

Hasan SA, Hayat S, Ahmad A (2011) Brassinosteroids protect photosynthetic machinery against the cadmium induced oxidative stress in two tomato cultivars. Chemosphere 84:1446–1451

Hayat Q, Hayat S, Irfan M, Ahmad A (2010) Effect of exogenous salicylic acid under changing environment: a review. Environ Exp Bot 68:14–25

He J, Xu R, Zhao Y, He JJ, Xu RJ, Zhao YJ (1996) Enhancement of senescence by epibrassinolide in leaves of mung bean seedling. Acta Phytophysiol Sinica 22:58–62

He Y, Tang W, Swain JD, Green AL, Jack TP, Gan S (2001) Networking senescence-regulating pathways by using *Arabidopsis* enhancer trap lines. Plant Physiol 126:707–716

He YH, Fukushige H, Hildebrand DF, Gan SS (2002) Evidence supporting a role of jasmonic acid in *Arabidopsis* leaf senescence. Plant Physiol 128:876–884

He P, Osaki M, Takebe M, Shinano T, Wasaki J (2005) Endogenous hormones and expression of senescence-related genes in different senescent types of maize. J Exp Bot 56:1117–1128

He K, Gou X, Yuan T, Lin H, Asami T, Yoshida S, Russell SD, Li J (2007) BAK1 and BKK1 regulate brassinosteroid-dependent growth and brassinosteroid-independent cell-death pathways. Curr Biol 17:1109–1115

Hensel LL, Grbic V, Baumgarten DA, Bleecker AB (1993) Developmental and age-related processes that influence the longevity and senescence of photosynthetic tissues in Arabidoposis. Plant Cell 5:553–564

Hirayama T, Shinozaki K (2010) Research on plant abiotic stress responses in the post-genome era: past, present and future. Plant J 61:1041–1052

Husar S, Berthiller F, Fujioka S, Rozhon W, Khan M, Kalaivanan F, Elias L, Higgins GS, Li Y, Schuhmacher R, Krska R, Seto H, Vaistij FE, Bowles D, Poppenberger B (2011) Overexpression of the UGT73C6 alters brassinosteroid glucoside formation in *Arabidopsis thaliana*. BMC Plant Biol 11:51

Itzhaki H, Borochov A, Mayak S (1990) Age-related-changes in petal membranes from attached and detached rose flowers. Plant Physiol 94:1233–1236

Jing HC, Sturre MJG, Hille J, Dijkwel PP (2002) *Arabidopsis* onset of leaf death mutants identify a regulatory pathway controlling leaf senescence. Plant J 32:51–63

Jing HC, Schippers JH, Hille J, Dijkwel PP (2005) Ethylene-induced leaf senescence depends on age-related changes and OLD genes in *Arabidopsis*. J Exp Bot 56:2915–2923

Jung C, Lyou SH, Yeu S, Kim MA, Rhee S, Kim M, Lee JS, Choi YD, Cheong JJ (2007) Microarray-based screening of jasmonate-responsive genes in *Arabidopsis thaliana*. Plant Cell Rep 26:1053–1063

Kang J-H, Wang L, Giri A, Baldwin IT (2006) Silencing threonine deaminase and JAR4 in *Nicotiana attenuata* impairs jasmonic acid-isoleucine-mediated defenses against *Manduca sexta*. Plant Cell 18:3303–3320

Kant S, Bi YM, Zhu T, Rothstein SJ (2009) SAUR39, a small auxin-up RNA gene, acts as a negative regulator of auxin synthesis and transport in rice. Plant Physiol 151:691–701

Kato Y, Murakami S, Yamamoto Y, Chatani H, Kondo Y, Nakano T, Yokota A, Sato F (2004) The DNA-binding protease, CND41, and the degradation of ribulose-1,5-bisphosphate carboxylase/oxygenase in senescent leaves of tobacco. Planta 220:97–104

Kelly MO, Davies PJ (1986) Genetic and photoperiodic control of the relative rates of reproductive and vegetative development in peas. Ann Bot 58:13–21

Kelly MO, Davies PJ (1988) Photoperiodic and genetic-control of carbon partitioning in peas and its relationship to apical senescence. Plant Physiol 86:978–982

Kieber JJ, Rothenberg M, Roman G, Feldmann KA, Ecker JR (1993) CTR1, a negative regulator of the ethylene response pathway in *Arabidopsis*, encodes a member of the Raf family of protein kinases. Cell 72:427–441

Kim HJ, Ryu H, Hong SH, Woo HR, Lim PO, Lee IC, Sheen J, Nam HG, Hwang I (2006) Cytokinin-mediated control of leaf longevity by AHK3 through phosphorylation of ARR2 in *Arabidopsis*. Proc Natl Acad Sci USA 103:814–819

Kim JH, Woo HR, Kim J, Lim PO, Lee IC, Choi SH, Hwang D, Nam HG (2009) Trifurcate feed-forward regulation of age-dependent cell death involving miR164 in Arabidopsis. Science 323:1053

Kim JH, Chung KM, Woo HR (2011a) Three positive regulators of leaf senescence in *Arabidopsis*, ORE1, ORE3 and ORE9, play roles in crosstalk among multiple hormone-mediated senescence pathways. Genes Genomics 33:373–381

Kim JI, Murphy AS, Baek D, Lee SW, Yun DJ, Bressan RA, Narasimhan ML (2011b) YUCCA6 over-expression demonstrates auxin function in delaying leaf senescence in *Arabidopsis thaliana*. J Exp Bot 62:3981–3992

Kinoshita N, Berr A, Belin C, Chappuis R, Nishizawa NK, Lopez-Molina L (2010) Identification of growth insensitive to ABA3 (gia3), a recessive mutation affecting ABA Signaling for the control of early post-germination growth in *Arabidopsis thaliana*. Plant Cell Physiol 51:239–251

Koornneef M, Alonso-Blanco C, Peeters AJM, Soppe W (1998) Genetic control of flowering time in arabidopsis. Annu Rev Plant Physiol Plant Mol Biol 49:345–370

Krishna P (2003) Brassinosteroid-mediated stress responses. J Plant Growth Regul 22:289–297

Lara MEB, Garcia MCG, Fatima T, Ehness R, Lee TK, Proels R, Tanner W, Roitsch T (2004) Extracellular invertase is an essential component of cytokinin-mediated delay of senescence. Plant Cell 16:1276–1287

Lee SC, Luan S (2012) ABA signal transduction at the crossroad of biotic and abiotic stress responses. Plant, Cell Environ 35:53–60

Lee IC, Hong SW, Whang SS, Lim PO, Nam HG, Koo JC (2011) Age-dependent action of an ABA-inducible receptor kinase, RPK1, as a positive regulator of senescence in *Arabidopsis* leaves. Plant Cell Physiol 52:651–662

Li J, Yu K, Wei J, Ma Q, Wang B, Yu D (2010) Gibberellin retards chlorophyll degradation during senescence of *Paris polyphylla*. Biol Plant 54:395–399

Lim PO, Kim HJ, Gil Nam H (2007) Leaf senescence. Annu Rev Plant Biol 58:115–136

Lim PO, Lee IC, Kim J, Kim HJ, Ryu JS, Woo HR, Nam HG (2010) Auxin response factor 2 (ARF2) plays a major role in regulating auxin-mediated leaf longevity. J Exp Bot 61:1419–1430

Lumba S, Tsuchiya Y, Delmas F, Hezky J, Provart NJ, Shi Lu Q, McCourt P, Gazzarrini S (2012) The embryonic leaf identity gene FUSCA3 regulates vegetative phase transitions by negatively modulating ethylene-regulated gene expression in *Arabidopsis*. BMC Biol 10:8

Mauch-Mani B, Mauch F (2005) The role of abscisic acid in plant–pathogen interactions. Curr Opin Plant Biol 8:409–414

McCabe MS, Garratt LC, Schepers F, Jordi WJRM, Stoopen GM, Davelaar E, van Rhijn JHA, Power JB, Davey MR (2001) Effects of PSAG12-IPT gene expression on development and senescence in transgenic lettuce. Plant Physiol 127:505–516

Melotto M, Underwood W, Koczan J, Nomura K, He SY (2006) Plant stomata function in innate immunity against bacterial invasion. Cell 126:969–980

Merewitz EB, Gianfagna T, Huang BR (2010) Effects of SAG12-ipt and HSP18.2-ipt expression on cytokinin production, root growth, and leaf senescence in creeping bentgrass exposed to drought stress. J Am Soc Hort Sci 135:230–239

Merewitz EB, Gianfagna T, Huang B (2011) Protein accumulation in leaves and roots associated with improved drought tolerance in creeping bentgrass expressing an ipt gene for cytokinin synthesis. J Exp Bot 62:5311–5333

Merewitz EB, Du H, Yu W, Liu Y, Gianfagna T, Huang B (2012) Elevated cytokinin content in ipt transgenic creeping bentgrass promotes drought tolerance through regulating metabolite accumulation. J Exp Bot 63:1315–1328

Mewis I, Appel HM, Hom A, Raina R, Schultz JC (2005) Major signaling pathways modulate *Arabidopsis* glucosinolate accumulation and response to both phloem-feeding and chewing insects. Plant Physiol 138:1149–1162

Miao Y, Laun T, Zimmermann P, Zentgraf U (2004) Targets of the WRKY53 transcription factor and its role during leaf senescence in Arabidopsis. Plant Mol Biol 55:853–867

Miceli F, Craftsbrandner SJ, Egli DB (1995) Physical restriction of pod growth alters development of soybean plants. Crop Sci 35:1080–1085

Morris K, Mackerness SAH, Page T, John CF, Murphy AM, Carr JP, Buchanan-Wollaston V (2000) Salicylic acid has a role in regulating gene expression during leaf senescence. Plant J 23:677–685

Nakashita H, Yasuda M, Nitta T, Asami T, Fujioka S, Arai Y, Sekimata K, Takatsuto S, Yamaguchi I, Yoshida S (2003) Brassinosteroid functions in a broad range of disease resistance in tobacco and rice. Plant J 33:887–898

Noh YS, Amasino RM (1999) Identification of a promoter region responsible for the senescence-specific expression of SAG12. Plant Mol Biol 41:181–194

Nooden LD (1988) The phenomena of senescence and aging. In: Nooden LD, Leopold AC (eds) Senescence and aging in plants. Academic Press, San Diego

Ogawa T, Pan L, Kawai-Yamada M, Yu LH, Yamamura S, Koyama T, Kitajima S, Ohme-Takagi M, Sato F, Uchimiya H (2005) Functional analysis of Arabidopsis ethylene-responsive element binding protein conferring resistance to Bax and abiotic stress-induced plant cell death. Plant Physiol 138:1436–1445

Oh SA, Park JH, Lee GI, Paek KH, Park SK, Nam HG (1997) Identification of three genetic loci controlling leaf senescence in *Arabidopsis thaliana*. Plant J 12:527–535

Ori N, Juarez MT, Jackson D, Yamaguchi J, Banowetz GM, Hake S (1999) Leaf senescence is delayed in tobacco plants expressing the maize homeobox gene knotted1 under the control of a senescence-activated promoter. Plant Cell 11:1073–1080

Pandey S, Gupta K, Mukherjee A (2007) Impact of cadmium and lead on *Catharanthus roseus*-A phytoremediation study. J Environ Biol 28:655–662

Park JH, Halitschke R, Kim HB, Baldwin IT, Feldmann KA, Feyereisen R (2002) A knock-out mutation in allene oxide synthase results in male sterility and defective wound signal transduction in Arabidopsis due to a block in jasmonic acid biosynthesis. Plant J 31:1–12

Perilli S, Moubayidin L, Sabatini S (2010) The molecular basis of cytokinin function. Curr Opin Plant Biol 13:21–26

Philosoph-Hadas S, Hadas E, Aharoni N (1993) Characterization and use in elisa of a new monoclonal-antibody for quantitation of abscisic-acid in senescing rice leaves. Plant Growth Regul 12:71–78

Qin F, Kodaira KS, Maruyama K, Mizoi J, Tran LSP, Fujita Y, Morimoto K, Shinozaki K, Yamaguchi-Shinozaki K (2011) SPINDLY, a Negative Regulator of Gibberellic Acid Signaling, Is Involved in the Plant Abiotic Stress Response. Plant Physiol 157:1900–1913

Quirino BF, Normanly J, Amasino RM (1999) Diverse range of gene activity during *Arabidopsis thaliana* leaf senescence includes pathogen-independent induction of defense-related genes. Plant Mol Biol 40:267–278

Rao SSR, Vidya Vardhini B, Sujatha E, Anuradha S (2002) Brassinosteroids: a new class of phytohormones. Curr Sci 82:1239–1245

Rivero RM, Gimeno J, Van Deynze A, Walia H, Blumwald E (2010) Enhanced Cytokinin Synthesis in Tobacco Plants Expressing P(SARK):IPT Prevents the Degradation of Photosynthetic Protein Complexes During Drought. Plant Cell Physiol 51: 1929–1941

Robatzek S, Somssich IE (2001) A new member of the *Arabidopsis* WRKY transcription factor family, AtWRKY6, is associated with both senescence-and defence-related processes. Plant J 28:123–133

Robert-Seilaniantz A, Grant M, Jones JDG (2011) Hormone crosstalk in plant disease and defense: more than just JASMONATE-SALICYLATE Antagonism. In: NK VanAlfen, G Bruening, JE Leach (eds) Annual Review of Phytopathology 49:317–343

Rodrigues C, Vandenberghe LPD, de Oliveira J, Soccol CR (2011) New perspectives of gibberellic acid production: a review. Crit Rev Biotechnol 32:263–273

Saglam-Cag S (2007) The effect of epibrassinolide on senescence in wheat leaves. Biotechnol Biotechnol Equip 21:63–65

Schenk PM, Kazan K, Rusu AG, Manners JM, Maclean DJ (2005) The *SEN1* gene of *Arabidopsis* is regulated by signals that link plant defence responses and senescence. Plant Physiol Biochem 43:997–1005

Schippers JHM, Jing HC, Hille J, Dijkwel PP (2007) Developmental and hormonal control of leaf senescence. In: Gan S (ed) Senescence Processes in Plants. Blackwell, Oxford, pp 145–170

Schippers JHM, Nunes-Nesi A, Apetrei R, Hille J, Fernie AR, Dijkwel PP (2008) The *Arabidopsis* onset of leaf death5 Mutation of quinolinate synthase affects nicotinamide adenine dinucleotide biosynthesis and causes early ageing. Plant Cell 20:2909–2925

Schmulling T, Werner T, Riefler M, Krupkova E, Manns IBY (2003) Structure and function of cytokinin oxidase/dehydrogenase genes of maize, rice, Arabidopsis and other species. J Plant Res 116:241–252

Schommer C, Palatnik JF, Aggarwal P, Chetelat A, Cubas P, Farmer EE, Nath U, Weigel D (2008) Control of jasmonate biosynthesis and senescence by miR319 targets. PLoS Biol 6:e230

Seltmann MA, Stingl NE, Lautenschlaeger JK, Krischke M, Mueller MJ, Berger S (2010) Differential Impact of lipoxygenase 2 and jasmonates on natural and stress-induced senescence in *Arabidopsis*. Plant Physiol 152:1940–1950

Shakirova FM, Panov VE, Clark PF (2007) New records of the Chinese mitten crab, *Eriocheir sinensis* H. Milne Edwards, 1853, from the Volga River, Russia. Aquat Invasions 2: 169–173

Shan X, Wang J, Chua L, Jiang D, Peng W, Xie D (2011) The role of *Arabidopsis* Rubisco activase in jasmonate-induced leaf senescence. Plant Physiol 155:751–764

Sharabi-Schwager M, Lers A, Samach A, Guy CL, Porat R (2010) Overexpression of the CBF2 transcriptional activator in *Arabidopsis* delays leaf senescence and extends plant longevity. J Exp Bot 61:261–273

Shi Y, Tian S, Hou L, Huang X, Zhang X, Guo H, Yang S (2012) Ethylene signaling negatively regulates freezing tolerance by repressing expression of CBF and type-A ARR genes in *Arabidopsis*. Plant Cell 24:2578–2595

Skirycz A, Claeys H, De Bodt S, Oikawa A, Shinoda S, Andriankaja M, Maleux K, Eloy NB, Coppens F, Yoo SD, Saito K, Inze D (2011) Pause-and-stop: the effects of osmotic stress on cell proliferation during early leaf development in *Arabidopsis* and a role for ethylene signaling in cell cycle arrest. Plant Cell 23:1876–1888

Sklensky DE, Davies PJ (2011) Resource partitioning to male and female flowers of *Spinacia oleracea* L. in relation to whole-plant monocarpic senescence. J Exp Bot 62:4323–4336

Stintzi A, Weber H, Reymond P, Farmer EE (2001) Plant defense in the absence of jasmonic acid: the role of cyclopentenones. Proc Natl Acad Sci USA 98:12837

Swartzberg D, Hanael R, Granot D (2011) Relationship between hexokinase and cytokinin in the regulation of leaf senescence and seed germination. Plant Biol 13:439–444

Szekeres M, Nemeth K, KonczKalman Z, Mathur J, Kauschmann A, Altmann T, Redei GP, Nagy F, Schell J, Koncz C (1996) Brassinosteroids rescue the deficiency of CYP90, a cytochrome P450, controlling cell elongation and de-etiolation in arabidopsis. Cell 85:171–182

Ueda J, Kato J, Yamane H, Takahashi N (1981) Inhibitory effect of methyl jasmonate and its related compounds on kinetin-induced retardation of oat leaf senescence. Physiol Plant 52:305–309

Ülker B, Shahid Mukhtar M, Somssich IE (2007) The WRKY70 transcription factor of *Arabidopsis* influences both the plant senescence and defense signaling pathways. Planta 226:125–137

van der Graaff E, Schwacke R, Schneider A, Desimone M, Flugge UI, Kunze R (2006) Transcription analysis of arabidopsis membrane transporters and hormone pathways during developmental and induced leaf senescence. Plant Physiol 141:776–792

Van Zhong G, Burns JK (2003) Profiling ethylene-regulated gene expression in *Arabidopsis thaliana* by microarray analysis. Plant Mol Biol 53:117–131

Vlot AC, Dempsey DMA, Klessig DF (2009) Salicylic acid, a multifaceted hormone to combat disease. Annu Rev Phytopathol 47:177–206

von Saint Paul V, Zhang W, Kanawati B, Geist B, Faus-Kessler T, Schmitt-Kopplin P, Schaffner AR (2011) The *Arabidopsis* glucosyltransferase UGT76B1 conjugates isoleucic acid and modulates plant defense and senescence. Plant Cell 23:4124–4145

Wang KLC, Li H, Ecker JR (2002) Ethylene biosynthesis and signaling networks. Plant Cell 14:S131–S151

Wasternack C (2007) Jasmonates: an update on biosynthesis, signal transduction and action in plant stress response, growth and development. Ann Bot 100:681–697

Weaver LM, Gan SS, Quirino B, Amasino RM (1998) A comparison of the expression patterns of several senescence-associated genes in response to stress and hormone treatment. Plant Mol Biol 37:455–469

Werner T, Motyka V, Laucou V, Smets R, Van Onckelen H, Schmülling T (2003) Cytokinin-deficient transgenic *Arabidopsis* plants show multiple developmental alterations indicating opposite functions of cytokinins in the regulation of shoot and root meristem activity. Plant Cell 15:2532–2550

Wintermans J, De Mots A (1965) Spectrophotometric characteristics of chlorophylls *a* and *b* and their phenophytins in ethanol. Biochimica et Biophysica Acta (BBA)-Biophysics including Photosynthesis 109:448–453

Wittenbach VA (1982) Effect of pod removal on leaf senescence in soybeans. Plant Physiol 70:1544–1548

Woltering EJ, Vandoorn WG (1988) Role of ethylene in senescence of petals—morphological and taxonomical relationships. J Exp Bot 39:1605–1616

Woodward AW, Bartel B (2005) Auxin: regulation, action, and interaction. Ann Bot 95:707–735

Xiao S, Chye ML (2011) Overexpression of *Arabidopsis* ACBP3 enhances NPR1-dependent plant resistance to Pseudomonas syringe pv tomato DC3000. Plant Physiol 156:2069–2081

Xiao S, Dai L, Liu F, Wang Z, Peng W, Xie D (2004) COS1: an Arabidopsis coronatine insensitive1 suppressor essential for regulation of jasmonate-mediated plant defense and senescence. Plant Cell 16:1132–1142

Xiao S, Gao W, Chen QF, Chan SW, Zheng SX, Ma J, Wang M, Welti R, Chye ML (2010) Overexpression of *Arabidopsis* Acyl-CoA binding protein ACBP3 promotes starvation-induced and age-dependent leaf senescence. Plant Cell 22:1463–1482

Xu J, Li Y, Wang Y, Liu H, Lei L, Yang H, Liu G, Ren D (2008) Activation of MAPK kinase 9 induces ethylene and camalexin biosynthesis and enhances sensitivity to salt stress in *Arabidopsis*. J Biol Chem 283:26996–27006

Yan J, Zhang C, Gu M, Bai Z, Zhang W, Qi T, Cheng Z, Peng W, Luo H, Nan F, Wang Z, Xie D (2009) The *Arabidopsis* CORONATINE INSENSITIVE1 protein is a Jasmonate receptor. Plant Cell 21:2220–2236

Yang JC, Zhang JH, Wang ZQ, Zhu QS, Liu LJ (2002) Abscisic acid and cytokinins in the root exudates and leaves and their relationship to senescence and remobilization of carbon reserves in rice subjected to water stress during grain filling. Planta 215:645–652

Yang SD, Seo PJ, Yoon HK, Park CM (2011) The Arabidopsis NAC transcription factor VNI2 integrates abscisic acid signals into leaf senescence via the COR/RD genes. Plant Cell 23:2155–2168

Yin YH, Wang ZY, Mora-Garcia S, Li JM, Yoshida S, Asami T, Chory J (2002) BES1 accumulates in the nucleus in response to brassinosteroids to regulate gene expression and promote stem elongation. Cell 109:181–191

Yoshimoto K, Jikumaru Y, Kamiya Y, Kusano M, Consonni C, Panstruga R, Ohsumi Y, Shirasu K (2009) Autophagy negatively regulates cell death by controlling NPR1-dependent salicylic acid signaling during senescence and the innate immune response in *Arabidopsis*. Plant Cell 21:2914–2927

Yu K, Wei J, Ma Q, Yu D, Li J (2009a) Senescence of aerial parts is impeded by exogenous gibberellic acid in herbaceous perennial Paris polyphylla. J Plant Physiol 166:819–830

Yu K, Wang Y, Wei J, Ma Q, Yu D, Li J (2009b) Improving rhizome yield and quality of Paris polyphylla through gibberellic acid-induced retardation of senescence of aerial parts. Plant Signal Behav 4:413

Zentgraf U (2007) Oxidative stress and leaf senescence. In: Gan S (ed) Annual plant reviews: senescence processes in plants. Blackwell Publishing Ltd, Oxford, UK, p 26

Zhang K, Gan SS (2012) An abscisic acid-AtNAP transcription factor-SAG113 protein phosphatase 2C regulatory chain for controlling dehydration in senescing Arabidopsis leaves. Plant Physiol 158:961–969

Zhang Y, Liang C, Xu Y, Gianfagna T, Huang B (2010) Effects of ipt gene expression on leaf senescence induced by nitrogen or phosphorus deficiency in creeping bentgrass. J Am Soc Hort Sci 135:108–115

Zhang K, Xia X, Zhang Y, Gan SS (2012) An ABA-regulated and golgi-localized protein phosphatase controls water loss during leaf senescence in *Arabidopsis*. Plant J 69:667–678

Zhao Y, Christensen SK, Fankhauser C, Cashman JR, Cohen JD, Weigel D, Chory J (2001) A role for flavin monooxygenase-like enzymes in auxin biosynthesis. Science 291:306–309