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REVIEW PAPER

Salicylic acid beyond defence: its role in plant growth and development

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Abstract

In recent years salicylic acid (SA) has been the focus of intensive research due to its function as an endogenous signal mediating local and systemic plant defence responses against pathogens. It has also been found that SA plays a role during the plant response to abiotic stresses such as drought, chilling, heavy metal toxicity, heat, and osmotic stress. In this sense, SA appears to be, just like in mammals, an 'effective therapeutic agent' for plants. Besides this function during biotic and abiotic stress, SA plays a crucial role in the regulation of physiological and biochemical processes during the entire lifespan of the plant. The discovery of its targets and the understanding of its molecular modes of action in physiological processes could help in the dissection of the complex SA signalling network, confirming its important role in both plant health and disease. Here, the evidence that supports the role of SA during plant growth and development is reviewed by comparing experiments performed by exogenous application of SA with analysis of genotypes affected by SA levels and/or perception.

Key words: Development, growth, phytohormones, salicylic acid.

Introduction

Salicylic acid (SA) is a phenolic compound (Fig. 1) which, despite its broad distribution in plants, has basal levels differing widely among species, with up to 100-fold differences having been recorded (Raskin *et al.*, 1990). This disparity can be observed within members of the same family. For example, in the Solanaceae, whereas tobacco (*Nicotiana tabacum*) contains low basal levels of SA [<100 ng g^{-1} fresh weight (FW)] in leaves (Yalpani *et al.*, 1991; Malamy *et al.*, 1992), potato (*Solanum tuberosum*) might contain up to 10 μg of total SA g^{-1} FW (Coquoz *et al.*, 1998; Navarre and Mayo, 2004). In the model plant *Arabidopsis thaliana*, basal levels of total SA range from 0.250 μg to 1 μg g^{-1} FW (Nawrath and Métraux, 1999; Wildermuth *et al.*, 2001; Brodersen *et al.*, 2005). SA is synthesized through two distinct and compartmentalized pathways that employ different precursors: the phenylpropanoid route in the cytoplasm initiates from phenylalanine, and the isochorismate pathway takes place in the chloroplast. Most of the SA synthesized in plants is glucosylated and/or methylated (Fig. 1). Glucose conjugation at the hydroxyl group of SA results in formation of the SA glucoside [SA 2-*O*-β-D-glucoside] as a major conjugate, whereas glucose conjugation at the SA carboxyl group produces the SA glucose ester in minor amounts (Fig. 1). These conjugation reactions are catalysed by cytosolic SA glucosyltransferases that are induced by SA application or pathogen attack in tobacco and *Arabidopsis* plants (Lee and Raskin, 1999; Song, 2006). SAG is actively transported from the cytosol into the vacuole of soybean and tobacco cells, where it may function as an inactive storage form that can release free SA (Dean and Mills, 2004; Dean *et al.*, 2005). Interestingly, SA is also converted to methyl salicylate (MeSA) by an SA carboxyl methyltransferase, and this volatile derivate is an important long-distance signal in tobacco and *Arabidopsis* systemic acquired resistance (Shulaev *et al.*, 1997; Chen *et al.*, 2003; Park *et al.*,

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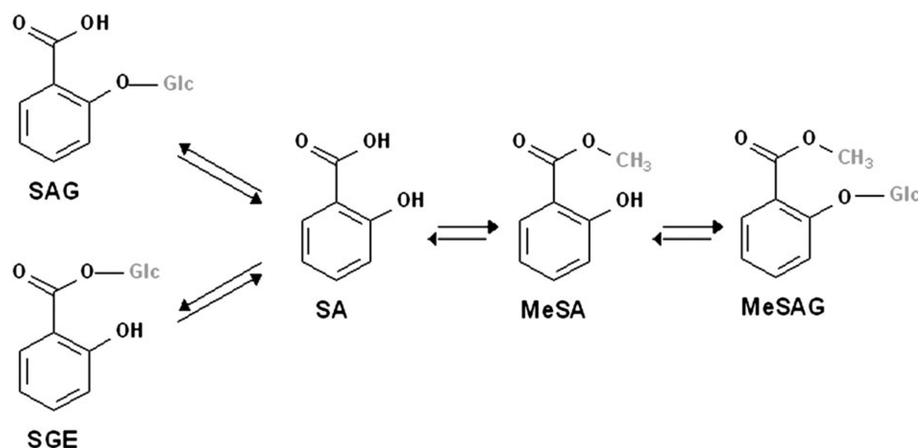

Fig. 1. Structures of salicylic acid and its derivates. SA, salicylic acid; SAG, SA 2-*O*-β-D-glucoside; SGE, SA glucose ester; MeSA, methyl salicylate; MeSAG, methyl salicylate 2-*O*-β-D-glucose.

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2007; Vlot *et al.*, 2008). MeSA can be further glucosylated to produce MeSA 2-*O*-β-D-glucose, but this SA-conjugated form is not stored in the vacuole (Dean *et al.*, 2005). The reader is referred to excellent reviews dealing with the enzymes and regulation of these biosynthetic routes (Klessig and Malamy, 1994; Lee *et al.*, 1995; Shah, 2003; Chen *et al.*, 2009; Vlot *et al.*, 2009).

SA has been recognized as a regulatory signal mediating plant response to abiotic stresses such as drought (Munné-Bosch and Peñuelas, 2003; Chini *et al.*, 2004), chilling (Janda *et al.*, 1999; Kang and Saltveit 2002), heavy metal tolerance (Metwally *et al.*, 2003; Yang *et al.*, 2003; Freeman *et al.*, 2005), heat (Larkindale and Knight, 2002; Larkindale *et al.*, 2005), and osmotic stress (Borsani *et al.*, 2001). However, most of the research on this hormone has focused on its role in the local and systemic response against microbial pathogens, and on defining the transduction pathway leading to gene expression induced by SA. Again, there are several reviews on this subject (Klessig and Malamy, 1994; Durner *et al.*, 1997; Shah, 2003; Durrant and Dong, 2004; Vlot *et al.*, 2009).

The focus of this review is on the role of SA in plant growth and development as there is evidence that this hormone regulates processes such as seed germination, vegetative growth, photosynthesis, respiration, thermogenesis, flower formation, seed production, senescence, and a type of cell death that is not associated with the hypersensitive response. In addition, SA could contribute to maintaining cellular redox homeostasis through the regulation of antioxidant enzymes activity (Durner and Klessig, 1995, 1996; Slaymaker *et al.*, 2002) and induction of the alternative respiratory pathway (Moore *et al.*, 2002), and to regulating gene expression by inducing an RNA-dependent RNA polymerase that is important for post-transcriptional gene silencing (Xie *et al.*, 2001).

This review summarizes the recent advances in the understanding of the physiological functions of SA, and relevant insights regarding SA mechanisms that control these events are highlighted.

SA-regulated physiological functions

Seed germination

Effect of exogenous SA on seed germination. Environmental factors and interactions between the plant hormones abscisic acid (ABA), jasmonic acid (JA), gibberellins (GAs), ethylene (ET), brassinosteroids (BRs), auxins (AUXs), and cytokinins (CKs) regulate seed germination. The role of SA in seed germination has been controversial as there are conflicting reports suggesting that it can either inhibit germination or increase seed vigour. The reported contradictory effects can be related to the SA concentrations employed. In *A. thaliana*, SA concentrations >1 mM delay or even inhibit germination (Rajjou *et al.*, 2006). In barley, doses >0.250 mM SA inhibit seed germination (Xie *et al.*, 2007), while maize germination is completely inhibited by SA doses ranging from 3 mM to 5 mM (Guan and Scandalios, 1995). SA's effect as a negative regulator of seed germination is presumably due to an SA-induced oxidative stress. In *Arabidopsis* plants treated with SA (1–5 mM), hydrogen peroxide (H_2O_2) levels increase up to 3-fold as a result of increased activities of Cu, Zn-superoxide dismutase and inactivation of the H_2O_2-degrading enzymes catalase and ascorbate peroxidase (Rao *et al.*, 1997).

SA improvement of seed germination under abiotic stress. Interestingly, when low doses are applied exogenously, SA significantly improves *Arabidopsis* seed germination and seedling establishment under different abiotic stress conditions (Rajjou *et al.*, 2006; Alonso-Ramírez *et al.*, 2009). Under salt stress (100–150 mM NaCl) only 50% of *Arabidopsis* seeds germinate, but in the presence of SA (0.05–0.5 mM) seed germination increases to 80%. Exogenous application of SA also partially reverses the inhibitory effect of oxidative (0.5 mM paraquat) and heat stress (50 °C for 3 h) on seed germination (Alonso-Ramírez *et al.*, 2009). These observations are in agreement with the delayed germination phenotype observed in the *Arabidopsis sid2* mutant under high salinity (Alonso-Ramírez *et al.*, 2009).

This mutant is affected in the isochorismate synthase gene and thus contains low SA levels (Table 1). However, *NahG* transgenic lines expressing a bacterial salicylate hydroxylase also have lower SA levels than wild-type plants, but germination is not affected by high salinity (Borsani *et al.*, 2001). This apparent discrepancy is due to the antioxidant effect of catechol, the product of the salicylate hydroxylase that accumulates in the *NahG* seeds and seedlings (Lee *et al.*, 2010). Thus the germination promotion effect of SA under high salinity conditions is by reducing oxidative damage. Moreover, proteomic analyses showed that two superoxide dismutases are induced by SA in *Arabidopsis* germinating seeds, which might contribute to an enhanced antioxidant capacity (Rajjou *et al.*, 2006). SA treatment (0.5 mM for 24 h) also causes a strong up-regulation of translation initiation and elongation factors, proteases, and two subunits of the 20S proteasome, supporting the hypothesis that SA improves seed germination by promoting the synthesis of proteins that are essential for germination, and the mobilization or degradation of seed proteins accumulated during seed maturation. In addition, the biosynthesis of several enzymes involved in metabolic pathways such as the glyoxylate cycle, the pentose phosphate pathway, glycolysis, and gluconeogenesis is also strongly activated by SA, suggesting that SA promotes the release from a quiescence state to the establishment of a vigorous seedling (Rajjou *et al.*, 2006).

SA cross-talk with ABA and GAs during germination. During this early developmental stage, a complex interaction between SA and both ABA and GAs determines germination outcome. In *Arabidopsis*, GAs have a role in SA biosynthesis and the SA pathway. Imbibition of 50 μM GA_3 by seeds for 24 h, as well as the overexpression of a GA-stimulated gene from beechnut (*FcGASA*$_4$) in *Arabidopsis* plants, induces a 2-fold increase in SA levels compared with seeds imbibed in water and wild-type plants. Furthermore, increased expression of the *ICS1* (*isochorismate synthase*) and *NPR1* (*nonexpressor of PR-1*) genes, involved in SA biosynthesis and perception, respectively, is observed in *FcGASA*$_4$-overexpressing lines, and in Col-0 seedlings grown in the presence of GA_3. Interestingly, exogenous SA (50 μM) partially rescues seed germination in the GA-deficient mutant *ga1-3*, whereas exogenous GA_3 (50 μM) slightly improves the germination of the SA-deficient *sid2* mutants under 150 mM NaCl stress (Alonso-Ramírez *et al.*, 2009). Although these results suggest a synergistic relationship between SA and GA, an antagonistic relationship was observed during barley germination that could be explained by the addition of a higher dose of SA. The inhibition of barley seed germination and post-germination growth by SA is accompanied by suppression of GA-induced α-amylase (*Amy32b*) expression through induction of a WRKY repressor (HvWRKY38). Expression of *HvWRKY38* in aleurone cells is down-regulated by GAs, but up-regulated by SA and ABA, so this transcription factor might serve as a converging node of the SA and ABA signal pathways involved in suppressing GA-induced seed germination (Xie *et al.*, 2007). Additional evidence supporting the cross-talk between ABA and SA signalling is the increased synthesis of ABA-regulated proteins, such as late embryogenesis abundant (LEA) proteins, dehydrins, and heat shock proteins, in *Arabidopsis* seeds germinated in the presence of 0.5 mM SA (Rajjou *et al*, 2006).

The ubiquitin–proteasome system (UPS) pathway in hormone signalling integration. Because GAs and ABA have opposing roles in the regulation of germination, SA might act as a rheostat contributing with both hormones. Recent biochemical evidence points to the UPS as a mechanism to balance the antagonic control of seed germination between ABA and GAs (Zentella *et al.*, 2007; Piskurewics *et al.*, 2008). In fact, in the UPS, many hormone signalling pathways converge (Santner and Estelle, 2009; Vierstra, 2009; Santner and Estelle, 2010) thus influencing many aspects of plant growth and development. Recently it was found that NPR1, the key transducer of SA signalling in plant defence responses, associates in the nucleus with Cullin3-based E3 ligases and other components of the COP9 signalosome, which controls proteasomal degradation. Moreover, NPR1 proteasome-mediated turnover is promoted by SA-induced phosphorylation of the Ser11 and Ser15 residues (Spoel *et al.*, 2009). Initially, it was found that the activity of NPR1 is regulated by its subcellular localization, because the transcriptional co-activator is predominantly sequestered in the cytoplasm as an oligomer, but in pathogen-infected cells SA accumulation promotes partial reduction of the NPR1 oligomer to a monomer, which is targeted to the nucleus by a bipartite nuclear localization sequence (Mou *et al.*, 2003). Interestingly, NPR1 also enters the nucleus when basal SA levels are low and no infection is occurring, and it has been suggested that it may regulate additional genes. If this scenario could be confirmed, it would be interesting to analyse the contribution of these genes to the regulation of germination, plant growth, and development.

It is also worth mentioning the role of the DELLA proteins as potential integrators of phytohormone signalling in the regulation of germination, cell redox state, growth, and stress responses (reviewed by Smirnoff and Grant, 2008; Grant and Jones, 2009; Harberd *et al.*, 2009). DELLA proteins are repressors of GA signalling and, in turn, GA derepresses its pathway by promoting proteasomal degradation of the DELLA protein RGA (Dill *et al.*, 2004). There are five DELLA genes in *Arabidopsis*: *GAI* (GA insensitive), *RGA* (repressor of GA1-3), *RGL1*, *RGL2*, and *RGL3* (RGA-like). *RGL2* is considered to be the main DELLA factor repressing germination (Lee *et al.*, 2002; Tyler *et al.*, 2004), although the other DELLA genes also contribute to regulate germination (Cao *et al.*, 2005). Recently, it was found that *RGL2* expression is strongly stimulated by ABA, and that RGL2 protein is necessary to elevate endogenous ABA and *ABI5* (another germination repressor) expression levels, specifically when GA levels are low (in *ga1-3* mutants, or in the presence of GA synthesis inhibitors). Moreover, RGL2 is necessary to repress testa

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Table 1. Examples of *Arabidopsis* genotypes with altered SA levels showing abnormal growth phenotypes

Transgene or mutation	Gene function	Effect on SA levels	Growth phenotype	References
NahG	Bacterial salicylate hydroxylase	**Reduction about 2- to 4-fold in leaves of healthy plants, and up to 20-fold in defence response against pathogens.**	Increased growth (leaf rosette biomass at early stages of reproduction 1.7-fold more than wild type).	Abreu and Munné-Bosch (2009)
			Faster growth rate at low temperature (4 °C) associated with enlarged cell size, extensive endoreduplication, and increased expression of CycD3.	Scott *et al.* (2004); Xia *et al.* (2009)
sid2	Isochorismate synthase		Increased growth (leaf rosette biomass at early stages of reproduction 1.7-fold more than wild type).	Abreu and Munné-Bosch (2009)
eds5/sid1	Multidrug and toxin extrusion transporter		Slightly less growth than wild type at 23 °C, but at 5 °C its growth is significantly greater compared with the wild type and very similar to *NahG*.	Nawrath and Métraux (1999); Scott *et al.* (2004)
acd5	Ceramide kinase	**Accumulation (from 2- to 200-fold of total SA)**	Reduced stature compared with the wild type, which is totally or partially suppressed by *NahG* and *npr1*, respectively.	Greenberg *et al.* (2000); Liang *et al.* (2003)
acd6	Encodes a novel protein with putative ankyrin and transmembrane regions		Reduced stature compared with the wild type, which is totally or partially suppressed by *NahG* and *npr1*, respectively. Interestingly, *acd6 npr1* double mutants develop abnormal growths that protrude above the abaxial leaf surface.	Rate *et al.* (1999)
acd11	Sphingosine transmembrane transporter		Dwarf phenotype that is fully suppressed by *NahG*.	Brodersen *et al.* (2002, 2005)
agd2	Member of ARF GAP domain (AGD)		Altered leaf morphology, enlarged cells and mild dwarfism. SA depletion with *NahG* cause tumour-like growths and cells with highly endoreduplicated DNA. Partial blockage of SA signalling by *npr1* decreases cell number and increases the ploidy of mesophyll cells compared with the wild type.	Rate and Greenberg (2001); Vanacker *et al.* (2001); Song *et al.* (2004)
atsr1	Ca^{2+}/calmodulin-binding transcription factor (CAMTA3)		Reduced growth at 19–21 °C, but no significant difference (compared with the wild type) at 25–27 °C. Not only does *NahG* expression revert the phenotype, but *atsr1 NahG* plants are bigger than the wild type.	Du *et al.* (2009)

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Table 1. *Continued*

Transgene or mutation	Gene function	Effect on SA levels	Growth phenotype	References
cpr1	Unknown	**Accumulation from 2- to 200-fold of total SA**	Small, narrow, dark green leaves densely covered with trichomes on the adaxial surface and relatively long siliques compared with the wild type.	Bowling *et al.* (1994)
			Growth much more inhibited at 5 °C.	Scott *et al.* (2004)
			The dwarf phenotype reverts when grown under high light (HL) conditions.	Mateo *et al.* (2006)
cpr5	Unknown		Significantly smaller than the wild type, and reduction in both trichome number and development.	Bowling *et al.* (1997)
			The dwarf phenotype partially reverts under HL conditions.	Mateo *et al.* (2006)
cpr6	Unknown		Loss of apical dominance and a reduction in overall plant size.	Clarke *et al.* (1998)
			The dwarf phenotype partially reverts under HL conditions.	Mateo *et al.* (2006)
dnd1	Cyclic nucleotide-gated ion channel (AtCNGC2)		Dwarf, and partially reverts under HL conditions.	Yu *et al.* (1998); Clough *et al.* (2000); Mateo *et al.* (2006)
dnd2	Cyclic nucleotide-gated ion channel (AtCNGC4)		Dwarf	Yu *et al.* (2000); Jurkowski *et al.* (2004)
lsd6	Unknown		Dwarf with distorted and curled leaves.	Weymann *et al.* (1995)
ssi1	Unknown		Reduced size	Shah *et al.* (1999)

rupture (Piskurewicz *et al.*, 2008). Other phytohormones such as AUXs and ET also modulate plant growth and morphogenesis through a DELLA-dependent mechanism (Achard *et al.*, 2003, 2006, 2007; Fu and Harberd, 2003).

Interestingly, DELLAs modulate the balance of SA/JA signalling in disease resistance, promoting JA perception and/or signalling, and repressing SA biosynthesis and signalling (Navarro *et al.*, 2008). However, it is necessary to determine whether this modulation of SA/JA signalling by DELLA proteins also occurs during growth and development. DELLAs also modulate the levels of reactive oxygen species (ROS), which are also involved in growth-regulatory mechanisms (Achard *et al.*, 2008). Because ROS are closely associated with SA signalling in an autoamplification loop (Shirasu *et al.*, 1997), it has been proposed that the attenuation of SA signalling by DELLAs is the result of diminishing ROS levels (Grant and Jones, 2009).

Photosynthesis

SA effects on leaf and chloroplast structure, and RuBisCO activity. Recent evidence also suggests that SA is an important regulator of photosynthesis because it affects leaf and chloroplast structure (Uzunova and Popova, 2000), stomatal closure (Mateo *et al.*, 2004; Melotto *et al.*, 2006), chlorophyll and carotenoid contents (Rao *et al.*, 1997; Chandra and Bhatt, 1998; Fariduddin *et al.*, 2003), and the activity of enzymes such as RuBisCO (ribulose-1,5-bisphosphate carboxylase/oxygenase) and carbonic anhydrase (Pancheva and Popova, 1998; Slaymaker *et al.*, 2002).

Again, it has been observed that the effects of exogenous SA on photosynthesis parameters differ depending on the dose and plant species tested. High SA concentrations (1–5 mM) cause a reduction in the photosynthetic rate (P_N) and RuBisCO activity in barley plants (Pancheva *et al.*, 1996), and reduced chlorophyll contents in cowpea, wheat, and *Arabidopsis* (Rao *et al.*, 1997; Chandra and Bhatt, 1998; Moharekar *et al.*, 2003). The decline of RuBisCO activity was attributed to a 50% reduction in protein levels compared with non-treated plants (Pancheva and Popova, 1998), while total soluble protein decreased ~68%. Exogenous SA induces alterations in leaf anatomy that consist of a reduced width of the adaxial and abaxial epidermis, and of the mesophyll tissue. Such changes correlate ultrastructurally with an increase in chloroplast volume, swelling of grana thylakoids, and coagulation of the stroma (Uzunova and Popova, 2000). Thus, the diminished photosynthetic activity at high concentrations of SA is due to its effects on the thylakoid membranes and light-induced reactions linked to them.

A lower concentration of SA (10 μM) improves the photosynthetic net CO_2 assimilation in mustard seedlings.

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As P_N increases, carboxylation efficiency, chlorophyll content, and the activities of carbonic anhydrase and nitrate reductase are also up-regulated (Fariduddin *et al.*, 2003). It was suggested that the beneficial effects of this low dose of SA in photosynthesis might be related to the prevention of AUX oxidation by SA, since elevated AUX levels increases P_N and nitrate reductase activity (Ahmad *et al.*, 2001).

SA-mediated protection to oxidative stress. An additional positive effect of SA on photosynthesis is the protection conferred to barley seedlings and maize plants against oxidative stress induced by paraquat (Pq) and cadmium, respectively (Ananieva *et al.*, 2002; Krantev *et al.*, 2008). Pq is a non-selective contact herbicide that accepts electrons from photosystem I (PSI) and transfers them to molecular oxygen. This reaction results in accumulation of ROS that cause extensive damage including lipid peroxidation, chlorophyll breakdown, loss of photosynthetic activity and membrane integrity, as well as electrolyte leakage. Treatment of barley seedlings with 0.5 mM SA for 24 h in the dark, followed by 6 h exposure in the light, decreases photosynthesis and transpiration rates by 25% compared with non-treated controls. Pre-treatment of seedlings with the same SA concentration 24 h before exposure to 10 μM Pq and light reduces Pq-induced chlorophyll losses, H_2O_2 production, lipid peroxidation, and electrolyte leakage, and completely blocked the inhibitory effect of the herbicide on photosynthesis. Similar results are observed in maize plants pre-treated with 0.5 mM SA before exposure to 10–25 μM cadmium (Krantev *et al.*, 2008). The observed protection of photosynthesis conferred by SA could be the result of a very rapid detoxification of ROS. It has been demonstrated in different plants species that pre-treatment with low concentrations of SA enhances tolerance toward most kinds of abiotic stresses due to an enhanced antioxidant capacity (reviewed by Horváth *et al.*, 2007).

SA contribution to light acclimation and redox homeostasis. In *A. thaliana* the SA signalling pathway contributes to achieving optimal photosynthetic activity through regulating light acclimation processes and redox homeostasis. The significant interplay between ROS and SA signalling was uncovered when applications of H_2O_2 and SA to tobacco and *Arabidopsis* plants induced each other, suggesting they are involved in a self-amplifying feedback loop (Leon *et al.*, 1995; Rao *et al.*, 1997; Shirasu *et al.*, 1997). SA inhibits the antioxidant enzymes catalase and ascorbate peroxidase (Chen *et al.*, 1993; Durner and Klessig, 1995, 1996), thus contributing to stabilizing H_2O_2 levels.

The role of SA in photosynthetic parameters and short-term acclimation to high light (HL) was deduced from the phenotypes shown by *A. thaliana* plants with contrasting endogenous SA levels. The *Arabidopsis* mutants *dnd1-1* and *cpr5-1*, with high constitutive SA levels, exhibit decreased maximum efficiency of PSII (F_v/F_m), reduced the quantum yield of PSII (ΦPSII), increased thermal dissipation of absorbed light energy (NPQ), and reduced stomatal conductance in low light (LL; 100 μmol m^{-2} s^{-1}) conditions. In contrast, decreased SA levels in *sid2-2* and *NahG* plants slightly impaired PSII operating efficiency and enhanced thermal energy dissipation in LL (Mateo *et al.*, 2006). SA deficiency in these genotypes correlates with reduced damage to PSII (indicated by the F_v/F_m ratios) compared with wild-type plants, and does not significantly alter leaf water, nutrient contents, and chlorophyll levels (Abreu and Munné-Bosch, 2009). However, the reduced SA levels in *NahG* and *sid2* impair its acclimation to HL (750 μmol m^{-2} s^{-1}), whereas plants with high SA levels (*dnd1-1*, *cpr5-1*) acclimate similarly to wild-type plants (Mateo *et al.*, 2006). Impairment of the light acclimation process in lines with a low SA content is attributed to a higher oxidative stress since the amount of anthocyanins after short-term HL treatment is higher in *NahG* and *sid2* compared with wild-type plants.

These results are consistent with the fact that the SA signalling pathway is activated during light acclimation (Mühlenbock *et al.*, 2008; Chang *et al.*, 2009). Foliar levels of conjugated SA, ascorbate, and glutathione increase 1.5-, 1.8-, and 2-fold, respectively, in *Arabidopsis* plants cultivated in HL (450 μmol m^{-2} s^{-1}), compared with plants cultivated in LL (100 μmol m^{-2} s^{-1}; Chang *et al.*, 2009). Likewise, excess excitation energy (EEE; 2200 μmol m^{-2} s^{-1}) induces a 2-fold increase in foliar SA levels (Mühlenbock *et al.*, 2008). This documented regulation of light acclimation by SA is probably the result of the integration of multiple hormonal and ROS signalling pathways because accumulation of ET and ROS precedes SA accumulation in *Arabidopsis* leaves in response to EEE exposure, and the light stress also induces the expression of genes regulated by ET, ROS, glutathione, SA, ABA, AUX, and sugar signalling (Mühlenbock *et al.*, 2008).

Additional evidence supporting SA involvement in light acclimation is that the *Arabidopsis* response to EEE is regulated by *LSD1* (*LESION SIMULATING DISEASE1*), *PAD4* (*PHYTOALEXIN DEFICIENT4*), and *EDS1* (*ENHANCED DISEASE SUSCEPTIBILITY1*), all genes of the SA signalling pathway leading to disease resistance (Rustérucci *et al.*, 2001; Mateo *et al.*, 2004). *LSD1* is a negative regulator of SA-dependent programmed cell death and plant disease resistance (Dietrich *et al.*, 1997; Torres *et al.*, 2005), whereas *EDS1* and *PAD4* exert a positive regulation on the SA pathway in plant immunity (Wiermer *et al.*, 2005). Both *EDS1* and *PAD4* modulate ET and ROS production in EEE stress signalling, while *LSD1* limits the spread of cell death, induced by EEE or avirulent pathogens, by suppressing ROS production through the regulation of superoxide dismutase and catalase gene expression and activities. From these results, it was proposed that *LSD1*, *EDS1*, and *PAD4* constitute a ROS/ET homeostatic switch to control acclimatory and pathogen defence mechanisms (Mühlenbock *et al.*, 2008).

SA's role in stomatal closure. Stomatal closure is another important factor for photosynthesis and is subjected to control by various phytohormones (reviewed by Acharya and Assmann, 2009). Recent evidence links stomatal closure

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to innate plant immunity, highlighting the role of SA in the function of the guard cells (Melotto *et al.*, 2006). In *Arabidopsis*, 0.4 mM SA induces rapid stomatal closure within 2 h and a 4-fold reduction of stomatal gas exchange (Mateo *et al.*, 2004). Endogenous SA levels promote stomatal closure upon pathogen attack. Both human- (*Escherichia coli*) and plant-pathogenic bacteria (*Pseudomonas syringae* pv. *tomato* DC3000) can induce stomatal closure within the first hour of contact with *Arabidopsis* leaves. This response is compromised in the SA-deficient *NahG* and *eds16-2* genotypes, and in the ABA-deficient mutant *aba3-1*, suggesting that a positive cross-talk between SA and ABA is required to promote stomatal closure upon pathogen perception (Melotto *et al.*, 2006). The stomatal closure promoted by ABA involves calcium (Ca^{2+}) and sphingosine-1-phosphate (Coursol *et al.*, 2003), so it would be interesting to evaluate the relationship to or dependence of SA-induced stomatal closure on these signalling molecules to determine if they are specific or common elements in the phytohormonal control of stomatal aperture, and maybe in development regulation since a close relationship between sphingolipid metabolism and SA signalling profoundly affects plant growth (Table 1).

Respiration

SA regulation of the alternative oxidase (AOX) pathway. SA is involved in the regulation of the AOX pathway in thermogenic and non-thermogenic plants by inducing its gene expression (Kapulnik *et al.*, 1992; Rhoads and McIntosh, 1992). In tobacco cell suspension culture, addition of 2–20 μM SA causes an increased cyanide-resistant O_2 uptake within 2 h, which is accompanied by a 60% increase in the rate of heat evolution from cells, measured by calorimetry (Kapulnik *et al.*, 1992). Moreover, SA treatment induces *NtAOX1* gene expression in a concentration-dependent manner, which correlates with protein abundance. *NtAOX1* transcript abundance increases 2- to 6-fold after 4 h of SA treatment and decreases nearly to basal levels after 24 h (Norman *et al.*, 2004).

AOX couples ubiquinol oxidation with the reduction of molecular oxygen to yield water in a reaction that is insensitive to inhibitors of the cytochrome oxidase pathway. Because AOX is a non-proton-driven carrier, it allows a flexible control of ATP synthesis to maintain growth rate homeostasis (Moore *et al.*, 2002) and is a potential target of SA for plant growth regulation. In addition, AOX is thought to limit ROS production in mitochondria. In cultured tobacco cells, overexpression of *AOX* results in a 57% decrease of ROS abundance, whereas antisense suppression of *AOX* causes a 5-fold increase in ROS levels compared with wild-type cells. It has been suggested that a second oxidase downstream of the ubiquinone (UQ) pool could maintain upstream electron transport components in a more oxidized state, thereby lowering ROS generation by the respiratory chain (Maxwell *et al.*, 1999).

Inhibition of mitochondrial electron transport. Besides the induction of the alternative respiration pathway, that is dependent on the expression of the *AOX* gene, SA might control electron transport and oxidative phosphorylation in plant mitochondria (Xie and Chen, 1999; Norman *et al.*, 2004). SA at concentrations as low as 20 μM inhibits both ATP synthesis and respiratory O_2 uptake within minutes of incubation in tobacco cell cultures, although a significant inhibition occur only at SA concentrations >50 μM. Treatment with 500 μM SA decreases ATP levels by 50% within the first 30 min of incubation, after which the ATP levels continue to decrease to as low as 15% of control levels (Xie and Chen, 1999).

The SA-induced inhibition (from 20 μM to 500 μM) of ATP synthesis in tobacco cell cultures probably does not depend on the induction of the alternative pathway because it occurs within minuters after the addition and does not require *de novo* protein synthesis. In contrast, the induction of alternative respiration by SA is associated with *de novo* synthesis of AOX and requires hours to reach maximum levels (Kapulnik *et al.*, 1992).

Further experiments using a range of substrates and well-coupled isolated mitochondria showed that low concentrations (<1 mM) of SA stimulate the respiration (O_2 uptake) of whole cells and isolated mitochondria in the absence of added ADP by acting as an uncoupler. At higher concentrations (1–5 mM), SA inhibits respiration apparently by preventing electron flow from the substrate dehydrogenases to the UQ pool. Because of its phenolic nature, it has been suggested that SA at millilmolar concentrations may act as a quinone analogue, preventing the interaction between dehydrogenases and the UQ pool (Norman *et al.*, 2004). Respiration in isolated mitochondria can be partially recovered from inhibition by isolating the organelles from SA-treated tobacco cells and resuspending them in fresh reaction medium (Xie and Chen, 1999; Norman *et al.*, 2004). The impact of SA on mitochondrial function is not unique to tobacco as similar uncoupling and inhibitory effects on soybean mitochondria (Norman *et al.*, 2004) and SA uncoupling of mammalian mitochondria have also been reported (Jorgensen *et al.*, 1976). It is possible that both the uncoupling and inhibitory effects of SA in respiration would act to lower cell ATP levels in the *Arabidopsis* mutants that accumulate SA, restricting their growth.

Growth

The role of SA in plant growth has been little studied compared with other plant hormones. Most reviews on this topic do not include SA, or its role is barely described (Santner and Estelle, 2009; Santner *et al.*, 2009; Wolters and Jürgens, 2009).

Effects of exogenous SA on vegetative growth. The effect of exogenous SA on growth depends on the plant species, developmental stage, and the SA concentrations tested. Growth-stimulating effects of SA have been reported in soybean (Gutiérrez-Coronado *et al.*, 1998), wheat

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(Shakirova *et al.*, 2003), maize (Gunes *et al.*, 2007), and chamomile (Kovácik *et al.*, 2009). In soybean plants treated with 10 nM, 100 μM, and up to 10 mM SA, shoot and root growth increase ~20% and 45%, respectively, 7 d after application. Wheat seedlings treated with 50 μM SA develop larger ears, and enhanced cell division is observed within the apical meristem of seedling roots (Shakirova *et al.*, 2003). Likewise, 50 μM SA stimulates the growth of leaf rosettes and roots of chamomile plants by 32% and 65%, respectively, but higher concentrations (250 μM) have the opposite effect (Kovácik *et al.*, 2009). It has been suggested that the growth-promoting effects of SA could be related to changes in the hormonal status (Shakirova *et al.*, 2003; Abreu and Munné-Bosch, 2009) or by improvement of photosynthesis, transpiration, and stomatal conductance (Stevens *et al.*, 2006).

In *A. thaliana*, exogenous SA (100 μM and 1 mM) has a negative effect on trichome development because its application reduces trichome density and number (Traw and Bergelson, 2003). Although the biochemical events involved in the regulation of cell division and growth by SA are still unknown, these results correlate well with the antiproliferative properties in mammalian tumour cell lines of the acetylated derivative (Rüschoff *et al.*, 1998; Dihlmann *et al.*, 2001).

Relationship between the SA signalling pathway and Arabidopsis growth rate. More direct evidence supporting the key role of endogenous SA in the regulation of plant cell growth comes from the characterization of *Arabidopsis* mutant or transgenic plants affected in the SA signalling pathway (Table 1). *Arabidopsis* plants that overexpress the SA-inducible *DOF* (DNA binding with one finger) transcription factor *OBP3* show a decreased growth rate in both roots and aerial parts of the plants, which in the most severe cases led to death (Kang and Singh, 2000). This dwarf phenotype is also observed in *Arabidopsis* mutants that have constitutively high levels of SA, such as *cpr5* (*constitutive expressor of PR5*; Bowling *et al.*, 1997), *acd6-1* (*accelerated cell death*; Rate *et al.*, 1999), and *agd2* (*aberrant growth and death*; Rate and Greenberg, 2001). In contrast, the SA-depleted *Arabidopsis NahG* transgenic plants have a higher growth rate (Abreu and Munné-Bosch, 2009; Du *et al.*, 2009) that is reflected by a 1.7-fold increase in leaf biomass when compared with wild-type plants (Abreu and Munné-Bosch, 2009). The effects of SA depletion on plant growth are more evident at low temperature; *Arabidopis NahG* transgenic plants grow faster at 4 °C than wild-type plants and show a similar growth phenotype to the *amp1* mutant that has increased CK levels (Xia *et al.*, 2009). The elevated CK levels or the decreased SA levels improve plant growth at low temperatures through different mechanisms. The higher growth rate of *amp1* in the cold is associated with a continuous cell division rather than enhanced cell expansion, whereas the increased growth of *NahG* plants at 4 °C results from enhanced cell expansion rather than continuous cell division (Scott *et al.*, 2004; Xia *et al.*, 2009). Moreover, the enlarged cell size of *NahG* plants is associated with an extensive endoreduplication. *NahG* plants have approximately one additional endocycle compared with wild-type plants, resulting in DNA values as high as 32C. It has been suggested that SA negatively regulates expression of cyclin D3 (CYCD3; which drives the G_1/S phase transition) because an increased expression is found in *NahG* plants grown at 4 °C (Xia *et al.*, 2009). These results suggest an unexplored cross-talk between SA, CK, and BR signalling pathways since the latter two are positive regulators of *CYCD3* expression (Riou-Khamlichi *et al.*, 1999; Hu *et al.*, 2000).

Although most of the evidence suggests that SA is a negative regulator of cell division, its role is much more complex. Depletion of SA levels through *NahG* transgene expression reverts the *acd6-1* phenotype, but lead to the appearance of abnormal tumour-like growths in the *agd2* mutant background. The same effect is also observed in *acd6 NahG* plants treated with the SA analogue benzothiadiazole *S*-methylester (BTH; Rate *et al.*, 1999). In *acd6-1* mutants, SA stimulates endoreduplication and cell enlargement, while in the *agd2* background SA suppresses both processes. This discrepancy could possibly be the result of SA interaction with multiple receptors or signalling pathways that control cell growth and development.

NPR1 involvement in the balance of growth regulation and cell death. The transcriptional regulator NPR1 is the key transducer of the SA signal as *npr1* mutants are SA insensitive. Despite NPR1 is required for SA perception, it is not considered to be the SA receptor, which has not yet been identified. Although there are five paralogues of *NPR1* in the *Arabidopsis* genome, there is a partial redundancy in SA perception as determined by the insensitivity to BTH in a high-throughput mutant screening (Canet *et al.*, 2010*a*, *b*). The *npr1-1* mutation in the *acd6* background causes a reduction and delay in the cell death phenotype and partially reverts the reduced stature of *acd6* mutants. However, the *acd6 npr-1* double mutants develop abnormal growths that protrude on the abaxial leaf surface (Vanacker *et al.*, 2001). It would be interesting to determine whether NPR1 is involved in a cross-talk with other phytohormones which could help to explain the abnormal growth, and whether this interaction resembles those between NPR1 and the JA, ABA, and ET pathways, to modulate plant defence responses against pathogens (Spoel *et al.*, 2003; Yasuda *et al.*, 2008; León-Reyes *et al.*, 2009).

Evidence of a cross-talk between SA and AUX signalling during vegetative growth. The discovery that the SA-inducible *DOF* transcription factors *OBP1*, *OBP2*, and *OBP3* are also responsive to AUXs (Kang and Singh, 2000) provides a strong link between the SA and AUX signalling pathways. The increased cell division rate observed in wheat seedlings treated with 50 μM SA correlates with an increase in the endogenous levels of the AUX indole acetic acid (IAA; Shakirova *et al.*, 2003). Interestingly, the reduced apical dominance and stunted growth phenotypes in the *Arabidopsis cpr5*, *cpr6*, and *snc1* mutants that contain

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increased endogenous SA levels are reminiscent of AUX-deficient or AUX-insensitive mutants. This association indicates that SA might interfere with the AUX-mediated responses. In support of this link, these SA-accumulating mutants contain lower endogenous levels of free IAA and reduced sensitivity to AUXs compared with wild-type plants, although exogenous treatment of wild-type plants with SA had little effect on free AUX levels (D. Wang *et al.*, 2007). Moreover, the cross of the AUX-overproducing mutant *yucca* with the SA-accumulating mutants *cpr6* or *snc1* suppresses most of the phenotypes associated with *yucca*. This suppression is due to a repression of the AUX response and not to a reduction in its synthesis. Transcriptomic analysis of *Arabidopsis* plants treated with the SA analogue BTH showed that 21 genes involved in AUX signal transduction are repressed, including *AUX1* and *PIN7* (encoding an AUX importer and exporter, respectively), *TIR1* and *AFB1* (genes for AUX receptors), and *Aux/IAA* family genes (D. Wang *et al.*, 2007). AUX is not the only growth phytohormone targeted by SA because several *Arabidopsis* genes involved in the GA pathway are also down-regulated in response to BTH treatment (Wang *et al.*, 2006).

An additional interesting finding is that the inhibitory growth effect of high SA levels in several *Arabidopsis cpr* mutants is partially overcome at HL intensities. The dwarf phenotype of *cpr6-1*, *cpr5-1*, and *dnd1-1* is partially reverted under HL conditions, whereas *cpr1-1* reverts to almost normal growth. Growth retardation in these mutants is due to impaired photosynthetic activity, and they are able to improve the operating efficiency of PSII during acclimatory responses to HL (Mateo *et al.*, 2006). Although the precise mechanisms are still unknown, SA appears to be a key molecule to maintain a proper balance between photosynthesis and growth.

The SA, ROS, and mitogen-activated protein kinase (MAPK) pathway in plant growth regulation. A very important aspect to consider in plant growth regulation is the tight and complex relationship between SA, ROS, and MAPK cascades, although this has been more thoroughly described for the plant defence response. *Arabidopsis* MPK6 is the orthologue of tobacco SIPK (SA-induced protein kinase; Zhang and Klessig, 1997), and its activity is essential for normal growth and development (Bush and Krysan, 2007; H. Wang *et al.*, 2007, 2008). *Arabidopsis* MPK4 is also closely related to the SA signalling pathway by acting as a negative regulator. The *mpk4* mutants have a severely dwarf phenotype that might be due to SA accumulation. Two lines of evidence support this conclusion because mutations that disrupt SA biosynthesis (*eds1* and *pad4*) or overexpression of the *NahG* transgene partially revert the *mpk4* phenotype (Petersen *et al.*, 2000; Brodersen *et al.*, 2006). Further evidence linking SA to *Arabidopsis* growth comes from the characterization of the null *mkp1* (*map kinase phosphatase1*) and *ptp1* (*protein tyrosine phosphatase1*) mutants, which are negative regulators of MPK6 and MPK3 (Bartels *et al.*, 2009). The *mkp1* and *mkp1 ptp1* mutants have growth defects, increased levels of endogenous SA, and constitutive defence responses including *PR* gene expression and resistance to the bacterial pathogen *P. syringae*. Reduction of SA levels by the *NahG*, *pad4*, or *eds1* genotypes largely suppresses the *mkp1* and *mkp1 ptp1* dwarf phenotypes and the constitutive *PR* gene expression. In addition, *mpk6* and *mpk3* null mutations partially and differentially suppress the *mkp1* (Col-0) phenotype. From these results it is concluded that MKP1 and PTP1 regulate plant growth homeostasis (with MKP1 having the predominant role) acting as repressors of the stress-induced MAPK pathway involving MPK3 and MPK6, which leads to SA biosynthesis and expression of *PR* genes (Bartels *et al.*, 2009).

MAPK cascades are important mediators of the interplay between SA, other phytohormones, and ROS signalling in cell growth regulation. ROS produced by NADPH oxidases are important regulators of polarized growth of root hairs and pollen tubes, by controlling cell wall rigidity and cell signalling events involving Ca^{2+} and MAPK cascades (Foreman *et al.*, 2003; Potocký *et al.*, 2007). *Arabidopsis* PTP1 and MPK6 activities are redox regulated; PTP1 is reversibly inactivated by 1 mM H_2O_2, whereas MPK6 is strongly activated under these conditions, suggesting that PTP1 could be a primary target for ROS signalling in plants (Gupta and Luan, 2003).

Flowering

Flowering-inducing activity of SA. The contribution of SA to flowering regulation has been well known for a long time. Initially it was found that 4 μM SA promotes flower bud formation from tobacco callus (Lee and Skoog, 1965). SA was later identified as the phloem-transmissible factor secreted in the aphid honeydew responsible for inducing flowering in *Lemna gibba* plants kept under a non-photoinductive light cycle (Cleland and Ajami, 1974). SA (3–10 μM) also stimulates flowering in various genera of the Lemnaceae family, including long day (LD), short day (SD), and photoperiod-insensitive types (Khurana and Cleland, 1992). In the SD species *Pharbitis nil*, flowering is induced by poor-nutrition stress. However, flowering under this condition was prevented by treatment with amino-oxyacetic acid, a phenylalanine ammonia-lyase inhibitor, but is restored by SA application. Such behaviour is observed only under stress conditions; thus it appears that SA might be necessary but not sufficient to induce flowering (Wada and Takeno, 2010; Wada *et al.*, 2010).

Further studies have demonstrated that the inflorescences of thermogenic plants have high endogenous SA levels (Raskin *et al.*, 1990), and that in non-thermogenic plants such as tobacco and *Arabidopsis*, SA levels increase 5- and 2-fold in their leaves at the initiation of or during transition to flowering, respectively (Yalpani *et al.*, 1993; Abreu and Munné-Bosch, 2009). In accordance with these findings, SA-deficient *Arabidopsis* plants (*NahG*, *sid1/eds5*, and *sid2*) exhibit a late-flowering phenotype under both SD (8 h light

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and 16 h dark) and LD (16 h light, 8 h dark) conditions, which suggests an interaction of SA with photoperiod and autonomous pathways (Martínez *et al.*, 2004).

A key element linking SA and flowering was recently described in sunflower. The transcription factor HAHB10 belongs to the HD-Zip II family and, when it is constitutively expressed in *Arabidopsis*, induces flowering by up-regulating specific flowering transition genes and repressing genes related to biotic stress. Interestingly, HAHB10 expression is induced after SA treatment and after infection with *P. syringae* (Dezar *et al.*, 2011).

SA interaction with the photoperiod and autonomous pathways. Flowering is regulated by an integrated network of several pathways in *Arabidopsis*, and the role of many genes has been characterized. *CONSTANS* (*CO*) is a key regulator of the photoperiod pathway, the gene *FLOWERING LOCUS C* (*FLC*) is a flowering repressor that integrates autonomous and vernalization pathways, and these pathways converge on a small number of integrators such as *FLOWERING LOCUS T* (*FT*) and *SUPPRESSOR OF OVEREXPRESSION OF CONSTANS 1* (*SOC1*). The photoperiod and autonomous pathways converge on the *SOC1* gene that encodes a MADS box protein which is activated by *CO* and repressed by *FLC* (Mouradov *et al.*, 2002). Recent discoveries have demonstrated that SA is involved in regulating transcription of these genes (Martínez *et al.*, 2004).

The late-flowering phenotype of SA-deficient plants correlates with a 2- to 3-fold higher expression of the floral repressor gene *FLC*, and decreased levels of the *FT* transcript compared with wild-type plants, under either SD or LD conditions. Moreover, exogenous application of 100 μM SA to *Arabidopsis* wild-type plants causes a decrease in *FLC* transcript levels, and UV-C light irradiation that induces SA accumulation activates *FT* expression. Interestingly, although SA seemed to be a repressor of *FLC* expression, this gene is not essential for the late-flowering phenotype of SA-deficient plants because *flc-3 NahG* mutant transgenic lines do not differ in flowering time compared with their parental plants grown under LD and SD conditions. Likewise, expression of other genes such as *CO* and *SOC1* in SA-deficient plants is different under SD and LD conditions. In LD-grown SA-deficient plants, levels of *CO* and *SOC1* transcripts decrease ~50% when compared with wild-type plants, but in SD-grown SA-deficient plants the transcript levels of *CO* increase 2- to 3-fold and *SOC1* expression does not change, compared with wild-type plants. Genetic analysis of the interactions of SA with these components of the photoperiod pathway showed that exogenous SA (100 μM) could revert the late flowering phenotype of the *co-1* mutant, but not of the *soc1* mutant, under LD conditions. Thus, this evidence suggests that SA regulates flowering by interacting with the photoperiod-dependent pathway through a CO-independent branch (Martínez *et al.*, 2004).

Arabidopsis SIZ1 is a key flowering regulator through the control of SA-mediated floral promotion. Loss-of-function *siz1* mutants have an early flowering phenotype under SDs that correlates with high SA levels. Upon *NahG* overexpression in these mutants, the early flowering is suppressed. SIZ1 positively regulates FLC expression, probably through sumoylation of FLD (*FLOWERING LOCUS D*), a plant orthologue of the human histone demethylase 1 (Jin *et al.*, 2008).

Additional evidence obtained from genetic approaches has shown that the transition to flowering promoted by SA also depends on *LD*, *FVE*, and *FCA* genes of the autonomous pathway. Application of 100 μM SA to *ld-1*, *fve-3*, and *fca-9* mutants does not affect their delayed flowering phenotype. Moreover, *fve-3 NahG*, and *fca-9 NahG* plants flower later than their parental plants under SD conditions, but under LD conditions only *fca-9 NahG* plants flower after *fca-9* does. It has been suggested that in LD-grown plants, SA regulates flowering time through an *FCA*-independent pathway that may be the one mediated by *FVE*, whereas under LD conditions, SA could exert its regulation in parallel to both branches of the autonomous pathway in order to regulate integrator genes such as *FT* and *SOC1*. The vernalization and GA pathways do not appear to be affected by SA as *NahG*-overexpressing plants are fully responsive to cold temperatures, exogenous GAs, or constitutive activation of the GA signalling pathway in the *spy-3* mutant background for flower development (Martínez *et al.*, 2004).

Further research must answer key questions such as how the SA signalling pathway interacts with other hormones implicated in the control of flowering time in *Arabidopsis*, the mediators of this cross-talk (i.e. MAPKs, transcriptional regulators, or transcription factors), and whether regulation of flowering by SA is mediated by NPR1.

Senescence

SA requirement for senescence regulation. After reviewing the important role of SA in cell redox homeostasis and photosynthesis, it is not surprising that this phytohormone is also involved in senescence regulation. Senescence is characterized by a decline in photosynthetic activity and increased ROS levels due to a loss of antioxidant capacity. These events are probably partially due to SA accumulation. In *Arabidopsis* senescent leaves, SA levels increase ~4-fold at the mid-senescent stage. Consistent with this observation, *Arabidopsis* plants affected in SA biosynthesis, such as the transgenic *NahG* and the mutant *pad4*, or with a disrupted SA signalling pathway, such as *npr1*, exhibit altered senescence patterns that include delayed yellowing and reduced necrosis compared with wild-type plants (Morris *et al.*, 2000).

SA regulation of senescence-associated genes (SAGs). Senescence is accompanied by important changes in gene expression, and SA contributes greatly to this process. Transcripts of several SAGs, such as *SAG12*, are considerably reduced or undetectable in SA-deficient *Arabidopsis* plants (Morris *et al.*, 2000). Moreover, SA activates the expression of the *Arabidopsis* senescence-related

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genes αVPE, γVPE, *WRKY6*, *WRKY53*, and *SEN1* that encode two vacuolar processing enzymes, two transcription factors, and a protease, respectively (Kinoshita *et al.*, 1999; Robatzek and Somssich, 2001; Miao *et al.*, 2004; Schenk *et al.*, 2005).

The involvement of the SA signalling pathway in senescence was confirmed through a detailed gene expression analysis in *Arabidopsis* senescent leaves (Buchanan-Wollaston *et al.*, 2005). Almost 20% of the up-regulated genes during senescence show at least 2-fold reduced expression in SA-deficient *NahG* transgenic plants. Most of the senescence-enhanced genes that are dependent on the SA pathway encode kinases, transferases, and hydrolases, but their function in senescence progression remains to be elucidated. Although a great deal of effort has been put into identifying the signalling factors required for senescence regulation, further research must determine whether SA is involved in different stages of senescence, and the interconnecting networks with other phytohormones that promote (ABA, JA, an ET) or delay (CKs and GAs) senescence.

WRKY53 in the integration of SA and JA signalling for senescence regulation. The transcription factor WRKY53 is a master regulator of senescence, and also a convergence node with the JA signalling pathway by interacting with the JA-inducible protein ESR (epithiospecifier senescence regulator). Expression of *WRKY53* and *ESR* genes is antagonistically regulated in response to JA and SA, and each one negatively influences the other. ESR appears to have a dual function in *Arabidopsis*, one in senescence and the other in pathogen defence, most probably depending on its cellular localization (Miao and Zentgraf, 2007). ESR is localized in the cytoplasm in the absence of *WRKY53*, where it could function as a cofactor of myrosinase to drive the conversion of glucosinolates into nitriles, which is important for resistance to fungal and bacterial pathogens (de Torres Zabala *et al.*, 2005). In the presence of *WRKY53*, ESR is directed to the nucleus where it inhibits WRKY53 binding to DNA and affects the transcription of SAGs such as *SAG12* and *SAG101* (Miao *et al.*, 2004). These results support the hypothesis that the SA-inducible *WRKY53* gene is expressed early during leaf senescence, then the increase of JA levels during progression of leaf senescence induces ESR expression to modulate WRKY53 action in the nucleus, and WRKY53 expression is suppressed after the onset of senescence (Hinderhofer and Zentgraf, 2001). Recently, it has been found that WRKY53 degradation is also tightly regulated and is mediated by the HECT E3 ubiquitin ligase UPL5 (Miao and Zentgraf, 2010).

Conservation of the SA signalling pathway in the senescence process of different tissues. The importance of the SA pathway in this developmental stage is highlighted by a comparative analysis of genes expressed during silique, leaf, and petal senescence (Wagstaff *et al.*, 2009). Most of the genes that show SA-dependent expression during leaf senescence (Buchanan-Wollaston *et al.*, 2005) also show increased expression during senescence in petal and siliques, whereas the other phytohormones cause differential expression profiles in the three plant tissues studied. ET biosynthesis and binding appear to be more important in silique and petal senescence than in leaves (although some elements are conserved in the three tissues), while genes linked to AUX biosynthesis and response are strongly up-regulated in petals but down-regulated in leaves. In contrast, the SA pathway is active in the three tissues during senescence (Wagstaff *et al.*, 2009).

Autophagy induction by SA during developmental leaf senescence. Autophagy is an important process for plant development, especially during senescence and in the defence response (Kwon and Park, 2008). The importance of autophagy in the senescence process became evident by the characterization of *Arabidopsis* knock-out plants affected in different *ATG* (autophagy) genes (*ATG4*, *ATG5*, *ATG7*, *ATG9*, *ATG10*, and *ATG18a*). These plants display an enhanced senescence phenotype under nutrient-rich conditions (reviewed by Bassham *et al.*, 2006). The autophagy genes *ATG5*, *ATG8*, and *ATG12* are highly expressed in senescent tissues (Wagstaff *et al.*, 2009). In the *atg5* mutant the senescence phenotype is associated with SA accumulation as its endogenous levels are ~3-fold higher compared with wild-type plants (Yoshimoto *et al.*, 2009). These mutants also accumulate high levels of H_2O_2, and highly express the senescence marker gene *SAG12*, as well as the SA-responsive defence genes *PR1* and *PR2*. Although *atg5* also shows increased levels of other phytohormones (JA, AUXs, and ABA), the early senescence phenotype can be attributed only to SA because disruption of its signalling pathway by *NahG* overexpression or *sid2* and *npr1* mutations reverted the phenotype. In contrast, mutations in the JA (*coi1* or *jar1*) or ET (*ein2*) signalling pathways do not affect the *atg5* early senescence phenotype. Interestingly, starvation- and dark-induced senescence in the *atg2* and *atg5* mutants is not suppressed by SA depletion in *NahG*-overexpressing plants. These results correlate with previous findings showing that SA-responsive genes are only up-regulated during developmental leaf senescence, but not in dark-induced senescence (van der Graaff *et al.*, 2006). Autophagy induction by SA was further confirmed by the observation of numerous autophagosome structures in root cells of *Arabidopsis* seedlings expressing green fluoresent protein (GFP)–ATG8a and treated with the SA analogue BTH (100 μM, 8 h). This response is not present in BTH-treated *atg2* and *atg5* mutant roots, and, notably, NPR1 is essential for this response because *npr1* mutant roots do not show rapid accumulation of autophagic bodies after BTH treatment (Yoshimoto *et al.*, 2009).

Autophagy induced by SA is regulated by ACBP3, an acyl-CoA-binding protein that binds phosphatidylcholine and phosphatidylethanolamine, thus interfering with the formation of the ATG5–phosphatidylethanolamine complex and disrupting autophagosome formation and subsequent degradation of ATG8 (Xiao and Chye, 2010; Xiao *et al.*, 2010).

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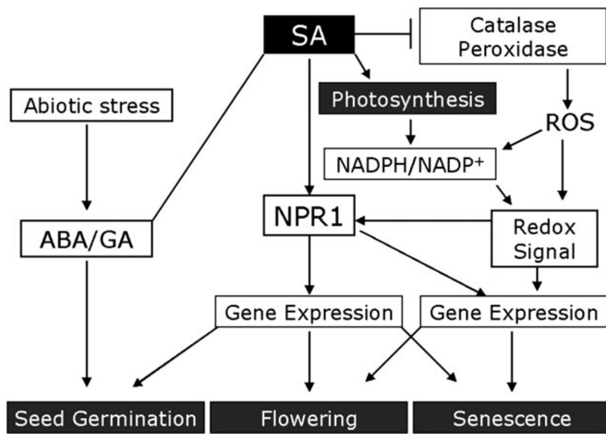

Fig. 2. Descriptive model of salicylic acid function in plant growth and development. SA is perceived by NPR1, a transcriptional activator that regulates gene expression that might participate in seed germination, flowering, and/or senescence regulation. In addition, SA is a key regulator of plant cell redox status by inhibiting catalase and peroxidase activity, and thus modulating reactive oxygen species (ROS) levels. The positive effect of SA on photosynthesis contributes to electron acceptor availability and redox status. NPR1 oligomerization is redox modulated.

Although it is still controversial whether autophagy functions as a cell survival mechanism or as an alternative cell death pathway (Hayward *et al.*, 2009; Hofius *et al.*, 2009; Yoshimoto *et al.*, 2009), SA has a key role in both scenarios, as it has been demonstrated that SA can induce autophagy (generally conceived as a negative regulator of programmed cell death), and proper SA levels are critical to execute cell death fully.

Conclusion

SA is a true plant hormone that goes beyond the defence reaction in plant immunity and response to abiotic stress. In coordination with CKs, ET, AUXs, GAs, JA, and ABA, SA importantly contributes to growth and development regulation, although the biochemical mechanisms that mediate most of these responses remain largely unknown. Further analysis of the dual role of SA in stress responses and development will allow the identification of plant mechanisms devoted to maintaining a proper balance between growth and defence.

Despite the fact that several SA-binding proteins (SABPs) have been identified, the identification and characterization of the SA receptor is probably the most anticipated discovery. Although *NPR1* is not a receptor itself, it is the only known gene that, when mutated, generates plants insensitive to SA (Canet *et al.*, 2010*b*) and causes a clear phenotype on plant defence response and some effects on development. However, not all SA-induced genes depend on a functional NPR1, as demonstrated in microarray analysis in wild-type and *npr1* genotypes. For example, senescence-associated WRKY53 transcription is induced by SA but, in the *npr1* mutant, transcript levels are not significantly different from those of the wild type in *Arabidopsis* seedlings treated with 0.5 mM SA for 2.5 h (Blanco *et al.*, 2009). In this context, characterization of NPR1 paralogues and alleles must reveal their function, both during defence response (Zhang *et al.*, 2006), as has been determined for *NPR3* and *NPR4*, and during development (Canet *et al.*, 2010*a*, *b*).

In *Arabidopsis* it is clear that NPR1 subcellular localization is regulated through a redox-sensitive mechanism mediated by conserved cysteine residues that form intermolecular disulphide bonds that upon SA accumulation are reduced and the monomers translocated into the nucleus (Mou *et al.*, 2003). Once in the nucleus, the NPR1 monomer functions as a co-activator of gene transcription, and the nuclear levels of this protein are kept in check by proteasome-mediated degradation (Spoel *et al.*, 2009). However, this might not be a universal mechanism in all plant species as it has recently been shown that tobacco NPR1 lacks the conserved cysteine residues, and differs in subcellular localization and transactivation potential from AtNPR1, as well in its sensitivity to SA (Maier *et al.*, 2011). Thus future research should emphasize the functional genomics of NPR1 paralogues in various species, as well as the mechanism through which SA modulates redox potential in the plant cell.

The role of SA in plant growth and development is still a controversial field in plant biology; however, various phenotypes are associated with deregulated SA levels (Table 1) and new discoveries and mutant characterization should shed more light on this topic. SA's complex role is not limited only to its canonical signal transducer, NPR1, but also involves its role in modulating the plant cell redox status (Fig. 2).

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