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Interactions between hormone and redox signalling pathways in the control of growth and cross tolerance to stress

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ABSTRACT

The ability of plants to respond to a wide range of environmental stresses is highly flexible and finely balanced through the interaction of hormonal plant growth regulators and the redox signalling hub, which integrates information from the environment and cellular metabolism/physiology. Plant hormones produce reactive oxygen species (ROS) as second messengers in signalling cascades that convey information concerning changes in hormone concentrations and/or sensitivity to mediate a whole range of adaptive responses. Cellular redox buffering capacity that is determined largely by the abundance of ascorbate has a profound influence on the threshold at which hormone signalling is triggered and on the interactions between different hormones. Other antioxidants such as glutathione, glutaredoxins and thioredoxins are also central redox regulators of hormone signalling pathways. The complex network of cross-communication between oxidants and antioxidants in the redox signalling hub and the different hormone signalling pathways maximises productivity under stress-free situations and regulates plant growth, development, reproduction, programmed cell death and survival upon exposure to stress. This interactive network confers enormous regulatory potential because it allows plants to adapt to changing and often challenging conditions, while preventing boom or bust scenarios with regard to resources, ensuring that energy is produced and utilised in a safe and efficient manner.

1. Introduction

Cross-tolerance to environmental stresses is a common phenomenon in plants, whereby exposure to one type of stress confers a general increase in resistance to a range of different stresses (Pastori and Foyer, 2002; Suzuki et al., 2012). Cross-tolerance occurs because of synergistic co-activation of non-specific stress-responsive pathways that cross biotic–abiotic stress boundaries (Bostock, 2005). Cross-tolerance phenomena are frequently linked to the enhanced production of reactive oxygen species (ROS) such as H_2O_2, oxidative signalling and the associated regulation of gene expression through the redox signalling hub, as illustrated in Fig. 1.

It is widely accepted that H_2O_2 and other ROS are important signalling molecules in abiotic and biotic stress responses, often because they serve as messengers for the activation of defence genes (Foyer and Noctor, 2009, 2012). Tight spatial-temporal control of redox signalling molecules allows different and sometimes diametrically opposed physiological events and generates signal specificity that is integrated with the action of plant hormones such as ethylene (ET), salicylic acid (SA), abscisic acid (ABA) and jasmonates (JA) (Xiong et al., 2002; Glazebrook et al., 2003; Fujita et al., 2006). For example, exposure to the atmospheric pollutant ozone generates ROS in the apoplast of plant cells and initiates an oxidative signalling cascade that shares many signalling and regulatory response components with ROS-mediated responses to biotic and abiotic stresses (Baier et al., 2005). Many plant hormones promote ROS production, often through the activation of NADPH oxidases (Rbohs), or alter redox signalling hormones and so induce

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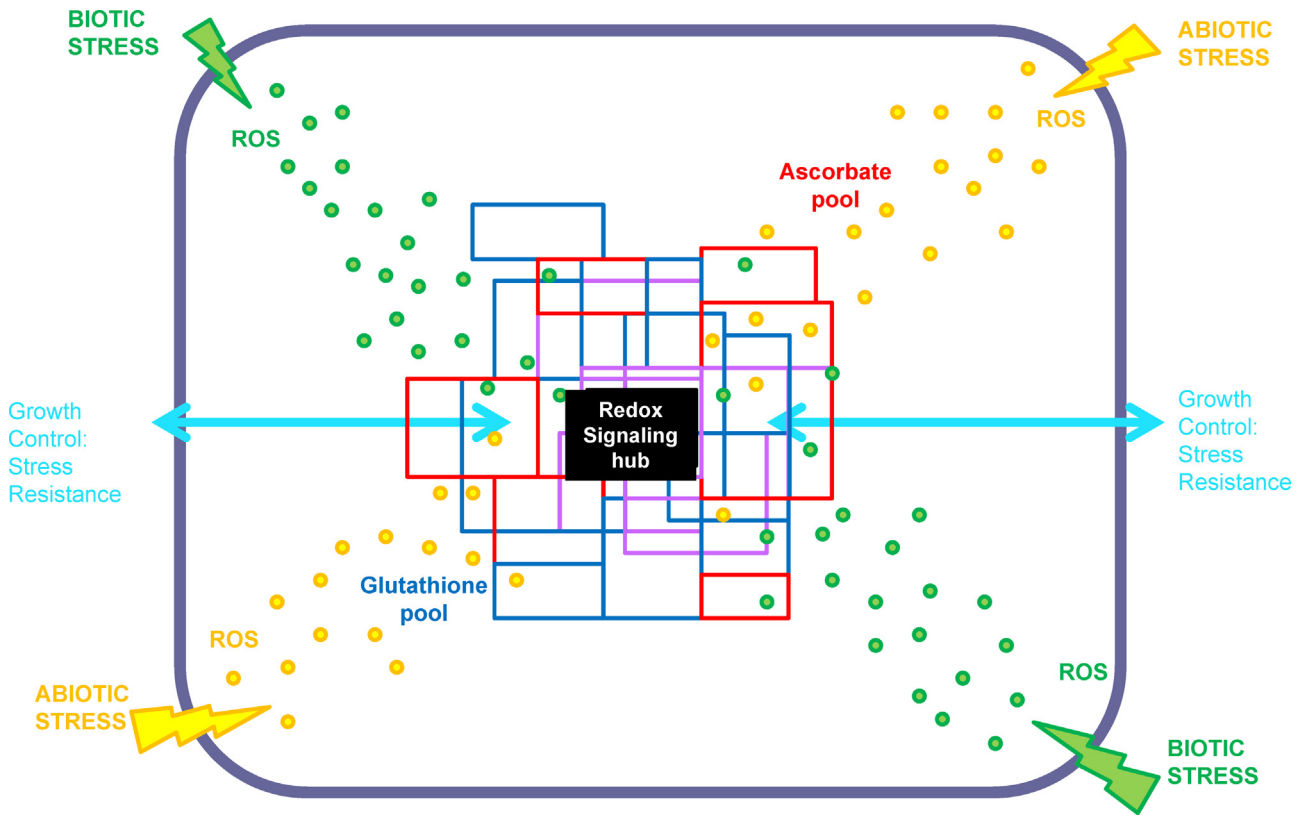

Fig. 1. The redox signalling hub. Reactive oxygen species (ROS).

tolerance to a wide spectrum of stresses (Foyer and Noctor, 2009). Again using the ozone example, ozone fumigation induces ET production (Nakajima et al., 2002) and the activation of abiotic stress responses (Leubner-Metzger et al., 1998), as well as triggering the hypersensitive response (HR) and the induction of programmed cell death, which are intrinsic feature of plant responses to pathogens (Overmeyer et al., 2003). Extensive cross talk is observed between the SA, JA and ET pathways that are induced in response to oxidative stresses such as ozone (Glazebrook et al., 2003) with many points of reciprocal control that can involve neutral, synergistic and antagonistic interactions (Tosti et al., 2006). We discuss the evidence showing that the major plant hormones interact with the cellular redox signalling hub in order to control growth and defence processes in response to environmental stresses.

2. Auxin

Auxin, principally indole-3-acetic acid (IAA) is an essential plant hormone that fulfils numerous roles in plant growth and development including stem elongation, phototropic and gravitropic responses, apical dominance, and lateral and adventitious root formation (Grieneisen et al., 2007; Kleine-Vehn et al., 2008). Like brassinosteroids (BRs) and gibberellins (GAs), auxin promotes cell elongation and controls plant height. BRs and auxin produce ROS as second messengers by activation of NADPH oxidases (Joo et al., 2001; Xia et al., 2009, 2011).

Plant development is regulated by precisely controlled fluctuations in auxin biosynthesis, transport, accumulation, and degradation. The fine-tuning auxin concentrations with local auxin maxima, directional cell-to-cell transport and auxin gradients, together with the differential distribution of the auxin signalling pathways in specific tissues at specific stages of development allow the correct setting of developmental cues in embryogenesis, organogenesis, vascular tissue formation and directional growth in response to environmental stimuli. Auxin transport is controlled by a family of influx facilitators (AUX1, LAX1–LAX3) and two families PIN-FORMED (PIN) and type B ATP binding cassette proteins of efflux carriers. Much attention has focussed on the coordination of the polar sub-cellular localisation of PIN proteins that are responsible for the direction of auxin fluxes (Friml, 2010). The auxin model is considered to be a paradigm for cellular signal transduction pathways (Teale et al., 2008). Moreover, the dynamic interplay between auxin signalling pathways and redox signalling pathways (Fig. 2) permits flexible regulation that is highly responsive of cell metabolism (Pasternak et al., 2005; Tognetti et al., 2012).

The balance between oxidative (ROS) and reductive (antioxidant) signals regulates auxin biology at multiple levels from biosynthesis, conjugation/oxidation, and transport to signal transduction (Gazarian et al., 1998; Cosio and Dunand, 2009; Chen and Xiong, 2009; Tognetti et al., 2010). For example, ROS function as downstream components in auxin-mediated signal transduction to control gravitropism responses in roots (Joo et al., 2001). Genes encoding antioxidant enzymes are among primary auxin-response genes, suggesting a role for auxin in plant stress and defence responses (Abel and Theologis, 1996; Tyburski et al., 2009; George et al., 2010). Changes in auxin distribution and ROS metabolism precede transcriptional regulation (Joo et al., 2001; Pasternak et al., 2005).

To date, relatively little information is available on the complex interplay between ROS and auxin signalling. However, there is evidence in support of a dynamic dialogue and reciprocal dependence between redox (ROS-antioxidant) signalling and the prioritisation of polar auxin transport and signalling. Polar auxin transport, which enables cells to establish local auxin concentrations, is a key feature of auxin homeostasis that is actively controlled by PIN auxin efflux carrier family. The expression of at least some of the PIN

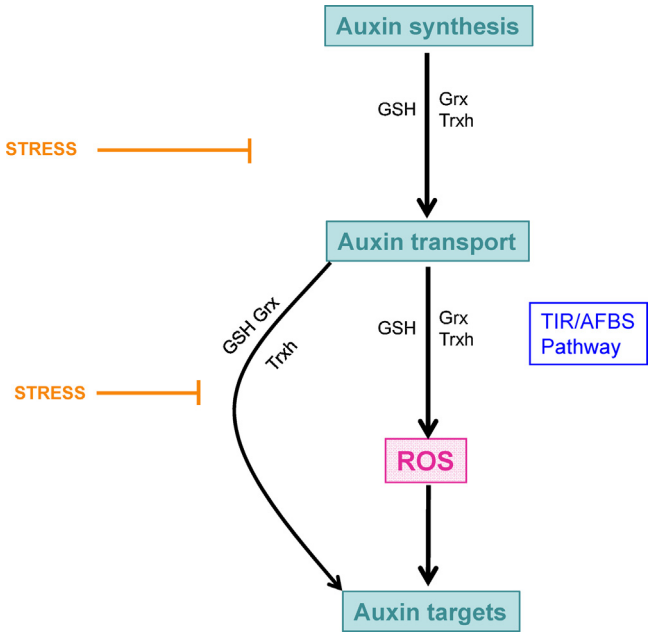

Fig. 2. A simple representation of the auxin signalling pathway, showing possible sites of regulation by GSH, Grx and the cytosolic throredoxin h (Trxh). Reactive oxygen species (ROS).

transporters is regulated by glutathione (Bashandy et al., 2010; Koprivova et al., 2010). The PIN proteins are constantly cycled in and out of the plasma membranes. They are exchanged between the membrane and the "early" endosomes by a process called "constitutive cycling" that allows rapid changes in plasma membrane composition by virtue of the presence of a pool of plasma membrane proteins that are available in nearby early endosomes. The internalisation of PIN proteins occurs by a clathrin-dependent endocytosis mechanism (Chen et al., 1998; Friml et al., 2002). Any exogenous or endogenous stimulus that perturbs cellular redox balance can activate auxin homeostasis because NADPH oxidase-dependent ROS production influences polar auxin transport (Joo et al., 2005). The activation of phosphatidylinositol 3-kinase (PtdIns 3-kinase), which produces PtdIns(3)P, is required for auxin-induced ROS production by NADPH oxidases in the root cells (Joo et al., 2005). PtdIns(3)P plays a regulatory role in endocytosis and vesicle trafficking in plants. Therefore, ROS and phospholipid signalling pathways may cooperate in the control of PIN-dependent auxin transport (Matsuoka et al., 1995; Kim et al., 2001; Zegzouti et al., 2006). Flavonoids may also participate in this regulation via repression of polar auxin transporters (Peer and Murphy, 2007) because they are versatile modulators of PIN interactions with regulatory proteins particularly, PP2AA phosphatase and PINOID kinase (Brown et al., 2001; Santelia et al., 2008; Friml, 2010).

A hypothetical model, in which the activation of the phosphoinositide signalling pathways and plasma membrane endocytosis leads to NADPH oxidase-mediated ROS production, was described by Leshem et al. (2007) in relation to the acquisition of salt tolerance. ROS accumulate in different sub-cellular compartments such as chloroplast and mitochondria in response to environmental stresses. Differential ROS accumulation at various sites within the cell might also depend on vesicle trafficking, according to the phosphorylation state of the PIN proteins that regulates membrane binding. Auxin signalling by the auxin receptor AUXIN-BINDING PROTEIN 1 (ABP1) inhibits the clathrin-mediated internalisation of PIN proteins. Thus, ABP1 acts as a positive regulator of clathrin recruitment to the plasma membrane (Robert et al., 2010). While many uncertainties remain concerning the extent of cross-talk between auxin-mediated vesicle trafficking, and the redox and phosphoinositide signal pathways, redox signals exert a major influence on auxin synthesis and auxin-vesicle transport (Meyer et al., in press).

2.1. The central role of glutathione and thioredoxins

Glutathione (GSH), glutaredoxins (Grx), peroxiredoxins (Prxs), thioredoxins (Trxs) and NADPH-thioredoxin reductases (NTRs) are central elements of the thiol-disulphide redox regulatory hub of plant cells. These components are key regulators for many stress signalling pathways and responses (Gómez et al., 2004; Meyer et al., in press; Noctor et al., 2012). Glutathione can modulate the activities of MAP kinases in a number of ways such as thiol-disulphide exchange and glutathionylation (Foyer and Noctor, 2005) and by influencing cytosolic Ca^{2+} signalling (Foreman et al., 2003; Evans et al., 2005). Little is known about the redox regulation of auxin-related proteins, or the extent to which they undergo regulatory posttranslational modifications, such as glutathionylation and nitrosylation.

Extensive cross talk exists between the reduced GSH/Grx system and the cytosolic Trx and in the control of auxin synthesis, transport and meristem development. Auxin synthesis and polar transport are perturbed in *Arabidopsis* mutants lacking the monothiol glutaredoxin (Grx) Atgrx17. The *Atgrx17* mutants accumulate higher ROS levels than the wild type and they have defective cell cycle control at high temperature (Chen et al., 2011). Moreover, high temperature-induced membrane leakage was increased in the *Atgrx17* mutants indicating that glutathione-mediated redox regulation is critical for auxin transport during heat stress (Chen et al., 2011).

Knock out *Arabidopsis* mutants in the *GSH1* gene, which encodes γ-glutamyl cysteine synthetase (γ-ECS) the enzyme that catalyses the first step of GSH biosynthesis pathway, are lethal at the embryo stage (Cairns et al., 2006). However, the *rootmeristemless1* (*rml1*) mutant, which has a less severe mutation in the *GSH1* gene and thus allows accumulation of about 5% of the wild-type glutathione contents, shows a striking phenotype because it fails to develop a root apical meristem while the shoot meristem is largely unaffected (Vernoux et al., 2000). When the *rml1* mutant was crossed with mutants that are deficient in the two cytosolic NTRs (*ntra*, *ntrb*), the triple mutants that are deficient in both reduced Trx and GSH had an additive shoot meristemless phenotype (Reichheld et al., 2007). The cytosolic Trx, Trxh3 may therefore provide an alternative to the GSH/Grx reduction system for the regulation of shoot auxin metabolism and transport (Meyer et al., in press).

The *A. thaliana cad2* mutant, which has a less severe mutation in the *GSH1* gene than the *rml1* mutant, has about 25% of wild-type GSH contents and it is characterised by increased sensitivity to cadmium. The *cad2* mutant produces less lateral roots than the wild type and has impaired auxin transport but it does not display a *pin* phenotype. Triple mutants deficient in *ntra, ntrb* and *cad2* lack apical dominance and have a *pin1* phenotype, without flower formation (Bashandy et al., 2010). The triple mutants show a limited ability to transport auxin (Bashandy et al., 2010).

In addition to genetic approaches, the specific inhibitor of γ-ECS, buthionine sulphoximine (BSO) has frequently been used to study the effects of glutathione depletion. Treatment of the tips of primary roots with BSO alters auxin transport (Koprivova et al., 2010). However, the auxin-resistant *axr1* and *axr3* mutants are less sensitive to BSO than the wild-type *A. thaliana* plants (Koprivova et al., 2010). GSH synthesis is also required for pollen germination and pollen tube growth (Zechmann et al., 2011). In this study, glutathione depletion was shown to result from disturbances in auxin metabolism and transport (Zechmann et al., 2011).

Once auxins reach the site of action, they activate a MAPK cascade, which modulates gene expression and represses auxin-dependent signalling (Kovtun et al., 2000). Biotic and abiotic stresses also activate MAPK signalling pathways that trigger the expression of antioxidant and defence genes (Hirt, 2000). For example, the expression of a cytosolic ascorbate peroxidase (APX1) is increased by high light stress through a MAPK-dependent pathway (Davletova et al., 2005). Knock out mutants that are deficient in APX1 (*apx1*) have higher H_2O_2 levels (Davletova et al., 2005).

2.2. Auxin and stress signalling

Auxin is considered to be a component of the plant stress-induced morphogenic response (SIMR; Potters et al., 2007; Potters et al., 2009) that serves to limit the adverse effects of environmental stress (Pasternak et al., 2005; Park, 2007; Tognetti et al., 2012). Many auxin responsive genes are repressed by abiotic stimuli such as wounding, oxidative stress and selenium (Pfeiffer and Hoftberger, 2001; Cheong et al., 2002; Joo et al., 2005; Van Hoewyk et al., 2008; Jain and Khurana, 2009; Huang et al., 2010; Kieffer et al., 2010). The suppression of auxin signalling is considered to enhance tolerance to biotic and abiotic stresses, with a reciprocity between auxin-dependent reprogramming of gene expression and that triggered in response to stress (Navarro et al., 2006; Park et al., 2007; Wolters and Jurgens, 2009; Wang et al., 2010).

Auxin functions by directly binding to the TRANSPORT INHIBITOR RESPONSE1 (TIR1), the F-box protein subunit of an ubiquitin protein ligase (E3) called SCF^{TIR1}, which controls SCF-mediated targeted protein degradation. Auxin binding destabilises interactions between TIR1/AUXIN-BINDING F-BOX PROTEIN (TIR1/AFB) families of auxin receptors controlling the expression of auxin-regulated genes. In the absence of auxin, the auxin response factors (ARFs, which are transcription factors that regulate the expression of target genes) are bound to negative regulators that keep them in an inactive state. The binding of IAA to TIR1 liberates the ARFs allowing the expression of target genes. For example, activation of the TIR/AFBs pathway by asymmetric auxin flow occurs at the root tip in response to a gravitropic stimulus and this leads to curvature of the roots (Pan et al., 2009). Arabidopsis *tir1/afbs* mutants have higher levels of ascorbic acid and higher APX activities implicating the TIR/AFBs signalling pathway in the control of antioxidant metabolism (Iglesias et al., 2010).

In addition to glutathione, glutaredoxins and Trx, Prx are also important components of the cellular redox hub (Dietz, 2008). Prx are antioxidative enzymes, which have a broad range of substrate specificity, eliminating H_2O_2, alkyl hydroperoxides and peroxynitrite through Grx- and Trx-based peroxide reductase activity. They are found in many compartments of plant cells. As well as participating in redox signalling they protect the nuclei (1CPrx), plastids (2CPrxA,2CPrxB, PrxQ, and PrxIIE), cytosol (PrxIIB, PrxIIC, and PrxIID) and mitochondria (PrxIIF) from excessive oxidation under stressful conditions (Romero-Puertas et al., 2007; Dietz, 2008). PrxIIE mediates the cross talk between reactive nitrogen and redox signalling pathways (Romero-Puertas et al., 2007). Like ROS, NO regulates auxin-mediated signalling cascades and controls a diverse set of auxin-mediated processes, including developmental and defence responses (Lamattina et al., 2003; Neill et al., 2003; Wendehenne et al., 2004; Delledonne, 2005; Correa-Aragunde et al., 2007; Besson-Bard et al., 2008; Palavan-Unsal and Arisan, 2009; Yoshioka et al., 2009). NO can reduce the level of glutathione and other antioxidants and also maintain the auxin equilibrium by reducing IAA oxidase activity in stressed tissues (Xu et al., 2010). ROS and NO-signalling therefore interact in the regulation of auxin-mediated mechanisms. ROS, antioxidant and NO-signalling pathways participate in the regulation of cell division, differentiation and the programmed cell death, partly through the regulation of the expression of genes such as thylakoid *APX* (Corpas et al., 2001; Tarantino et al., 2005; de Pinto et al., 2006; Zago et al., 2006).

3. Salicylic acid

Salicylic acid is a phenolic phytohormone (monohydroxybenzoic acid) required in the signal transduction cascades that regulate plant defence mechanisms against biotic and abiotic stresses. It is particularly important in systemic acquired response (SAR), which is a broad-spectrum plant immune response involving profound transcriptional reprogramming (Cao et al., 1994; Dempsey et al., 1999; Vlot et al., 2009; Rivas-San Vicente and Plasencia, 2011). *Arabidopsis* mutants with differential endogenous SA contents have been very useful in resolving SA functions and deciphering SA-mediated signal transduction pathways. For example, mutants that are defective in CONSTITUTIVE EXPRESSION OF PR GENES5 (*cpr5*) have constitutively elevated SA levels (Clarke et al., 2000), whereas *sid2* is defective in isochorismate synthase and hence cannot produce SA (Wildermuth et al., 2001), *npr1* cannot respond to the SA signal because of a lesion in the NONEXPRESSOR OF PATHOGENESIS-RELATED genes 1 (NPR1) transcriptional regulator (Cao et al., 1994). Other constitutive defence mutants often have elevated SA levels and show growth retardation phenotypes (Robert-Seilaniantz et al., 2011).

SA is synthesised either from phenylalanine via cinnamic acid or from chorismate by isochorismate synthase. SA acts as a central regulator of cell fate by the reprogramming of gene expression, a process that involves the activation of plasma membrane-bound NADPH oxidases. Together with cell wall peroxidases, NADPH oxidases are responsible for the oxidative burst and accompanying cytosolic Ca^{2+} release that occurs in the apoplast in response to the perception of biotic and abotic stresses (Kawano and Muto, 2000). The apoplastic oxidative burst and resultant ROS accumulation in the extracellular space is characteristic of plant cells exposed to abiotic stresses including physical and chemical shocks, insects and herbivores, symbiotic microorganisms and pathogens. Class III peroxidases may participate in SA-induced ROS metabolism. It has been suggested that SA could act as an e^- donor for Prx, a process that would generate SA radicals (Kawano et al., 2004). Some of the SA-dependent ROS production in plant cells might therefore depend on the interaction between SA and Prx as well as the activation of NADPH oxidases (Almagro et al., 2009). SA is also considered to be an inhibitor of the respiratory alternative oxidase (Hayat et al., 2007).

The activation of NADPH oxidases also serves to suppress the spread of pathogen- and SA-induced cell death (Pogány et al., 2009). The plant plasma membrane NADPH oxidases were discovered on the basis of their sequence similarity to the mammalian respiratory burst NADPH oxidase subunit gp91phox and are therefore also called respiratory burst oxidase (RBOH) proteins. In Arabidopsis the 10 genes encoding these proteins are called *rboh* with different genes designated by letters from A to J. The RBOH proteins fulfil different functions in the regulation of plant responses to environmental stresses. For example, RBOHD triggers death in leaf cells that are under fungal attack but it simultaneously inhibits death in the neighbouring cells by the suppression of SA and ET (Pogány et al., 2009). Mutants defective in the RBOHD or RBOHF proteins show enhanced SA-induced cell death (Torres et al., 2005).

The Arabidopsis *lesion simulating disease 1* (*lsd1*) mutant is characterised by a ROS-dependent spreading cell death phenotype when grown under high or continuous light or upon infection with avirulent pathogens (Aviv et al., 2002; Dietrich et al., 1997; Jabs et al., 1996; Kliebenstein et al., 1999; Mateo et al., 2004, 2006). Mutations in SA signalling genes such as *Phytoalexin Deficient 4* (*PAD4*) and *Enhanced Disease Susceptibility 1* (*EDS1*) block runaway

cell death in *lsd1* (Aviv et al., 2002; Rustérucci et al., 2001). Moreover, crossing *lsd1* with a transgenic line carrying a bacterial salicylate hydroxylase (NahG) or in *npr1* mutants attenuated runaway cell death after SA treatment. It is a redox-sensitive cell death controller in plants exposed to stresses such as, high light and low temperatures (Epple et al., 2003; Huang et al., 2010; Mühlenbock et al., 2008). The amino acid sequence of the encoded LSD1 protein reveals that it contains two highly conserved Cys–Gly–His–Cys sites within the zinc fingers that are characteristic of protein disulphide isomerases, which regulate the formation, reduction and isomerisation of disulphide bonds associated with protein folding. The presence of this sequence suggests that the LSD1 protein may be regulated by Trx, Grx and glutathione, like another component of the SA signalling pathway, NPR1 (Wildermuth et al., 2001; Mou et al., 2003; Tada et al., 2008). NPR1 is an essential component of SA signalling cascade, which induces SAR. In the cytosol, NPR1 is present in an inactive oligomeric complex that is formed through intermolecular disulphide bonds. The SA-dependent induction of SAR requires monomerisation of the oligomeric cytosolic protein NPR1 (Després et al., 2003; Mou et al., 2003; Laloi et al., 2004a,b). The monomerisation process reveals a nuclear localisation signal motif that allows the protein to localise to the nucleus, where it interacts with TGA transcription factors that have a minimum as-1 cis-element of TGACG, and are themselves redox-sensitive (Mou et al., 2003). There are 10 TGA transcription factors in Arabidopsis of which seven (TGA1–TGA7) have been characterised with respect to their interaction with NPR1 (Kesarwani et al., 2007).

Glutathione (Mou et al., 2003) and *Trxh* (Tada et al., 2008) are linked in the activation of this pathway. Exogenous GSH can mimic fungal elicitors in activating the expression of defence-related genes such as, *PR1* (Gómez et al., 2004). Moreover, glutathione accumulation is triggered by pathogen infection (Vanacker et al., 2000; Parisy et al., 2006) in a similar manner to that reported following the application of SA or biologically active SA analogues (Mou et al., 2003). SA-deficient *NahG* Arabidopsis lines showed increased GSSG accumulation and enhanced salt stress tolerance (Borsani et al., 2001). In contrast, SA-deficient *NahG* rice lines had a decreased glutathione pool and showed increased susceptibility to high light stress (Kusumi et al., 2006). SA-inducible genes include certain glutathione S-transferases (GST), some of which are also considered to be markers for increased H_2O_2 (Vanderauwera et al., 2005; Queval et al., 2007, Chaouch et al., 2010).

In the absence of pathogen challenge, NPR1 is continuously cleared from the nucleus by the proteasome, which restricts its co-activator activity to prevent untimely activation of SAR (Spoel et al., 2009). The turnover of NPR1 is promoted by phosphorylation which facilitates its recruitment to a Cullin3-based ubiquitin ligase, which is part of the SFC^{coi} ubiquitin-ligase complex that is considered to regulate cross-talk between the SA- and JA-signalling pathways. The phosphorylated form of NPR1 is required for the expression of target genes and the establishment of SAR (Spoel et al., 2009).

Ascorbate-deficient Arabidopsis mutants such as vitamin C (vtc)1 and vtc2 have a slow growth phenotype and enhanced basal resistance to biotrophic pathogens (Pastori et al., 2003; Pavet et al., 2005; Mukherjee et al., 2010). The *vtc1-1*, *vtc2-1*, *vtc3-1*, and *vtc4-1* mutants were all more resistant to *Pseudomonas syringae* than the wild type plants (Mukherjee et al., 2010). Consistent with these observations, a large number of transcripts that encode the SA-inducible proteins are constitutively expressed in the vtc1 *and* vtc2 *mutants* (Kiddle et al., 2003; Pavet et al., 2005; Brosche and Kangasjarvi, 2012). The abundance of leaf ascorbate has a key influence over both SA and JA signalling pathways (Kerchev et al., 2011; Brosche and Kangasjarvi, 2012).

Glutathione and glutathione-mediated redox control of SA signalling are considered to be important in the regulation of processes that underpin acclimation to high light as well plant immune responses (Dong, 2004; Pieterse and Van Loon, 2004; Chang et al., 2009). This pathway has also been studied intensively in mutants that are deficient in the catalase 2 (*cat2*) isoform, which is important in the removal of H_2O_2 generated by photorespiration (Chaouch et al., 2010; Queval et al., 2011). The *cat2* mutants show a conditional cell death phenotype with induction of associated defence responses that is completely dependent on SA (Chaouch et al., 2010). Moreover, SA-dependent cell death was abolished by myo-inositol, which also eliminated the H_2O_2-dependent reprogramming of pathogen defence responses (Chaouch and Noctor, 2010). The leaves of the *cat2* mutants showed a large increase in the total glutathione (GSH plus glutathione disulphide {GSSG}) pool with a markedly increased GSSG content under photorespiratory conditions (Chaouch et al., 2010; Queval et al., 2007). GSSG accumulation was increased even further in comparison to the wild type plants in *cat2 gr1* double mutants that are deficient in both the major catalase and also the cytosolic glutathione reductase, GR1 (Mhamdi et al., 2010). Taken together, these observations have led to the concept that glutathione is a modulator of SA and JA signalling pathways (Noctor et al., 2012).

More information was provided on the interaction between SA and oxidant signalling pathways by the analysis of mutants that are defective in singlet oxygen signalling (Wagner et al., 2004). Singlet oxygen is mainly produced in plants by the photosystem II reaction in the chloroplasts (Triantaphylides and Havaux, 2009) where it is quenched by α-tocopherol (Shao et al., 2008). Like H_2O_2, singlet oxygen is a highly reactive form of oxygen, whose accumulation triggers programmed cell death (Danon et al., 2005; Triantaphylides and Havaux, 2009). However, singlet oxygen-induced programmed cell death is abolished in the Arabidopsis *fluorescent* (*flu*) mutant that accumulates the sensitizing compound, protochlorophyllide, an intermediate in chlorophyll biosynthesis in the dark (op den Camp et al., 2003). When transferred into the light after a period of darkness, the *flu* mutant produces large amounts of $^1O_2$ leading to major changes in nuclear gene expression (op den Camp et al., 2003). This pathway requires the expression of the *EXECUTOR1* and *2* genes, which encode thylakoid proteins (Lee et al., 2007). The inactivation of the EXECUTER1 chloroplast protein was sufficient to prevent singlet oxygen-induced cell death in *flu* seedlings and prevent singlet oxygen-induced growth inhibition in mature plants (Wagner et al., 2004). Singlet oxygen selectively activates nuclear genes that are either not responsive or are less responsive to superoxide or H_2O_2 suggesting that activation by $^1O_2$ might require promoter elements that are different from those used by H_2O_2. When the *flu* mutant seedlings were made SA-deficient by expression of salicylate hydroxylase (NahG), they were partially protected from the singlet oxygen-mediated cell death indicating that SA is required for the induction of the cell death programme.

4. Ethylene

The metabolic precursor of ethylene (ET; C_2H_4), 1-aminocyclopropane-1-carboxylic acid (ACC), is produced by the S-adenosylmethionine pathway. The first committed step of ET biosynthesis is the conversion of S-adenosylmethionine to ACC by *S*-adenosyl-L-methionine methylthioadenosine-lyase (ACC synthase). ET participates in many aspects of plant biology from germination to dormancy, ripening and senescence, and the regulation of stomatal closure, as well as defences against biotic and abiotic stresses (Bleecker and Kende, 2000; Lin et al., 2009). Like ABA, ET signalling pathways are crucial to the survival of adverse environmental conditions and it is involved in the control of growth (Achard et al., 2003) as well as stress tolerance. For example, drought induced accumulation of the ET precursor,

1-aminocyclopropane-1-carboxylate and the activation of ET signalling lead to a reversible arrest of cell cycle (Skirycz et al., 2011). ET accumulation leads to an inhibition of cyclin-dependent kinase A activity in a manner that was independent of EIN3 transcriptional regulation (Skirycz et al., 2011). However, ET production can have a negative effect on crop production because it triggers senescence and hastens maturity, shortening the grain filling period and filling rate. ET also increases the incidence of embryo abortion, decreasing parameters such as thousand-grain weight.

ET induces ROS generation and H_2O_2 stimulates the expression of ET-responsive proteins and of the enzymes involved in ET biosynthesis (Vandenabeele et al., 2003). Moreover, ROS-dependent plant responses often require ET sensitivity. For example, in the process of leaf abscission, a special layer of cells is formed that must be destroyed to allow removal of the leaf. The programmed cell death pathways occurring in this layer depend on NADPH oxidase-dependent H_2O_2 generation triggered by ethylene. Antioxidants or inhibitors of ET or NADPH oxidases block the progress of leaf abscission (Sakamoto et al., 2008). ET acts upstream in ROS-dependent signalling cell death responses (Chae and Lee, 2001). ET enhances H_2O_2 production in plant cells that are destined to undergo programmed cell death (De Jong et al., 2001; Moeder et al., 2002). ET has been implicated in the sensitivity of plants to stresses such as UV-B exposure (Mackerness et al., 1999) and exposure to heavy metals such as lithium (Bueso et al., 2007). Mutants with reduced ET sensitivity are less sensitive to lithium, which triggers H_2O_2 accumulation (Bueso et al., 2007).

ET perception involves a family of two-component histidine kinase-like receptors that can form both homo- and heterodimers. In the absence of ET, these receptors repress ET responses by signalling through the negative regulator Raf-like MAPKK, CTR1 (Kieber et al., 1993). Once ET is perceived the kinase domain of CTR1 is inactivated and this allows signalling to proceed through the EIN2 protein and the DNA binding proteins EIN3 and EIL1. EIN3 activates *ethylene response factor 1* (*ERF1*) transcription factor, which is a GCC-box-binding transcription activator responsible for the activation of the transcription of ET-responsive genes. EIN3 is regulated by the stability and turnover of the ERF1 signalling pathway, which is regulated by a MAP kinase signalling cascade involving MAPK3 and MAPK6. The Arabidopsis MAPK3 and MAPK6 signalling pathways are also involved in H_2O_2 signalling (Kovtun et al., 2000).

The ERF proteins, which are defined by a conserved DNA-binding domain, belong to the AP2/ERF transcription factor superfamily. They contribute to the regulation of gene transcription regulation of plant growth and development, particularly in relation to environmental stress. Overexpression of *ERF1* activates the expression of genes related to pathogenesis and enhances the resistance to pathogens such as *Botrytis cinerea* and *Plectosphaerella cucumerina*. ERF proteins have also been implicated in the regulation of plant metabolism, for example they are considered to regulate the synthesis of JA, gibberellins, ET, lipids and waxes (De Boer et al., 2011).

Stomatal closure required the integrated cooperation of different signalling pathways including those mediated by ABA, ET, brassinosteroids, H_2O_2 and NO (Desikan et al., 2005; Wilkinson and Davies, 2010). H_2O_2 synthesis in the guard cells involves the activation of NADPH oxidase isoforms AtrbohD and AtrbohF that can be triggered by either ABA or ET, with downstream functions for a range of signalling components such as *ABA insensitive 2* (*ABI2*) PP and MPK3. The NADPH oxidase-deficient *AtrbohD* or *F* mutants show decreased ET–induced stomatal closure and similarly, the ET receptor mutants *etr1-1* and *etr1-3* that are ET-insensitive have impaired H_2O_2-dependent stomata closure (Desikan et al., 2006; Grefen et al., 2008). While ABA can induce H_2O_2 accumulation in *etr1-1*, the H_2O_2 that is produced does not result in stomatal closure in the *etr1-1* mutant, which is hence insensitive to H_2O_2 (Desikan et al., 2006). Histidine kinases are considered to be H_2O_2 sensors and H_2O_2 might interact with the ETR1 histidine kinase receptor (Desikan et al., 2006). The stomata of the ET signalling mutants, *ein2-1* and *arr2*, do not close in response to either ET or H_2O_2 but do generate H_2O_2 following ET stimulation. The ET signalling pathway may therefore involve components that are upstream of EIN2 and ARR2 that activate AtrbohD and F activity. Moreover, ET signalling that is downstream of EIN2 and ARR2 is also required for H_2O_2 perception. NO-associated H_2O_2 synthesis also participates in the interaction between the ABA and ET signalling pathways that regulate stomatal closure. ABA induces NO synthesis in guard cells but this requires prior activation of AtrbohD and F and NADPH oxidase-dependent H_2O_2 synthesis (Bright et al., 2006).

Transgenic plants constitutively expressing dehydroascorbate reductase have a higher ratio of reduced to oxidised ascorbate and they show decreased stomatal closure, consistent with the key role of ROS in stomata opening (Chen and Gallie, 2004). Relatively little is known about the regulation of ascorbate synthesis genes but the F-box protein AMR1 was shown to negatively regulate the expression of genes encoding pathway enzymes (Zhang et al., 2009). Similarly, members of the AP2/ERF transcription factor family such as *Sub1A* and *JERF3*, which enhance stress tolerance by activation of antioxidant enzyme-related pathways (Wu et al., 2008; Fukao et al., 2011), may also regulate the expression of genes encoding ascorbate synthetic enzymes. The *ein2-1*, *ein3-1* and *ein4* mutants have higher leaf ascorbate than the wild type plants, while *ctr1-1* mutants that are defective in the ET-regulated constitutive triple response factor have lower leaf ascorbate levels (Gergoff et al., 2010a). Tomato fruits with defects in ET sensitivity also display greater ascorbate accumulation (Alba et al., 2005). Treatment with ethephon produces a rapid decrease in leaf ascorbic acid contents. Moreover, post-harvest treatment of fruits and vegetables with ET inhibitors delays senescence and extends shelf life, consistent with a higher accumulation of ascorbic acid or other antioxidants (Watkins, 2006; Gergoff et al., 2010b). Fruit and vegetables that have a climacteric ripening process show a high ET peak and enhanced ET sensitivity during organ ripening that is followed by decreased antioxidant contents (Bartoli et al., 1996; Egea et al., 2010).

5. Jasmonic acid

JA belongs to a group of compounds called oxylipins that are formed via oxygenation of fatty acids. JA synthesis begins in the chloroplasts where lipoxygenases convert linolenic acid into hydroperoxylinolenic acid. A range of different metabolites are produced from hydroperoxylinolenic acid including cis+-12-oxophytodienoic acid (OPDA). OPDA can either be retained in the chloroplasts where it is used as a precursor for the synthesis of other oxylipins or it can be transported to the peroxisomes. Similar auxin, the last step of JA synthesis occurs within the peroxisomes, where it is dependent on *b*-oxidation.

JA and related compounds regulate plant responses to wounding and necrotrophic pathogens (Devoto and Turner, 2005). JA-mediated reprogramming of gene expression is perhaps been best characterised in relation to plant–pathogen interactions (Overmeyer et al., 2003; Pauwels et al., 2009). JA can act synergistically with ET in the regulation of defences against necrotrophic pathogens and herbivorous insects. Chewing insects and necrotrophic pathogens activate JA and ET-dependent defence pathways (Kerchev et al., 2012). In contrast, SA-dependent defence pathways are more often activated in response to biotrophic pathogens. Antagonistic and synergistic relationships between the SA and JA/ET defence pathways have been reported. For example, JA

can antagonise the spread of programmed cell death through the suppression of SA biosynthesis and signalling, and also by attenuation of ET sensitivity (Overmeyer et al., 2003). There is also considerable evidence for cross talk between the hormone-induced defence-signalling pathways and the redox signalling pathways that might be important in defining the plant defence strategy depending on the type of attacker (Kerchev et al., 2012). The increased ozone sensitivity of JA mutants supports the view that JA participates in the containment of the ROS-dependent lesion propagation. Similarly, JA may influence ET-dependent lesion propagation by reducing the ET-dependent ROS generation (Overmeyer et al., 2003). The JA signalling pathways control growth through effects on DELLA proteins (see Section 8; Robert-Seilaniantz et al., 2011). DELLA proteins physically interact with JASMONATE ZIM-domain (JAZ) proteins that activate JA signalling pathways by preventing AtMYC2 repression (Robert-Seilaniantz et al., 2011).

The JAZ proteins repress JA signalling pathways when JA levels are low, through interactions with the MYC2 transcription factor (Thines et al., 2007). The isoleucine conjugate of JA promotes interaction between JAZ proteins and the SCF^{coi1} ubiquitin ligase, leading to JAZ degradation via the 26S proteasome. JA synthesis stimulates JAZ binding to COI1, which is an F-box protein that interacts with JAZ transcriptional repressors. Binding to COI1 triggers JAZ degradation via the ubiquitin/26S proteasome pathway, and releasing MYC2 from repression (Chung and Howe, 2009). In this way, JAZ repressors are removed and transcription factors that had previously been bound to JAZ proteins are able to stimulate JA-dependent gene expression. The expression of JA-responsive genes is responsive to the abundance of ascorbate (Kerchev et al., 2011) and to the redox state of the glutathione pool. For example, a large number of JA-responsive genes are repressed in *gr1* mutants that lack the cytosolic/peroxisomal form of glutathione reductase, whereas *gr1 cat2* double mutants that lack both GR and the major leaf form of catalase show H_2O_2-induced expression of these and other JA-associated genes (Mhamdi et al., 2010).

Increases in tissue JA contents and the expression of JA-associated defensive proteins occurs in response to a wide range of environmental stimuli including pathogen attack, touch, elicitation, wounding and osmotic stress (Rao et al., 2000). JA signalling pathways can be systemic in nature. For example, JA accumulation and the expression of JA-responsive genes that occur after wounding to a single leaf is also observed in leaves that are distant from the wound site (Koo et al., 2009). Heavy metal stress also triggered JA accumulation in leaves of *Arabidopsis thaliana* and *Phaseolus coccineus* (Maksymiec et al., 2005). JA has been implicated in NADPH oxidase activation, with H_2O_2 acting as a second messenger regulating the defence response (Orozco-Cárdenas et al., 2001). The JA-response genes include antioxidants and associated defence proteins such as genes encoding enzymes involved in ascorbate and glutathione synthesis (Xiang and Oliver, 1998; Wolucka et al., 2005). Wounding like JA favours increased ascorbic acid accumulation but this effect varies between plant species. For example wounding and JA lead to higher ascorbic acid in Arabidopsis leaves, but they lead to decreased ascorbic acid levels in tomato (Suza et al., 2010). However, water stress-induced JA accumulation in *A. cristatum* increased the transcripts and activities of APX, GR, MDHAR, DHAR, GalLDH, as well as enhancing the contents of ascorbic acid and glutathione (Shan and Liang, 2010). Ascorbic acid deficiency in the Arabidopsis *vtc1-1*, *vtc2-1*, and *vtc3-1*, mutants leads to increased levels of ABA (Pastori et al., 2003) and SA, involving the PAD4, EDS5 and NPR1 signalling pathways (Mukherjee et al., 2010) as well as ABA-dependent repression of JA-dependent signalling pathways (Kerchev et al., 2011).

The JA-dependent stimulation of antioxidant and associated defence proteins explain why wounding or applying JA before the exposure to the atmospheric oxidant ozone decreases the extent of ozone-induced programmed cell death in tobacco (Örvar et al., 1997) and the hybrid poplar (Koch et al., 2000). Methyl-JA (a biologically active derivative of JA) also decreased ozone-induced cell death in Arabidopsis (Rao et al., 2000). In this case, the exogenous application of methyl-JA attenuated ozone-induced H_2O_2 accumulation (Rao et al., 2000). However exposure to ozone resulted in the development of large lesions in the JA-insensitive mutant, *jar1*, and in the *fad3/7/8* mutant that is defective in JA biosynthesis (Rao et al., 2000). Such results demonstrate that JA-dependent signalling pathways play an important role in the regulation of programmed cell death that is triggered in response to ozone pollution and exposure to other oxidants. Ozone treatments result in a rapid induction of antioxidant genes in the Arabidopsis wild type but not in the JA-deficient (*opr3*) mutants (Sasaki-Sekimoto et al., 2005).

6. Strigolactones

Strigolactones (SLs) are signalling molecules that are synthesised from carotenoids in plastids mainly in the lower parts of the stem and in the roots in response to metabolic and environmental triggers (Domagalska and Leyser, 2011). They are important in the control of interactions with other organisms in the environment (Dun et al., 2009) such as mycorrhizal fungi (Bouwmeester et al., 2007; Xie et al., 2010) and parasitic plants (*Striga* sp. and *Orobranche* sp). SLs are transported by members of the ATP-binding cassette (ABC) transporter family (Kretzschmar et al., 2012) and they function downstream of auxin in the control shoot and root branching (Gomez-Roldan et al., 2008; Umehara et al., 2008). They are considered to second messengers in auxin signalling pathways that interact with auxin in a dynamic feedback loop in the control of organ development. By restricting auxin transport in a systemic and local manner they cause auxin accumulation to levels that inhibit growth for example in buds to control of axillary shoot branching. They also influence senescence and photomorphogenesis (Umehara et al., 2008; Gomez-Roldan et al., 2008; Tsuchiya et al., 2010; Ruyter-Spira et al., 2011). It is possible that the SL signalling pathway, like that of auxin and other hormones, produces ROS as second messengers, as suggested in Fig. 3. Several studies have indicated that SLs interact directly with the redox signalling network (Woo et al., 2004) but the precise nature of this interaction is not yet understood. For example, the delayed senescence mutant, *ore9*, is more tolerant to oxidative stress than the wild type. *ORE9* is a homologue of *MAX2*, which is a component of the SL signalling pathway (Woo et al., 2001; Stirnberg et al., 2002).

SL signalling also controls root architecture (Kapulnik et al., 2011a,b; Ruyter-Spira et al., 2011). The SL-dependent control of root branching is lost in the *max2* mutants, which are insensitive to the synthetic SL, GR24 (Fig. 4A). Treatment with GR24 causes some changes in the abundance of ascorbate and glutathione in the roots and shoots of Arabidopsis seedlings (Fig. 4B) but it has little or no impact on the activities of antioxidant enzymes such as catalase or superoxide dismutase.

7. Abscisic acid

The elucidation of the pathway of ABA synthesis in plants was greatly aided by the characterisation of mutants, particularly those that are impaired in zeaxanthin epoxidation, which were first identified by an ABA-deficient (Schwartz et al., 2003). While a “direct pathway” of synthesis had originally been proposed in which ABA is derived from farnesyl diphosphate, it is now generally accepted that ABA is produced in plants predominantly by an “indirect pathway” involving the oxidative cleavage of a 9-cis-epoxycarotenoid (C_{40}) to produce xanthoxin (C_{15}) and a C_{25} by-product the cleavage of carotenoids (Schwartz et al., 2003).

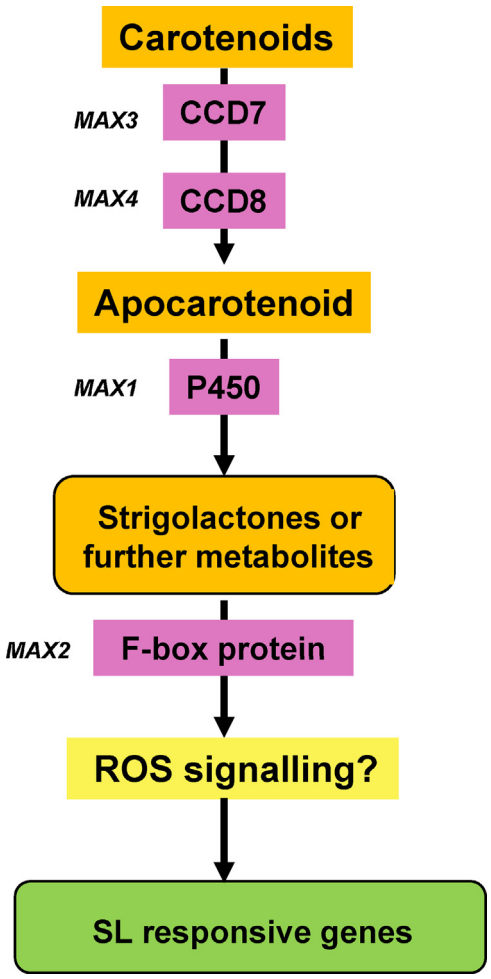

Fig. 3. A simple representation of the strigolactone synthesis pathway, showing the mutations that are available in either the synthesis pathway (*max3*, *max4* and *max1*) or in signalling (*max2*) in *A. thaliana*. This scheme suggests that SLs may regulate the production or signalling of ROS.

ABA is a positive regulator of leaf senescence that accumulates in response to stresses that involve water deficits, such as drought, salt or temperature extremes leading to a reprogramming of gene expression and adaptive responses such as stomatal closure and accumulation of osmo-compatible solutes (Chandrasekar et al., 2000). ABA triggers NADPH oxidase dependent ROS production that is important in mediating the closure of stomata and the regulation of MAPKinase signalling cascades (Guan et al., 2000; Pei et al., 2000; Zhang et al., 2001). ABA-induced H_2O_2 production by the plasma membrane NADPH oxidases, RbohD and RbohF, leads to the activation of calcium-permeable channels, the increase in cytosolic Ca^{2+} causing stomata closure (Kwak et al., 2006). This activation is impaired in the ABA-insensitive *gca2* mutants (Pei et al., 2000). Moreover, high ascorbic acid concentrations in guard cells made the stomata less responsive to addition of H_2O_2 or ABA (Chen and Gallie, 2004). ABA-induced H_2O_2 production by RbohD and RbohF is also important in the induction of plant defence responses (Torres and Dangl, 2005; Torres et al., 2002).

ABA-mediated activation of the OST1 protein kinase is also a key component of ROS generation in ABA-signalling. Mutations in *ost1* inhibit ABA-induced ROS production in guard cells (Kwak et al., 2006). H_2O_2 inactivates the ABI1 and ABI2 type PP2Cs that function as negative regulators of the ABA signalling pathway described above (Bailly et al., 2008). PP2Cs are therefore targets for ROS signalling, particularly in the co-ordinate regulation of ABA-mediated responses (Kwak et al., 2006).

The PYR/PYL/proteins, which are important ABA receptors, function upstream of the type 2C protein phosphatase (PP2Cs)-SNF1-related protein kinase 2 (SnRK2) protein kinase complexes and ROS production in the regulation of ABA-mediated functions. SnRK2 proteins are major regulators of ABA signalling in the control of plant development and responses to water stress. The ABA-activated protein kinase (AAPK) and the related SRK2E/OST1/SnRK2.6 protein kinase regulate the activities of anion channels and stomatal closure in response to ABA upstream of ROS production. Phosphorylated SnRK2 is required for the activation of ABA-induced gene expression. When ABA binds to the PYR/PYL/proteins, the resultant complex sequesters PP2Cs in a way that inhibits their phosphatase activities. The inhibition of PP2Cs allows the SnRK2 protein kinase to activate downstream targets including transcription factors such as AREB/ABF bZIP proteins and anion channels. ABA-induced ROS production and ABA activation of Ca^{2+} channels are impaired in Arabidopsis mutants that are

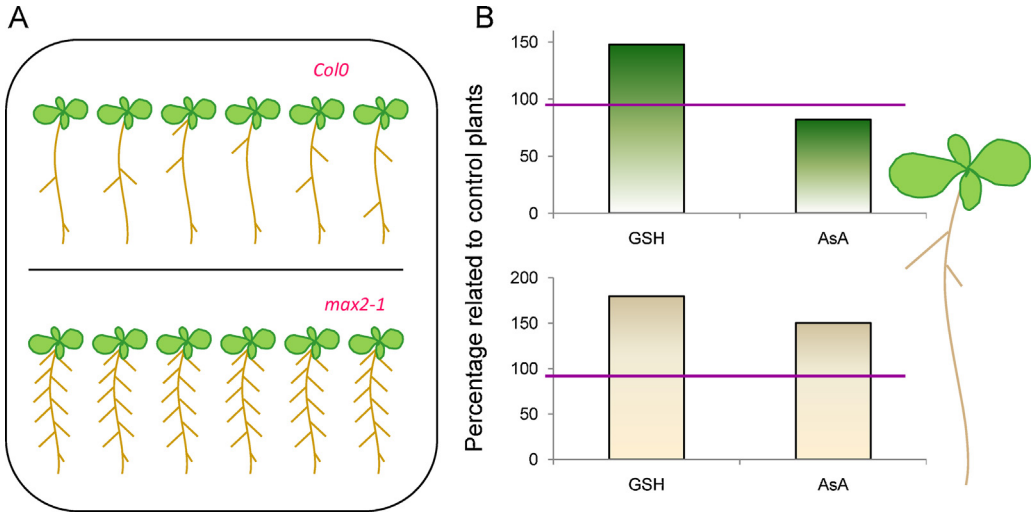

Fig. 4. (A) Schematic comparison of the effects of the strigolactone analogue, GR24, on lateral root development in 8-day-old *A. thaliana* (Colombia 0) seedlings and in the strigolactone signalling mutant, *max2-1*. (B) Effect of GR24 on the the leaf total glutathione (reduced glutathione plus glutathione disulphide) and ascorbate (reduced plus oxidised forms + DHA) pools of 8-day-old *A. thaliana* seedlings.

defective in PP2C (Murata et al., 2001). The SnRK2-interacting calcium sensor, which is important in ABA-mediated regulation of seed germination, probably functions by negative regulation of SnRK2 activity (Bucholc et al., 2011).

The heterotrimeric protein phosphatase 2A (PP2A) complex, which is comprised of a catalytic subunit and regulatory A and B subunits that modulate enzyme activity and mediate interactions with other proteins also plays a key role in the control of basal repression of defence responses through the Constitutive Expressor of Pathogenesis-Related Genes5 (*CPR5*) pathway (Trotta et al., 2011). The *CPR5* pathway, which functions upstream of SA in NPR1-dependent disease resistance, appears to have multiple roles in cell signalling and is involved in cell wall biogenesis, disease resistance, cell proliferation, cell death and sugar sensing (Brininstool et al., 2008). Mutants defective in *CPR5* constitutively express systemic acquired resistance (SAR) and forming spontaneous HR-like lesions. Several PP2A mutants have been characterised to date including the *roots curl in naphthylphthalamicacid1* (rcn1*)* and tonneau2 (ton2)/fass/gordo mutants. The rcn1 mutant shows altered auxin transport, inhibition of ET synthesis and an insensitivity of stomatal closure in response to blue light, ABA and JA (Kwak et al., 2006; Tseng and Briggs, 2010). It also exhibits premature senescence, H_2O_2 accumulation and constitutive activation of SA and JA-dependent defence responses (Trotta et al., 2011).

Further evidence in support of the concept that there is a close association between redox signalling and ABA signalling pathways comes from studies on the *A. thaliana vtc1* and *vtc2* mutants that accumulate lower levels of ascorbate than the wild type plants (Pastori et al., 2003; Kiddle et al., 2003; Kerchev et al., 2011). The leaves of the low ascorbate mutants have increased ABA levels compared to the wild type plants and they show a reprogramming of gene expression that is characteristic of ABA signalling responses (Pastori et al., 2003; Kiddle et al., 2003). Moreover, there is significant overlap between the transcriptome reprogramming in the *vtc1* and *vtc2* mutants and the *abi4* mutants that are deficient in the nuclear localised Apetala 2-type (AP2) transcription factor, ABSCISIC ACID (ABA)-INSENSITIVE-4 (ABI4). ABI4 is important in the ABA-dependent control of seed development and germination and also in orchestrating plant growth responses to variations in carbon/nitrogen availability (Signora et al., 2001; Kerchev et al., 2011). The negative effect of sucrose on ascorbate synthesis and accumulation was lost in the *abi4* mutant (Yabuta et al., 2007). ABA synthesis and signalling are not only important factors in the slow growth phenotype observed in the *vtc1* and *vtc2* mutants (Pastori et al., 2003) but the ascorbate-dependent regulation of plant growth has an absolute requirement for the ABI4 transcription factor (Kerchev et al., 2011). In the absence of a functional ABI4 transcription factor the low ascorbate-dependent slow growth phenotype is not expressed. Thus, like ABI1 and ABI2, ABI4 fulfils important functions in stress signalling cascades involving ROS as second messengers (Kerchev et al., 2011). Moreover, the decreased redox buffering capacity arising from low ascorbate availability in *vtc1* and *vtc2* mutants drives gene expression in a similar manner to that occurring when ABI4 is not functional (Kerchev et al., 2011).

ABA can have positive and negative effects on plant–pathogen interactions depending on the nature (necrotrophic and biotrophic) of the infection (Robert-Seilaniantz et al., 2011). The activation of ABA signalling pathways promotes the susceptibility to necrotrophic pathogens. For example, ABA decreases the resistance of soybean to incompatible nonpathogenic strains of *Phytophthora sojae* (Ward et al., 1989). Similarly, drought-induced ABA accumulation in Arabidopsis decreases resistance to avirulent Pst (Mohr, 2003). Conversely, ABA treatment enhanced the resistance of Arabidopsis to biotrophic pathogens (Ton et al., 2009).

ABA accumulation was associated with enhanced antioxidant enzyme activities in germinating wheat seedlings subjected to mild osmotic stress (Agarwal et al., 2005). ABA treatment of barley aleurone cells increased the activities of catalase, APX and superoxide dismutase and the susceptibility to ROS-induced programmed cell death was decreased (Fath et al., 2001). Similarly, ABA treatment increased the catalase, APX and superoxide dismutase activities of maize seedling together with a beneficial effect on the contents of ascorbate, glutathione, α-tocopherol and carotenoids (Jiang and Zhang, 2002b). Similar effects were observed in leaves exposed to moderate water stress (Jiang and Zhang, 2002a).

ABA can inhibit GA biosynthesis and its action is often antagonistic to GA, the ratio of ABA to GA being a fundamental determinant of growth or quiescence (Ross et al., 2011). For example, the GA-regulated DELLA protein called RGL2 inhibits seed germination by stimulating ABA synthesis and the activity of ABA INSENSITIVE5 (ABI5), which is a basic domain/leucine zipper transcription factor. Genetic studies have shown that RGL2 is epistatic to the ATP binding cassette (ABC) transporter called COMATOSE (CTS, also called PXA1 and PED3), which is involved in the import of substrates into the peroxisomes for *b*-oxidation pathways. The block on seed germination caused by a mutation in CTS cannot be rescued by GA, but germination is rescued by mutations at the ABI5 locus, providing genetic evidence for a direct link between the pathways (Kanai et al., 2010). Redox regulation of the balance between ABA and GA-signalling pathways has also been suggested to influence germination (Liu et al., 2010) and other processes such as dormancy and floral induction (Barth et al., 2004).

8. Gibberellins

Gibberellins are cyclic diterpene compounds that regulate plant growth and development. They stimulate elongation or expansion of organs via enhancement of cell elongation and, in some cases, also cell division. Furthermore, GAs may induce developmental switches, such as between the juvenile and adult phases or between vegetative and reproductive development. They have also been implicated in the function of the meristem, where they are thought to promote differentiation and suppress the maintenance of stem cells. GA functions are also responsive to environmental cues, including changes in light conditions, temperature or stress, (Yamaguchi, 2008) allowing GAs to translate these extrinsic signals into developmental changes. GA synthesis is also influenced by ascorbate availability. Ascorbate is a co-factor in the catalysis of 2-oxoacid-dependant dioxygenase (2ODD) reactions and the activities of these enzymes are enhanced by the addition of ascorbate in vitro (Arrigoni and De Tullio, 2000). Dioxygenases are important in the final stages of GA synthesis, where GA_{12} is converted to bioactive GAs (Hedden and Kamiya, 1997).

GAs mediate growth in response to environmental signals by relieving the constraints on gene expression imposed by a family of growth-repressing regulators, the nuclear growth-repressing DELLA proteins, as mentioned above (Peng et al., 1999; Harberd et al., 2009). GAs and auxin have many overlapping functions in the control of organ expansion. While auxin also appears to control DELLA stability independent of its regulation of GA, the two signalling pathways interact, with auxin, controlling growth at least in part by modulation of the GA signalling cascade.

GA levels was first linked to stress protection in an analysis of the responses of wild type and dwarf barley near-isogenic lines to heat stress (Vettakkorumakankav et al., 1999). *Arabidopsis* mutants that have a reduced GA content or containing a GA-insensitive form of DELLA proteins are more salt tolerant than wild-type plants, while plants with loss-of-function DELLA mutations have increased susceptibility to salt stress (Achard et al., 2006). It is now generally accepted that the DELLA proteins, which are transcriptional regulators that restrain plant growth, play a key role in the control

of growth and tolerance in response to biotic and abiotic stresses. GA promotes growth by stimulating the destruction of the DELLA proteins, which accumulate in plants exposed to adverse environmental conditions to restrain growth and enhance plant survival (Achard et al., 2003, 2006; Harberd et al., 2009). Plants exposed to salt stress had increased DELLA contents and showed less ROS accumulation and enhanced expression of genes encoding for antioxidant enzymes (Achard et al., 2008). The DELLA proteins also contribute to pathogen resistance because the GA/DELLA pathways alter the balance between the SA and JA/ethylene signalling (Achard et al., 2008). Higher DELLA protein contents favour increased resistance to necrotrophic pathogens (Achard et al., 2008). Moreover, the GA-deficient *ga1-3* Arabidopsis mutants that have higher levels of DELLA proteins are much less susceptible to ROS-dependent cell death (Achard et al., 2008).

The accumulation of DELLA proteins can remove the potential for cell proliferation (Skirycz et al., 2011). Stress conditions that enhance ABA/GA ratios favouring DELLA protein accumulation and lower ROS levels (Finkelstein et al., 2008). High ABA/GA ratios can also induce dormancy in tubers, buds and seeds. For example, ABA decreased ROS production in rice seeds leading to a repression of ascorbate and GA accumulation (Ye et al., 2012). In contrast, dormancy release is stimulated by GA and ROS but inhibited by antioxidants (Oracz et al., 2009).

The 'Green Revolution' that improved worldwide wheat yields from the 1960s was based upon a combination of new varieties and the increased use of fertilisers and pesticides. An important feature of the new varieties was reduced height: the semi-dwarf plants had increased yield, as less of the plants' energy was wasted on producing straw and more went into the harvested grain. The shorter plants were also stronger and more capable of bearing the increased yields without lodging (falling over) in wind and rain. These dwarf varieties of wheat carried genes (called "*Reduced Height*" or *Rht*) that made them unresponsive to GA, which normally increases stem height. The most commonly used alleles of these genes are *Rht-B1b* and *Rht-D1b* (previously called *Rht1* and *Rht2*) semi-dwarfing alleles derived from Norin 10, which reduce sensitivity to endogenous GAs. Wheat plants carrying altered function *Rht-B1b*/*Rht-D1b* alleles of *Rht-1* genes (dwarf plants) show increased antioxidant enzymes and retain higher chlorophyll contents when subjected to potassium deficiency (Moriconi et al., 2012).

Mutations in the DELLA proteins or GA (GA_3) treatment increase SA biosynthesis and signalling (Robert-Seilaniantz et al., 2011). A role for GA in SA-dependent responses has also been reported in plants subjected to abiotic stress (Alonso-Ramírez et al., 2009). The application of GA_3 reversed the inhibitory effect of salt, oxidative, and heat stresses on the germination and establishment of Arabidopsis seedlings (Alonso-Ramírez et al., 2009). The DELLAs proteins also regulate JA signalling pathways. GA promotes JA biosynthesis in stamens by a DELLA-dependent process (Robert-Seilaniantz et al., 2011). The over-expression of DREB/AP transcription factors, which are involved in many stress responses, results in GA-reversible growth inhibition and a reduction in bioactive GA concentration. Phosphate limitation-dependent changes in root architecture result, at least in part, from decreased levels of bioactive GAs. GA metabolism is influenced also by nitrogen availability, particularly when primary nitrogen assimilation is enhanced as a result of alteration in cellular redox state caused by the absence of mitochondrial Complex I (Pellny et al., 2008). Respiratory Complex I is the first enzyme of the respiratory electron transport chain. It is a rotenone-insensitive NADH ubiquinone reductase that couples electron transport to proton translocation. The near homoplasmic *Nicotiana sylvestris* CMSII mitochondrial DNA mutant is devoid of the NAD7 gene and Complex I assembly is impaired and thus the CMSII mutants lack the major NADH dehydrogenase of the respiratory electron transport chain (Dutilleul et al., 2005). However, the mutant is able to sustain electron flow through the engagement of non-phosphorylating alternative dehydrogenases that have a low affinity for NADH and this causes an alteration in cellular pyridine nucleotide homeostasis. The CMSII mutants show a large increase in tissue pyridine nucleotides and a much higher level of NADH available to drive NADH-requiring pathways such as primary nitrogen assimilation (Dutilleul et al., 2005). The enhanced NAD(P)H availability also exerts a significant influence over GA synthesis and signalling (Pellny et al., 2008). Nitrogen availability also influences the expression of genes encoding GA-biosynthetic enzymes, particularly the abundance of transcript for the GA-inactivating gene *GA2ox* and the biosynthetic gene *GA3ox* (Pellny et al., 2008). The CMSII mutants show slow growth phenotype but the wild type growth phenotype can be partially restored by GA treatment (Pellny et al., 2008). The CMSII mutants also showed large changes in the levels of the GA-biosynthetic intermediates suggesting redox control of GA synthesis and metabolism (Pellny et al., 2008). Further evidence in support of the concept that GA synthesis and metabolism are responsive to redox controls comes from an analysis of the cysteine-rich GASA4 protein, which promotes GA responses (Rubinovich and Weiss, 2010). The GAST1-like proteins are considered to be involved in redox reactions by virtue of their cysteine-rich domain and they may regulate the redox status of specific components to promote or suppress GA-related responses. The over-expression of GASA4 suppressed ROS accumulation enhanced resistance to NO (Rubinovich and Weiss, 2010).

9. Conclusions and perspectives

Accumulating evidence supports the concept that cellular redox signalling and hormone signalling pathways form an integrated redox-hormone network that regulates plant growth and defence pathways (Fig. 5). The efficient operation of this network requires extensive metabolic crosstalk and multiple points of reciprocal control. For example, stomatal closure is controlled by a number of hormones including ABA, brassinosteroids and ET and it requires redox regulation through NO and ROS production (Foyer et al., 2008) but the abundance of ascorbate in the guard cells modulates hormone action (Chen and Gallie, 2004). Root architecture is also tightly controlled by the integrated action of redox and hormone-related signals (Foreman et al., 2003; Jones et al., 2007; Takeda et al., 2008; Guo et al., 2009).

Drought-induced ET accumulation can quickly and reversibly cause cell cycle arrest in a manner that allows the cells to remain in a quiescent state from which they can recover when the environmental conditions improve (Skirycz et al., 2011). In this way ET accumulation in plants suffering environmental stress can reduce root and shoot growth and biomass accumulation by direct effects on growth process. ET also disrupts the ABA-mediated control of photosynthesis and leaf growth. ABA is a significant hormone in the control of drought stress because it favours stomatal closure and it can limit leaf growth in order to reduce plant water loss via transpiration. ET is therefore linked to stress sensitivity in terms of limiting crop yields, by increasing leaf injury, accelerating senescence. The sensitivity of stress responses may therefore be governed by the ratio between ET (together with its precursor ACC, 1-aminocyclopropane) and ABA rather than the concentration of either hormone alone. A much better understanding of such interactions is essential for the improvement of crop plants and to obtain high yields under stressful environmental conditions.

Programmed cell death is an important plant defence response that prevents the proliferation of pathogens. SA, JA and ET fulfil essential roles in mediating pathogen responses and they interact in the control of ROS generation and antioxidant enzyme

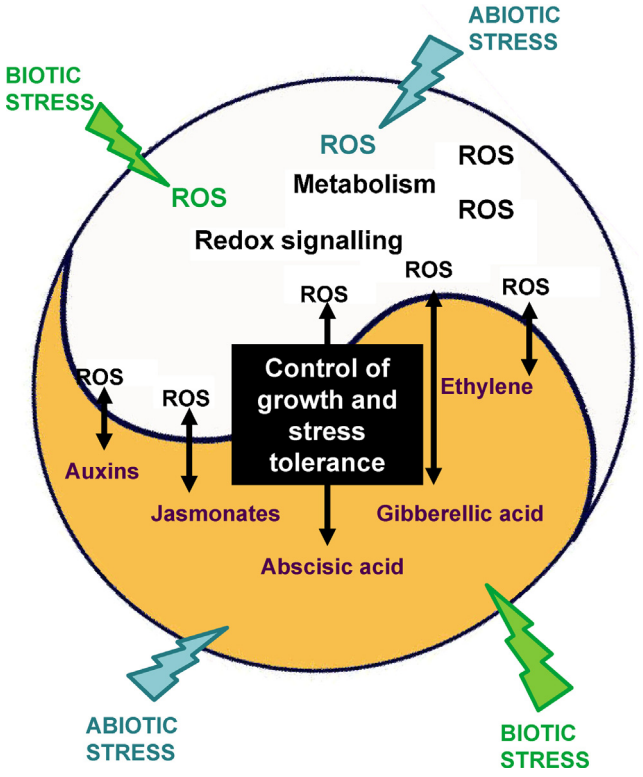

Fig. 5. Diagrammatic representation of the interactions between hormone and redox signalling pathways in the control of growth and defence responses. Reactive oxygen species (ROS).

activities to control stress responses and cell suicide pathways (Rao et al., 1997; Horváth et al., 2007; Ashraf et al., 2010). The threshold for such signalling events is strongly influenced by cellular redox buffering capacity, as illustrated in the low ascorbate mutants, which show enhanced resistance to biotrophic pathogens (Barth et al., 2004; Pavet et al., 2005; Mukherjee et al., 2010). Conversely, plants over-expressing ascorbate oxidase have a more highly oxidised ascorbate redox state in the apoplast show decreased pathogen resistance (Pignocchi et al., 2006). Glutathione also plays a key role in the SA, JA and ET interaction, as illustrated in the similarities between glutathione-associated genes and JA-dependent gene expression (Mhamdi et al., 2010). Genes that link glutathione and JA include those encoding GSTs and GRXs which could link glutathione to JA signalling pathways, in a similar manner to the regulation of auxin-signalling pathways by glutathione, as discussed above. The redox state of the glutathione pool is modulated by biotic challenges, while JA accumulation can stimulate the expression of the genes encoding the enzymes of glutathione synthesis. Such findings underline the potential importance of ascorbate and glutathione as regulators of redox-triggered signalling through the SA, JA and auxin signalling pathways.

The regulation of growth and defence in response to hormones and redox signals is highly dependent on cell identity and development stage. For example, the accelerated senescence of older leaves is an important trait for plant survival under unfavourable conditions. Stress-induced ET production and ROS accumulation are observed in mature leaves exposed to stress but these changes are not found in the young leaves, which are less likely to exhibit premature senescence (Pazmiño et al., 2011). Such considerations have to be taken into account in post harvest physiology, where the quality and nutritional characteristics of fresh fruits and vegetables have to be preserved between harvest and consumption (Kader, 2002). Detachment from the plant and storage (especially in dark and refrigerated storage) is stressful for edible organs and leads to loss of "freshness", characteristics such as appearance and firmness (Hodges and Toivonen, 2008). Loss of freshness is important because consumers select products on this basis (Bruhn, 2002). Consumers are also interested in foods with a high nutritional quality particularly vitamin, mineral and fibre contents.

The acquisition of cross-tolerance characteristics by post-harvest treatments with mild stresses such as low ozone, H_2O_2, heat shock and UV-C, has been successfully used to extend shelf life and improve post-harvest storage. For example, heat shock has been successfully used for extending the post-harvest life of spinach leaves (Gómez et al., 2008). Heat shock signalling is dependent on H_2O_2 for increasing abiotic stress tolerance (Gechev et al., 2002). Heat shock can be used to increase the antioxidant contents of plant organs during storage (Zhang et al., 2005; Vicente et al., 2006; Costa et al., 2005). A better understanding of plant ROS–antioxidant–hormone interactions will facilitate the further development of these "clean technology" approaches to plant produce conservation.

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