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Review

Interplays between the cell wall and phytohormones in interaction between plants and necrotrophic pathogens

Majse Nafisi, Lorenzo Fimognari, Yumiko Sakuragi *

Department of Plant and Environmental Sciences, University of Copenhagen, Thorvaldsensvej 40, Frederiksberg 1871, Denmark

This paper forms part of a special issue of *Phytochemistry* dedicated to the memory and legacy of Professor (Godfrey) Paul Bolwell, MA DSc (Oxon). (1946–2012), internationally-recognised plant biochemist and Regional Editor of *Phytochemistry* (2004–2012). He is much missed by his friends.

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ABSTRACT

The plant cell wall surrounds every cell in plants. During microbial infection, the cell wall provides a dynamic interface for interaction with necrotrophic phytopathogens as a rich source of carbohydrates for the growth of pathogens, as a physical barrier restricting the progression of the pathogens, and as an integrity sensory system that can activate intracellular signaling cascades and ultimately lead to a multitude of inducible host defense responses. Studies over the last decade have provided evidence of interplays between the cell wall and phytohormone signaling. This review summarizes the current state of knowledge about the cell wall-phytohormone interplays, with the focus on auxin, cytokinin, brassinosteroids, and abscisic acid, and discuss how they impact the outcome of plant–necrotrophic pathogen interaction.

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* Corresponding author. Tel.: +45 35333317.
E-mail address: ysa@plen.ku.dk (Y. Sakuragi).

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1. Introduction

The plant cell wall surrounds every cell in plants and forms a dynamic physical barrier that protects the cell from microbial infection. The main constituents of the plant cell wall are cellulose, hemicelluloses, pectins, and glycoproteins (Carpita and Gibeaut, 1993). Cellulose form crystalline microfibers and provides a scaffold to the cell wall, hemicelluloses crosslink with celluloses to provide support to the cellulose microfibrils network, while pectins not only crosslink cell wall polymers but also serve as hydrated extracellular matrix components (Somerville et al., 2004). The exact composition of the cell wall polysaccharides varies between tissues and can differ in the same tissues during developmental processes. During pathogen infections, the cell wall undergoes dramatic structural and chemical changes (Eggert et al., 2014; Voigt, 2014; Vorwerk et al., 2004), including lignification (Vance et al., 1980; Zhao and Dixon, 2014), deposition of callose (Luna et al., 2011), cell wall protein cross-linking (Bradley et al., 1992), accumulation of reactive oxygen species and antimicrobial compounds (phytoalexins) (Franke et al., 2005; Lamb and Dixon, 1997; O'Brien et al., 2012), which can culminate to restrict the infection and prevent further pathogen progression. Emerging notion is that the cell wall integrity is sensed by plants and, when compromised, it can activate intracellular events involving phytohormone signaling cascades that can in turn activate defense responses (Hamann, 2012). In addition, degradation of cell wall constituents, particularly by necrotrophic pathogens, are sensed by a plasmamembrane receptor(s), leading to activation of defense signaling cascades and eventual mounting of inducible defense responses (Fry et al., 1993; Monaghan and Zipfel, 2012).

The aim of the present review is to provide a brief summary of the current state of our understanding regarding the interplay between plant cell wall integrity and phytohormones in the context of defense against necrotrophic pathogens. Plant pathogens are often classified into two major classes: necrotrophic and biotrophic pathogens. Lifestyle, infection strategy, and host defense responses vary greatly between the two classes. Necrotrophic pathogens use a suite of cell wall degrading enzymes and toxins to kill and macerate the host tissues to feed on; in contrast, biotrophic pathogens cause relatively minor damage on the host cell wall and maintain host viability to acquire nutrients. Some plant pathogens can manifest biotrophic-like strategies at earlier stages of infection and then switch to necrotrophic-like strategies at later stages; such pathogens are referred to as hemibiotrophic (Laluk and Mengiste, 2010). The cell wall maceration and damages on the host tissue by necrotrophic phytopathogens causes devastating economic losses in agriculture (Williamson et al., 2007). In the *Botrytis cinerea* genome, the large capacity for plant cell wall degradation was illustrated by the identification of 118 genes unambiguously associated with plant cell wall degradation (Amselem et al., 2011). Plants defend against necrotrophic phytopathogens through a complex interplay of phytohormone signaling and defense responses, and this topic has been extensively reviewed during the past few years (Cao et al., 2011; De Bruyne et al., 2014; De Vleesschauwer et al., 2013; Grant and Jones, 2009; Perez and Goossens, 2013; Pieterse et al., 2012). Thus plant–necrotrophic pathogens interaction offers unique and valuable insights into interplays between cell wall stress perception and phytohormone signaling cascades that culminate to determine the necrotrophic disease outcome. In this review we discuss (i) roles of phytohormones, namely auxin, cytokinin, brassinosteroids (BR), and abscisic acid (ABA), in defense against necrotrophic phytopathogens, (ii) how these phytohormones modulate the cell wall properties, and (iii) how the cell wall can modulate the homeostasis of these phytohormones signalings and impact pathogen resistance. Although the main focus is on the interaction between the plant cell wall and necrotrophic pathogens, evidence based on interaction between plants and biotrophic pathogens are discussed where relevant. Involvement of jasmonates (JA), ethylene (ET), and salicylic acid (SA) in the cell wall-mediated defense has been extensively discussed in recent years (Hamann, 2012; Malinovsky et al., 2014) and will not be dealt with in this article.

2. Auxin

2.1. Roles of auxin in plant defense against necrotrophic pathogens

In plants, auxin can be found in the forms indole-3-acetic acid (IAA), 4-chloroindole-3-acetic acid, phenylacetic acid, and indole-3-butyric acid, however, IAA is the most potent auxin in plants (Woodward and Bartel, 2005). The negative impact of auxin in SA-mediated defense against biotrophic pathogens has recently been demonstrated (Chen et al., 2007; Wang et al., 2007) and a role(s) of auxin in defense against necrotrophic pathogens is also emerging (Kazan and Manners, 2009; Korolev et al., 2008; Llorente et al., 2008). Treatment of plants with the auxin transport inhibitor, 2,3,5-triiodobenzoic acid, has been shown to lead to increased necrotrophic infection by *Plectosphaerella cucumerina* (Llorente et al., 2008). Furthermore, the *aux1* mutant, which is defective in auxin influx, cannot develop induced systemic resistance against *Botrytis cinerea* (Korolev et al., 2008) and auxin signaling mutants, *axr1*, *axr2* and *axr6,* are more susceptible towards *B. cinerea* and *P. cucumerina* than the wild type (Korolev et al., 2008; Llorente et al., 2008). The altered defense response of auxin signaling mutants towards *B. cinerea* and *P. cucumerina* does not appear to be due to altered activation of SA and JA/ethylene defensive pathways as the expression of marker genes *PR1* and *PDF1.2* was not altered. These data suggest that auxin signaling is required for full defense capacity towards *B. cinerea* and *P. cucumerina* in Arabidopsis. The authors speculated that the changes in disease outcome in response to exogenous IAA were not directly caused by the hormone because IAA can induce ethylene and the IAA-induced resistance was not observed in the ethylene insensitive *ein2* mutant (Savatin et al., 2011). In the case of enhanced susceptibility, it was suggested that conversion of IAA to IAA-Asp is responsible (González-Lamothe et al., 2012). The study showed that the infection by *B. cinerea* increases the level of IAA-Asp in Arabidopsis and exogenous application of IAA-Asp and IAA, but not the synthetic auxin, 2,4D, enhanced susceptibility to *B. cinerea* and *Pseudomonas syringae.* Conversely the *gh3.2* mutant, defective in an auxin-conjugating enzyme and as a result has a lower level of IAA-Asp, exhibited enhanced resistance to *B. cinerea* and *P. syringae*. It was found that IAA-Asp promotes the expression of certain virulence genes in the pathogens during infection of plants (González-Lamothe et al., 2012).

It is noteworthy that *B. cinerea* can synthesize auxin and secrete it to the media when grown *in vitro* (Sharon et al., 2007), but the exact function of the pathogen-derived IAA during infection and in interaction with the plant host has not been elucidated. Following infection of Arabidopsis by *B. cinerea*, a number of the host IAA biosynthetic genes were upregulated (Windram et al., 2012) and the concentration of IAA increased (Pan et al., 2008), further supporting involvement of auxin in the response to necrotrophic pathogens. Interestingly, in a meta-analysis of publically available transcriptomic data it was found that many auxin signal-transduction related genes were downregulated upon infection with *B. cinerea* (Llorente et al., 2008). Given the importance of auxin signaling genes for full defense capacity against this pathogen, it might suggest that *B. cinerea* is manipulating the host to downregulate auxin signaling, although at present this is merely a speculation.

2.2. How does auxin impact the cell wall?

Auxin is involved in a number of growth and developmental processes and has a prominent role in the acid growth during cell expansion (McQueen-Mason et al., 1992; Woodward and Bartel, 2005; Zhao, 2010). Auxin lowers pH in the apoplast by activating plasma membrane proton pumps and this is known to activate expansins which causes the cell wall to relax (McQueen-Mason et al., 1992). The exact mechanisms by which expansins mediate creep of cell walls are not well understood (Cosgrove, 1998). They do not alter the covalent linkages in the wall, instead their loosening effect is observed only while the wall is in tension (Cosgrove, 1998). Expression of expansins are also induced by ABA (Zhao et al., 2012), BRs (Sun et al., 2005), gibberellins (Cho and Kende, 1997), cytokinins (Downes and Crowell, 1998), SA, JA and ethylene (Cho and Cosgrove, 2002).

Auxin influences the expression and activity of cell wall modifying enzymes. Auxin treatment induces expansins and pectin methylesterase (PME) activity in roots (Bryan and Newcom, 1954; Laskowski et al., 2006; Yoda, 1958). The most abundant enzymes among extracellular proteins are class III peroxidases (E.C 1.11.1.7). They are key players in secondary cell wall remodeling and have been proposed to act in the polymerization of phenolic monomers into lignins and suberins and to mediate the cross-linking between lignins, polysaccharides and proteins (Passardi et al., 2004). Two classes of peroxidases were isolated from the cell wall of pea roots: ionically bound peroxidases (iPOD) and covalently-bound peroxidases (cPOD), which are hypothesized to mediate cell elongation and cell wall stiffening, respectively (Kukavica et al., 2012). Treatment of pea roots with IAA, which inhibits root elongation, has led to decrease in the iPOD activity and increase in the cPOD activity (Kukavica et al., 2012). While in *Catharanthus roseus* cell suspension cultures the addition of the synthetic auxin 2,4D, which is required for cell division and growth, reduced the POD activity and enhanced the iPOD activity (Limam et al., 1998).

Pectins are highly methyl-esterified during biosynthesis but after secretion into the cell wall they are de-esterified by PMEs (Ridley et al., 2001). De-esterification makes pectin more susceptible to degradation by pathogen-secreted pectic enzymes (Limberg et al., 2000; Lionetti et al., 2007; Raiola et al., 2011), such as polygalacturonases (PGs) that can release elicitor active oligogalacturonides (OGs) as detailed below (Ferrari et al., 2013). A link between auxin signaling and the PME activity modulating cell wall physical properties at the shoot apical meristem was investigated with atomic force microscopy (Braybrook and Peaucelle, 2013). The study showed that local application of IAA led to demethylation of pectin and tissue softening, which in turn affected organ formation.

The Arabidopsis *WALLS ARE THIN 1 (wat1)* mutant, which shows enhanced resistance to *Ralstonia solanacearum* and other vascular pathogens, has reduced secondary wall deposition in fibers and reduced auxin content (Denance et al., 2012; Ranocha et al., 2010). The gene was found to encode an auxin vacuolar transporter, which suggests that auxin has a role in promoting xylem fiber development (Ranocha et al., 2013). However, it seems that defense responses in *wat1* are influenced by SA and not auxin, as the *wat1* mutant had a higher level of SA in roots compared to the wild type and the enhanced resistance could be circumvented by introduction of *NahG*, the bacterial SA-degrading salicylate hydroxylase gene (Denance et al., 2012). In addition, it was shown that the AUXIN BINDING PROTEIN 1 (ABP1) modulates the expression of cell wall related genes and remodeling of hemicellulose xyloglucan side chain structure (Paque et al., 2014). These studies highlight interplays between auxin signaling, cell wall biosynthesis and remodeling.

2.3. Interplay between cell wall, auxin, and resistance to necrotrophic pathogens

Necrotrophic pathogens secrete polygalacturonases and degrade the pectic homogalacturonan, which results in release of short fragments of OGs, effective in eliciting defense responses and thus are known as a damage associated molecular pattern (DAMP) (Cervone et al., 1989). OGs are also generated by the action of endogenous polygalacturonases induced by mechanical damage (Orozco-Cardenas and Ryan, 1999). These OGs can induce a number of defense responses in plants such as accumulation of phytoalexins (Davis et al., 1986), glucanase and chitinase (Broekaert and Pneumas, 1988; Davis and Hahlbrock, 1987), deposition of callose, production of reactive oxygen species (Bellincampi et al., 2000; Galletti et al., 2008), and nitric oxide (Rasul et al., 2012). Notably, exogenous application of OGs enhances resistance to *B. cinerea* in leaves of grape, tobacco and Arabidopsis (Aziz et al., 2004; Ferrari et al., 2007, 2008) and transgenic plants expressing a fungal PG exhibit constitutive activation of defense responses and enhanced resistance to *B. cinerea* (Ferrari et al., 2008). The optimal size of OGs for induction of defense responses is a degree of polymerization between 10 and 15 whereas OGs with a degree of polymerization between 3 and 6 cause developmental impact (Ferrari et al., 2013). Interestingly, the actions of OGs are antagonized by auxin and vice versa. It was shown that OGs inhibit auxin-induced elongation in pea stem segments (Branca et al., 1988), rooting of tobacco explants (Bellincampi et al., 1993), and IAA-induced DR5 expression and up-regulation of early IAA response genes (Savatin et al., 2011). Conversely, enhanced resistance to *B. cinerea* of tobacco plants expressing a fungal endo-PG, which is likely mediated by constitutive generation of OGs, could be abolished by auxin (Ferrari et al., 2008). However, exogenous application of OGs does not alter the level of auxins, thus the mechanisms of OG-auxin antagonisms are still unknown (Savatin et al., 2011; Ferrari et al., 2013).

Plants express PG inhibiting proteins (PGIPs) that slow down the catalytic activity of pathogenś PGs and limit the degradation of homogalacturonan, leading to generation of the elicitor-active OGs (Ferrari et al., 2013). Plants overexpressing PGIPs show enhanced resistance to *B. cinerea* (Agüero et al., 2005; Ferrari et al., 2003; Joubert et al., 2006; Powell et al., 2000). Interestingly, tobacco plants (*Nicotiana tabacum* SR1) overexpressing a grapevine PGIP (Vvpgip1) has an increased level of IAA and a slightly decreased level of SA (Alexandersson et al., 2011). Microarray studies revealed that 219 probes showed downregulation whereas 58 probes showed upregulation in the Vvpgip1 overexpressing line as compared to the wild type, with marked changes in the expression of genes in the following categories: cell wall biogenesis and organization, carbon metabolism, photosynthesis and stress defense signaling (Alexandersson et al., 2011). The transgenic plant had a higher content of lignins and a decreased level of xyloglucan endotransglycosylase activity. A detailed cell wall analysis by carbohydrate microarray polymer profiling revealed that overexpression of Vvpgip1 leads to constitutive compositional changes in the leaf arabinoxyloglucan network (Nguema-Ona et al., 2013). These data suggest that PGIP-induced initial changes in the cell wall lead to altered auxin accumulation and altered stress responses, leading to additional structural and compositional changes in the cell wall.

As described above, auxin modulates the expression of cell-wall modifying enzymes including peroxidases and PMEs. Arabidopsis *PME3* is induced upon fungal infection and is necessary for initial colonization by necrotrophic fungi (Raiola et al., 2011). Importance of the pectin methylesterification status in plant resistance was supported by a recent study that showed the level of pectin methylation decreased upon infection by *P. syringae* or *Alternaria*

brassicicola due to the action of PMEs and several *pme* mutants had enhanced susceptibility to *P. syringae* whereas resistance to *A. brassicicola* was unchanged (Bethke et al., 2014). Aside from PMEs, plant genomes also encode PME inhibiting proteins (PMEIs) to regulate pectin methylesterification. For instance, the Arabidopsis genome encodes two PMEIs, *AtPMEI-1* and *AtPMEI-2* (Raiola et al., 2004) and constitutive expression of *AtPMEI-1* or *AtPMEI-2* in Arabidopsis increases pectin methylesterification and enhanced resistance towards *B. cinerea,* possibly through decreased ability of the fungus to degrade the plant cell wall (Lionetti et al., 2007).

Auxin exerts an impact on the action of nitric oxide (NO). NO is an important signal in plants, it is generated in response to biotic and abiotic stresses and is emerging as an important player in plant–pathogen interaction and signaling (Crawford and Guo, 2005; Mur et al., 2013). Notably, NO has been implicated in the regulation of the cell wall structure and composition (Pacoda et al., 2004). High and low NO concentration inhibit or promote cellulose synthesis, respectively (Correa-Aragunde et al., 2008), and NO application has been found to alleviate cadmium and aluminum stress by altering cell wall composition (Xiong et al., 2009; Zhang et al., 2011). Generation and signaling of NO is modulated by phytohormones; for instance, a synergistic effect between NO and auxin have been observed in a number of developmental and morphological plant responses whereas the interaction between NO and cytokinin is complex and the outcome seems to depend on tissue type and plant species (Freschi, 2013).

3. Cytokinin

In plants, cytokinin can be found in the form of kinetin, zeatin and 6-benzylaminopurine and is involved in cell growth and differentiation (Hwang et al., 2012). Recently, cytokinins have been shown to be involved in nutrient assimilation and defense against pathogens.

3.1. How does cytokinin impact disease resistance?

The fungal pathogen *Plasmodiophora brassicae*, the causal agent of the Brassicaceae clubroot disease, has been shown to downregulate the cytokinin degradation pathway during infection of Arabidopsis, and transgenic overexpression of cytokinin oxidases/dehydrogenases suppressed clubroot development, indicating the importance of cytokinin in the pathogenicity of *P. brassicae* (Siemens et al., 2006). Transgenic plants with increased cytokinin levels exhibited delayed leaf senescence and enhanced resistance to *B. cinerea* infection in tomato (Swartzberg et al., 2008) and enhanced resistance to *A. brassicicola* KACC40036 in Arabidopsis (Choi et al., 2010), whereas in tobacco, increased cytokinin levels had no effect on resistance to *Schlerotinia schlerotiorum* and even enhanced susceptibility to *B. cinerea* (Grosskinsky et al., 2011). It appears that the role of cytokinin varies in different pathosystems, reflecting the outcome of coevolutionary interactions between pathogens and their host plants.

3.2. How does cytokinin impact the cell wall?

Application of cytokinin to plants or plant cell cultures induces expression of many cell wall-related genes such as cell wall loosening expansins (13 expansins, 4 laccases, 6 pectin-modifying enzymes) as well as reactive oxygen species (ROS) producers and scavengers and antioxidants (Brenner et al., 2012). One of the most frequently listed genes in a meta-analysis of cytokinin-induced genes was *EXPANSIN 1* (Brenner et al., 2012). Twelve other expansin genes were found in at least two microarray studies along with 18 other cell wall-related genes (Brenner et al., 2012). Previously, cytokinin was found to regulate an expansin in soy bean (Downes and Crowell, 1998) and a cytokinin-induced change of wall extensibility has also been directly measured (Thomas et al., 1981), whereas a negative influence of cytokinin signaling on cell wall thickness has been reported (Jung et al., 2008). Cytokinin-induced stress genes are suppressed by auxin secreted from *Agrobacterium tumefaciens* (Lee et al., 2009), suggesting that pathogen-derived auxin can specifically suppresses the cytokinin-induced defense response during infection (Choi et al., 2011).

To date, little is known about how alterations in the cell wall affect the cytokinin level in plants. OGs promote cytokinin (benzyladenine, BA)-induced vegetative shoot formation (Falasca et al., 2008). The level of cytokinin was enhanced in alfalfa mutants with lower lignin and constitutive defense responses, but also the levels of SA and JA were enhanced, pointing to an overall deregulation of hormone pathways in this mutants (Gallego-Giraldo et al., 2011).

4. Brassinosteroids

Brassinosteroids (BRs) comprise a class of functionally related steroid hormones where more than 40 variants have been isolated (Khripach et al., 2000). BRs are important for several plant processes such as stimulation of seedling development, second organogenesis and reproductive processes (Ryu and Hwang, 2013). They have been used as growth hormones in fields for many years and several encouraging outcomes have been reported, including improvement of crop yield and quality, resistance to environmental stresses, and in phytoremediation owing to the ability to interfere with plant ion homeostasis (Khripach et al., 2000).

4.1. How do BRs impact disease resistance?

BRs can interfere with pathogen responses mediated by JA, ethylene, SA, auxin, ABA, cytokinin, and gibberellic acid (Choudhary et al., 2012; Nakashita et al., 2003). During virus infection, BR precursors were found to accumulate (Nakashita et al., 2003). Effects of brassinolide, the most biologically active BR, have been studied on defense responses in tobacco and rice during infection with tobacco mosaic virus, *P. syringae pv. tabaci* and powdery mildew (Nakashita et al., 2003). Exogenous application of brassinolide increased plant resistance but did not affect the accumulation of the *PR1*, *PR2* and *PR5* transcripts, indicating that the effect was not mediated by SA signaling. Similarly, exogenous application of brassinolide was found to increase the resistance of cotton towards the necrotrophic fungal pathogen *Verticillium dahlia* (Gao et al., 2013) as well as reducing the virulence of *Xanthomonas oryzae pv. oryzae* and *Magnaporthe grisea* in rice, demonstrating a protective effect of BRs extends to monocot (Nakashita et al., 2003).

A higher resistance against *B. cinerea* was observed in Arabidopsis plants overexpressing 3-hydroxy-3-methylglutaryl-CoA synthase, an enzyme involved in the cytosolic mevalonate pathway that is important for providing precursors of several isoprenoids including BRs (Wang et al., 2012). The transgenic overexpression line exhibited a higher sterol content in leaves, higher expression of the last enzyme in the BR biosynthetic pathway (BR6OX2), higher resistance to *B. cinerea,* and reduced occurrence of cell death induced by hydrogen peroxide. The authors hypothesized that the higher resistance was due to activation of SA signaling pathway since SA-dependent genes (*PR1*, *PR2* and *PR5*) were upregulated. JA responsible genes were concomitantly upregulated, suggesting that BR-induced protection may also be mediated by JA. Notably, when the negative regulators of BR signaling, *GBL4-3-3c* and *GBL4-3-3d*, were downregulated by virus-induced gene silencing,

the transgenic cotton were found to be more resistant to the pathogen, underpinning the importance of BRs in plant resistance.

Direct alteration of MAMP-triggered immunity (MTI) by BRs has been investigated. Briefly, in the brassinosteroid perception and signaling pathway, BRs bind the BR receptor BRI1 (BRASSINOSTROID INSENSITIVE 1) in the plasmamembrane (Kinoshita et al., 2005) and trigger dissociation of BRI1-inhibitory protein (BKI1), allowing the complex formation with BAK1 (BRI1-ASSOCIATED RECEPTOR KINASE1), a coreceptor of BRI1 (Hao et al., 2013; Wang et al., 2008). Phosphorylation between BRI1 and BAK1 fully activates the BRI1 kinase activity, which, through a signal transduction cascade, ultimately leads to activation of two transcriptional factors, BES1 and BZR1, that control the expression of BR responsible genes (Wang et al., 2008). Apart from the BR signaling pathway, BAK1 is also involved in MTI through protein complex formation with FLS2 (FLAGELLIN-SENSING 2) and BIK1 (BOTRYTIS-INDUCED KINASE 1) (Belkhadir et al., 2012). It was shown that overexpression of BRI1 in Arabidopsis can inhibit MTI by titrating BAK1 away from forming the complex with FLS2 and BIK1. However, increased signaling in the BR pathway triggered by the hyperactive allele $BRI1^{sud}$ did not suppress MTI, rather it enhanced MTI as compared to the wild type plants. Interestingly, the $BRI1^{sud}$ enhancement of MTI defenses was BAK1 dependent, suggesting that BAK1 may mediate the positive effect of BR signaling on plant immunity (Belkhadir et al., 2012). The exact molecular mechanism by which BR signaling enhances MTI in a BAK1 dependent manner remains to be elucidated. To add more complexity to the interaction between BR signaling and MTI, it was found that brassinolide treatment hindered MTI in a loss of function *bak1-4* background (Albrecht et al., 2012). This outcome suggested that modulation of MTI caused by BR signaling may also be BAK1-independent or further downstream in the pathway.

4.2. How do BRs impact the cell wall?

BRs have been shown to exert notable impacts on cell wall properties. Transcriptomic analysis revealed that, in Arabidopsis, BR treatment upregulated a variety of genes involved in cell wall elongation and remodeling including xyloglucan endotransglycosylases, PMEs, putative expansins, putative pectin acetylesterases, endo-xylanases and others (Goda et al., 2002). Together with other microarray data (Mussig et al., 2002; Yin et al., 2002), these discoveries underpin the impacts of BRs on plant cell walls. It was found that BR-deficient or BR-perception deficient mutants contain less cellulose than the wild type (Xie et al., 2011). The expression of *CESA* genes, especially those involved in the primary cell wall synthesis, was reduced in *det2-1* and *bri1-301*, which was restored by application of BRs. Chromatin immunoprecipitation experiments showed that the BR-activated transcription factor BES1 can associate with upstream elements of the *CESA* genes involved in the primary cell wall synthesis. Furthermore, it was found that BIL2, a protein involved in BR signaling and BR gene expression, is required for cell elongation, and overexpression of BIL2 resulted in cell elongation even in the presence of BR biosynthesis inhibitors, suggesting that BIL2 functions downstream of BR reception (Bethke et al., 2014). BIL2 was found to be a mitochondria-localized Dan/Heat shock protein 40 (DnaJ/Hsp40) family protein involved in protein folding (Bekh-Ochir et al., 2013). It is speculated that BIL2 is required for proper folding of ATPase in mitochondria, while how this impacts the cell wall gene expression remains unknown.

4.3. How does altering cell wall impact BRs and defense capacity?

A direct impact of cell wall modification in BR synthesis or signaling is emerging (Wolf et al., 2012). It was recently found that alteration of Arabidopsis cell wall architecture by increasing the pectin methylesterification state either genetically (PMEI5 ectopic overexpression) or pharmacologically results in a number of growth phenotypes including cell swelling, root waving and twisted organs. Through a forward genetic screen, Höfte and colleagues identified that a mutation in the brassinosteroid receptor (BRI1) was responsible for suppressing the growth phenotypes caused by the altered cell wall methylesterification. As the pectin methylesterification level reached abnormally high levels, the stiffness of the cell wall increased due to the higher Ca^{2+} cross-links and root tissue exhibited loss of cell wall integrity. The assumption is that this triggers the BR signaling system leading to the BR-mediated expression of cell wall loosening genes and at least two PMEs in order to re-establish optimal cell wall architecture (Wolf et al., 2012).

Current evidence clearly indicates that the pectin methylesterification status is linked to the intracellular BR signaling cascade and that BR signaling homeostasis has a profound impact on cell wall biosynthesis, *e.g.* cellulose, and resistance against pathogens in a range of host-pathogen systems. Given the existing data, it is to be expected that the PMEI5 overexpressing Arabidopsis is to exhibit a higher resistance against at least *B. cinerea*, because of lower digestibility of the cell wall and elevation of the BR signaling, and possibly, increased cellulose deposition that may hinder the pathogen progress, although this assumption remains to be tested.

5. Abscisic acid (ABA)

Abscisic acid (ABA) is a plant hormone that plays important roles in regulating drought, cold stress and osmotic stress responses. Moreover, it plays a role in promoting seed dormancy while antagonizing the growth-inducing effects of phytohormones like gibberellin (Ng et al., 2014). As described below, several studies have demonstrated a negative role of ABA in plant resistance against necrotrophic pathogens. As in the case of BRs, ABA interferes with the other hormone signaling pathways.

5.1. How does ABA impact disease resistance?

Currently roles of ABA in plant resistance towards pathogens are better understood in the context of biotrophic interactions than necrotrophic interactions. ABA can promote resistance through its ability to induce stomata closure, thus interfering with pathogen entry (Lopez et al., 2008). On the other hand, ABA may promote pathogen virulence as increasing ABA signaling or exogenous ABA application enhanced *P. syringae* growth and vice versa (de Torres-Zabala et al., 2007). It was suggested that *P. syringae* effectors are able to modulate ABA signaling, leading to a favorable environment for infection. Callose deposition is a hallmark of inducible cell wall fortification upon pathogen infection (Huckelhoven, 2007; Ton and Mauch-Mani, 2004). ABA promotes the callose formation through repression of a callase gene, *PR2*. Overexpression of PR2 in the *pad3* mutant background of Arabidopsis reduced the callose content and further enhanced susceptibility towards *B. cinerea*, *A. brassica*, and hemibiotrophic *Leptosphaeria maculans* as compared to the *pad3* mutant (Oide et al., 2013). The effect of ABA on plant interaction against necrotrophic pathogens appears to be complex. Upon ABA treatment enhanced resistance of Arabidopsis was observed against the necrotrophic pathogens *A. brassicicola* and *P. cucumerina* (Ton and Mauch-Mani, 2004). It was suggested that the increased resistance was mediated by enhanced callose production. While *sitiens,* an ABA deficient mutant of tomato, is more resistant to *B. cinerea* (Asselbergh et al., 2007; Audenaert et al., 2002; Curvers et al., 2010) and exogenous application of ABA increased susceptibility

towards the fungus (Audenaert et al., 2002). It was suggested that the more permeable cell wall and cutin layer in *sitiens* may lead to faster diffusion of DAMPs or MAMPs and faster defense activation (Curvers et al., 2010). The same study also showed the differential methylesterification state of the cell wall in *sitiens* as compared to the wild type and that incubation of *B. cinerea* inoculation droplets on the leaves of the wild type and *sitiens* led to the release of oligosaccharides with different profiles, which may suggest that *sitiens* generates more bioactive OGs. In Arabidopsis, interplay between ABA, cuticle permeability and ROS has been also found to affect resistance against *B. cinerea* (L'Haridon et al., 2011). ABA biosynthesis mutants (*aba2* and *aba3*) were more resistant to *B. cinerea*, had higher cuticle permeability as well as higher ROS accumulation. Higher resistance against *B. cinerea* was also observed in cuticular mutants with enhanced surface permeability (L'Haridon et al., 2011). This outcome was explained as higher cuticle permeability in ABA mutants facilitates sensing of elicitors and therefore triggers faster ROS production.

Arabidopsis mutants defective in ABA synthesis or signaling were more resistant to the necrotrophic pathogen *P. cucumerina* but were more susceptible to *R. solanacearum* (Hernandez-Blanco et al., 2007; Sanchez-Vallet et al., 2012). Disrupting ABA synthesis or signaling has led to upregulation of defense marker genes (*PDF1.2*, *PR1*, *PR4*, and *ORA59*). Moreover, genetic upregulation of ABA signaling in the triple *hab1-1 abi1-2 abi2-2* mutant increased susceptibility of Arabidopsis towards *P. cucumerina* (Rubio et al., 2009). Interestingly, no correlation between the ABA mutants and ROS levels were observed (Sanchez-Vallet et al., 2012). The increased resistance to *P. cucumerina* was partly mediated by ethylene, SA and JA since mutations in these signaling pathways reverted the resistant phenotype of the ABA biosynthetic mutant.

5.2. How does ABA impact the cell wall?

A number of reports have described the roles of ABA in altering cell wall properties and compositions. ABA treatment was shown to induce expression of the cell wall loosening gene *EXPANSIN-LIKE A2* in Arabidopsis (Abuqamar et al., 2013). Mutations in this gene increased resistance to *B. cinerea* and oxidative stress, suggesting a link between ABA, the cell wall, and resistance towards *B. cinerea* in Arabidopsis (Abuqamar et al., 2013). Exogenously applied ABA induced enhanced accumulation of pectic arabinan in root meristem of Arabidopsis (Talboys et al., 2011). Because arabinan is involved in the cell-to-cell adhesion (Iwai et al., 2001), mechanical property of the cell wall (Ulvskov et al., 2005; Verhertbruggen et al., 2013), and that endo-arabinanase activity is required for the full virulence of *B. cinerea* (Nafisi et al., 2014), it is plausible that ABA-mediated change in arabinan too may have a role in influence the outcome of necrotrophic infection. ABA deficient tomato mutant, *sitiens*, exhibits several notable changes related to the cell wall: an elevated crosslinking in the cell wall as a result of a faster induction and enhanced level of ROS burst during infection with *B. cinerea,* and altered expression of cell wall modifications genes including pectin esterase and xyloglucan endotransglycosylase (Asselbergh et al., 2007). In addition, Curvers et al. (2010) observed that the *sitiens* has an abnormal cuticle and cell wall deposition and its leaves are more resistant to pectinase treatment. Sanchez-Vallet et al. (2012) found that the *aba1-6* mutant of Arabidopsis with impaired ABA biosynthesis displays an alteration in cell wall structure and composition by Fourier transform infrared spectroscopy. Interestingly, however, mutants with disrupted ABA signaling did not display the same differences in cell wall structure or composition. In contrast, an ABA signaling mutant, *abi8*, identified through a genetic screening exhibited stunted growth and was found to be allelic to *kobito1* (Pagant et al., 2002) and *elongation defective 1* (Cheng et al., 2000), which are impaired in cellulose synthesis cell elongation, respectively (Brocard-Gifford et al., 2004).

5.3. How does altering cell wall impact ABA and pathogen resistance?

In a forward genetic screen, a mutant of Arabidopsis, *leaf wilting 2* (*lew2*) that exhibits higher drought and salt tolerance than the wild type was identified (Chen et al., 2005). *lew2* was found to be allelic to *irx1/cesA8* (*IRREGULAR XYLEM1*) and retained the collapsed xylems phenotype characteristics of *irx* mutants (Turner and Somerville, 1997). These *irx* mutants are defective in secondary cell wall biosynthesis and exhibit collapsed xylems because their thinner cell walls are not able to cope with the negative pressure generated by water transport in the xylem. *lew2/irx1/cesA8* was shown to overaccumulate ABA and soluble sugars, which permitted higher fitness with respect to abiotic stresses. Despite these observations, it is unclear whether the increased ABA accumulation is due to a direct effect of cell wall alteration or due to the triggering of drought stress responses caused by the impaired water transport though the xylem.

A link between the secondary cell wall cellulose synthesis, ABA, and pathogen resistance has been identified (Hernandez-Blanco et al., 2007). Mutations in the secondary cell wall cellulose synthases, CESA4/IRX5, CESA7/IRX3, and CESA8/IRX1, resulted in increased resistance against *R. solanacearum* and *P. cucumerina*. Transcriptomics analysis of the mutants indicated upregulation of several genes involved in ABA signaling as well as defense-related genes (e.g. antimicrobial compounds and PR proteins). Mutants defective in ABA signaling (*abi1-1*, *abi2-1*) and biosynthesis (*aba1-6*) showed increased susceptibility to *R. solanacearum* and enhanced resistance to *P. cucumerina*. It was proposed that the increased resistance observed for the *cesA4/irx4*, *cesA7/irx3*, and *cesA1/irx8/lew2* mutants against the pathogens was due to possible accumulation of the secondary metabolites glucosinolates and camalexin based on the transcriptional upregulation of *CYP79B2* and *CYP79B3* involved in the biosynthesis of these compounds.

6. Conclusions

Based on the mounting evidence it is unmistakable that phytohormones and cell wall integrity are highly interconnected and control plant growth and development. Degradation of plant cell walls by necrotrophic phytopathogens is sensed by the host through DAMP recognition and likely through the cell wall integrity sensory systems. These events lead to activation of signaling cascades involving phytohormones crosstalks and consequently activate expression of defense related genes. Moreover, phytohormones exert a considerable impact on the cell wall composition and structure, altering not only the ability of necrotrophic phytopathogens to digest the cell wall but also the capacity of the cell wall to generate DAMP and possibly to activate the cell wall integrity sensory systems. These processes are highly integrated and likely to form a cell wall-phytohormone homeostasis that plays an important role in plant–necrotrophic pathogen interaction.

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References

Abuqamar, S., Ajeb, S., Sham, A., Enan, M.R., Iratni, R., 2013. A mutation in the expansin-like A2 gene enhances resistance to necrotrophic fungi and hypersensitivity to abiotic stress in *Arabidopsis thaliana*. Mol. Plant Pathol. 14, 813–827.

Agüero, C.B., Uratsu, S.L., Greve, C., Powell, A.L., Labavitch, J.M., Meredith, C.P., Dandekar, A.M., 2005. Evaluation of tolerance to Pierce's disease and *Botrytis* in transgenic plants of *Vitis vinifera* L. expressing the pear PGIP gene. Mol. Plant Pathol. 6, 43–51.

Albrecht, C., Boutrot, F., Segonzac, C., Schwessinger, B., Gimenez-Ibanez, S., Chinchilla, D., Rathjen, J.P., de Vries, S.C., Zipfel, C., 2012. Brassinosteroids inhibit pathogen-associated molecular pattern-triggered immune signaling independent of the receptor kinase BAK1. Proc. Natl. Acad. Sci. U.S.A. 109, 303–308.

Alexandersson, E., Becker, J.V., Jacobson, D., Nguema-Ona, E., Steyn, C., Denby, K.J., Vivier, M.A., 2011. Constitutive expression of a grapevine polygalacturonase-inhibiting protein affects gene expression and cell wall properties in uninfected tobacco. BMC Res. Notes 4, 493.

Amselem, J., Cuomo, C.A., van Kan, J.A., Viaud, M., Benito, E.P., Couloux, A., Coutinho, P.M., de Vries, R.P., Dyer, P.S., Fillinger, S., Fournier, E., Gout, L., Hahn, M., Kohn, L., Lapalu, N., Plummer, K.M., Pradier, J.M., Quevillon, E., Sharon, A., Simon, A., ten Have, A., Tudzynski, B., Tudzynski, P., Wincker, P., Andrew, M., Anthouard, V., Beever, R.E., Beffa, R., Benoit, I., Bouzid, O., Brault, B., Chen, Z., Choquer, M., Collemare, J., Cotton, P., Danchin, E.G., Da Silva, C., Gautier, A., Giraud, C., Giraud, T., Gonzalez, C., Grossetete, S., Guldener, U., Henrissat, B., Howlett, B.J., Kodira, C., Kretschmer, M., Lappartient, A., Leroch, M., Levis, C., Mauceli, E., Neuveglise, C., Oeser, B., Pearson, M., Poulain, J., Poussereau, N., Quesneville, H., Rascle, C., Schumacher, J., Segurens, B., Sexton, A., Silva, E., Sirven, C., Soanes, D.M., Talbot, N.J., Templeton, M., Yandava, C., Yarden, O., Zeng, Q., Rollins, J.A., Lebrun, M.H., Dickman, M., 2011. Genomic analysis of the necrotrophic fungal pathogens *Sclerotinia sclerotiorum* and *Botrytis cinerea*. PLoS Genet. 7, e1002230.

Asselbergh, B., Curvers, K., Franca, S.C., Audenaert, K., Vuylsteke, M., Van Breusegem, F., Hofte, M., 2007. Resistance to *Botrytis cinerea* in sitiens, an abscisic acid-deficient tomato mutant, involves timely production of hydrogen peroxide and cell wall modifications in the epidermis. Plant Physiol. 144, 1863–1877.

Audenaert, K., De Meyer, G.B., Hofte, M.M., 2002. Abscisic acid determines basal susceptibility of tomato to *Botrytis cinerea* and suppresses salicylic acid-dependent signaling mechanisms. Plant Physiol. 128, 491–501.

Aziz, A., Heyraud, A., Lambert, B., 2004. Oligogalacturonide signal transduction, induction of defense-related responses and protection of grapevine against *Botrytis cinerea*. Planta 218, 767.

Bekh-Ochir, D., Shimada, S., Yamagami, A., Kanda, S., Ogawa, K., Nakazawa, M., Matsui, M., Sakuta, M., Osada, H., Asami, T., Nakano, T., 2013. A novel mitochondrial DnaJ/Hsp40 family protein BIL2 promotes plant growth and resistance against environmental stress in brassinosteroid signaling. Planta 237, 1509–1525.

Belkhadir, Y., Jaillais, Y., Epple, P., Balsemao-Pires, E., Dangl, J.L., Chory, J., 2012. Brassinosteroids modulate the efficiency of plant immune responses to microbe-associated molecular patterns. Proc. Natl. Acad. Sci. U.S.A. 109, 297–302.

Bellincampi, D., Dipierro, N., Salvi, G., Cervone, F., De Lorenzo, G., 2000. Extracellular H_2O_2 induced by oligogalacturonides is not involved in the inhibition of the auxin-regulated *rolB* gene expression in tobacco leaf explants. Plant Physiol. 122, 1379–1385.

Bellincampi, D., Salvi, G., Delorenzo, G., Cervone, F., Marfa, V., Eberhard, S., Darvill, A., Albersheim, P., 1993. Oligogalacturonides inhibit the formation of roots on tobacco explants. Plant J. 4, 207–213.

Bethke, G., Grundman, R.E., Sreekanta, S., Truman, W., Katagiri, F., Glazebrook, J., 2014. Arabidopsis pectin esterases contribute to immunity against *Pseudomonas syringae*. Plant Physiol. 164, 1093–1107.

Bradley, D.J., Kjellbom, P., Lamb, C.J., 1992. Elicitor-induced and wound-induced oxidative cross-linking of a proline-rich plant-cell wall protein – a novel, rapid defense response. Cell 70, 21–30.

Branca, C., Delorenzo, G., Cervone, F., 1988. Competitive-inhibition of the auxin-induced elongation by alpha-D-oligogalacturonides in peastem segments. Physiol. Plant. 72, 499–504.

Braybrook, S.A., Peaucelle, A., 2013. Mechano-chemical aspects of organ formation in *Arabidopsis thaliana*: the relationship between auxin and pectin. PLoS ONE 8, e57813.

Brenner, W.G., Ramireddy, E., Heyl, A., Schmulling, T., 2012. Gene regulation by cytokinin in Arabidopsis. Front. Plant Sci. 3.

Brocard-Gifford, I., Lynch, T.J., Garcia, M.E., Malhotra, B., Finkelstein, R.R., 2004. The *Arabidopsis thaliana* ABSCISIC ACID-INSENSITIVE8 locus encodes a novel protein mediating abscisic acid and sugar responses essential for growth. Plant Cell 16, 406–421.

Broekaert, W.F., Pneumas, W.J., 1988. Pectic polysaccharides elicit chitinase accumulation in tobacco. Physiol. Plant. 74, 740–744.

Bryan, W.H., Newcom, K.H., 1954. Stimulation of pectin methylesterase activity of cultured tobacco pith by indoleacetic acid. Physiol. Plant., 290–297.

Cao, F.Y., Yoshioka, K., Desveaux, D., 2011. The roles of ABA in plant–pathogen interactions. J. Plant. Res. 124, 489–499.

Carpita, N.C., Gibeaut, D.M., 1993. Structural models of primary cell walls in flowering plants: consistency of molecular structure with the physical properties of the walls during growth. Plant J. 3, 1–30.

Cervone, F., Hahn, M.G., Delorenzo, G., Darvill, A., Albersheim, P., 1989. Host-pathogen interactions. A plant protein converts a fungal pathogenesis factor into an elicitor of plant defense responses. Plant Physiol. 90, 542–548.

Chen, Z., Agnew, J.L., Cohen, J.D., He, P., Shan, L., Sheen, J., Kunkel, B.N., 2007. *Pseudomonas syringae* type III effector AvrRpt2 alters *Arabidopsis thaliana* auxin physiology. Proc. Natl. Acad. Sci. U.S.A. 104, 20131–20136.

Chen, Z.Z., Hong, X.H., Zhang, H.R., Wang, Y.Q., Li, X., Zhu, J.K., Gong, Z.Z., 2005. Disruption of the cellulose synthase gene, AtCesA8/IRX1, enhances drought and osmotic stress tolerance in Arabidopsis. Plant J. 43, 273–283.

Cheng, J.C., Lertpiriyapong, K., Wang, S., Sung, Z.R., 2000. The role of the Arabidopsis ELD1 gene in cell development and photomorphogenesis in darkness. Plant Physiol. 123, 509–520.

Cho, H.T., Cosgrove, D.J., 2002. Regulation of root hair initiation and expansin gene expression in Arabidopsis. Plant Cell 14, 3237–3253.

Cho, H.T., Kende, H., 1997. Expression of expansin genes is correlated with growth in deepwater rice. Plant Cell 9, 1661–1671.

Choi, J., Choi, D., Lee, S., Ryu, C.M., Hwang, I., 2011. Cytokinins and plant immunity: old foes or new friends? Trends Plant Sci. 16, 388–394.

Choi, J., Huh, S.U., Kojima, M., Sakakibara, H., Paek, K.H., Hwang, I., 2010. The cytokinin-activated transcription factor ARR2 promotes plant immunity via TGA3/NPR1-dependent salicylic acid signaling in Arabidopsis. Dev. Cell 19, 284–295.

Choudhary, S.P., Yu, J.Q., Yamaguchi-Shinozaki, K., Shinozaki, K., Tran, L.S.P., 2012. Benefits of brassinosteroid crosstalk. Trends Plant Sci. 17, 594–605.

Correa-Aragunde, N., Lombardo, C., Lamattina, L., 2008. Nitric oxide: an active nitrogen molecule that modulates cellulose synthesis in tomato roots. New Phytol. 179, 386–396.

Cosgrove, D.J., 1998. Cell wall loosening by expansins. Plant Physiol. 118, 333–339.

Crawford, N.M., Guo, F.Q., 2005. New insights into nitric oxide metabolism and regulatory functions. Trends Plant Sci. 10, 195–200.

Curvers, K., Seifi, H., Mouille, G., de Rycke, R., Asselbergh, B., Van Hecke, A., Vanderschaeghe, D., Hofte, H., Callewaert, N., Van Breusegem, F., Hofte, M., 2010. Abscisic acid deficiency causes changes in cuticle permeability and pectin composition that influence tomato resistance to *Botrytis cinerea*. Plant Physiol. 154, 847–860.

Davis, K.R., Darvill, A.G., Albersheim, P., Dell, A., 1986. Host–pathogen interactions. characterization of elicitors of phytoalexin accumulation in soybean released from soybean cell-walls by endopolygalacturonic acid lyase. Z. Naturforsch. C. 41, 39–48.

Davis, K.R., Hahlbrock, K., 1987. Induction of defense responses in cultured parsley cells by plant-cell wall fragments. Plant Physiol. 84, 1286–1290.

De Bruyne, L., Hofte, M., De Vleesschauwer, D., 2014. Connecting growth and defense: the emerging roles of brassinosteroids and gibberellins in plant innate immunity. Mol. Plant 7, 943–959.

de Torres-Zabala, M., Truman, W., Bennett, M.H., Lafforgue, G., Mansfield, J.W., Egea, P.R., Bogre, L., Grant, M., 2007. *Pseudomonas syringae* pv. tomato hijacks the Arabidopsis abscisic acid signalling pathway to cause disease. EMBO J. 26, 1434–1443.

De Vleesschauwer, D., Gheysen, G., Hofte, M., 2013. Hormone defense networking in rice: tales from a different world. Trends Plant Sci. 18, 555–565.

Denance, N., Ranocha, P., Oria, N., Barlet, X., Riviere, M.P., Yadeta, K.A., Hoffmann, L., Perreau, F., Clement, G., Maia-Grondard, A., van den Berg, G.C., Savelli, B., Fournier, S., Aubert, Y., Pelletier, S., Thomma, B.P., Molina, A., Jouanin, L., Marco, Y., Goffner, D., 2012. Arabidopsis wat1 (walls are thin1)-mediated resistance to the bacterial vascular pathogen, *Ralstonia solanacearum*, is accompanied by cross-regulation of salicylic acid and tryptophan metabolism. Plant J..

Downes, B.P., Crowell, D.N., 1998. Cytokinin regulates the expression of a soybean beta-expansin gene by a post-transcriptional mechanism. Plant Mol. Biol. 37, 437–444.

Eggert, D., Naumann, M., Reimer, R., Voigt, C.A., 2014. Nanoscale glucan polymer network causes pathogen resistance. Sci. Rep. 4, 4159.

Falasca, G., Capitani, F., Della Rovere, F., Zaghi, D., Franchin, C., Biondi, S., Altamura, M.M., 2008. Oligogalacturonides enhance cytokinin-induced vegetative shoot formation in tobacco explants, inhibit polyamine biosynthetic gene expression, and promote long-term remobilisation of cell calcium. Planta 227, 835–852.

Ferrari, S., Galletti, R., Denoux, C., De Lorenzo, G., Ausubel, F.M., Dewdney, J., 2007. Resistance to *Botrytis cinerea* induced in Arabidopsis by elicitors is independent of salicylic acid, ethylene, or jasmonate signaling but requires PHYTOALEXIN DEFICIENT3. Plant Physiol. 144, 367–379.

Ferrari, S., Galletti, R., Pontiggia, D., Manfredini, C., Lionetti, V., Bellincampi, D., Cervone, F., De Lorenzo, G., 2008. Transgenic expression of a fungal endo-polygalacturonase increases plant resistance to pathogens and reduces auxin sensitivity. Plant Physiol. 146, 669–681.

Ferrari, S., Plotnikova, J.M., De Lorenzo, G., Ausubel, F.M., 2003. Arabidopsis local resistance to *Botrytis cinerea* involves salicylic acid and camalexin and requires EDS4 and PAD2, but not SID2, EDS5 or PAD4. Plant J. 35, 193–205.

Ferrari, S., Savatin, D.V., Sicilia, F., Gramegna, G., Cervone, F., Lorenzo, G.D., 2013. Oligogalacturonides: plant damage-associated molecular patterns and regulators of growth and development. Front. Plant Sci. 4, 49.

Franke, R., Briesen, I., Wojciechowski, T., Faust, A., Yephremov, A., Nawrath, C., Schreiber, L., 2005. Apoplastic polyesters in Arabidopsis surface tissues – a typical suberin and a particular cutin. Phytochemistry 66, 2643–2658.
Freschi, L., 2013. Nitric oxide and phytohormone interactions: current status and perspectives. Front. Plant Sci. 4, 398.
Fry, S.C., Aldington, S., Hetherington, P.R., Aitken, J., 1993. Oligosaccharides as signals and substrates in the plant cell wall. Plant Physiol. 103, 1–5.
Gallego-Giraldo, L., Escamilla-Trevino, L., Jackson, L.A., Dixon, R.A., 2011. Salicylic acid mediates the reduced growth of lignin down-regulated plants. Proc. Natl. Acad. Sci. U.S.A. 108, 20814–20819.
Galletti, R., Denoux, C., Gambetta, S., Dewdney, J., Ausubel, F.M., De Lorenzo, G., Ferrari, S., 2008. The AtrbohD-mediated oxidative burst elicited by oligogalacturonides in Arabidopsis is dispensable for the activation of defense responses effective against *Botrytis cinerea*. Plant Physiol. 148, 1695–1706.
Gao, W., Long, L., Zhu, L.F., Xu, L., Gao, W.H., Sun, L.Q., Liu, L.L., Zhang, X.L., 2013. Proteomic and cirus-induced gene silencing (VIGS) analyses reveal that gossypol, brassinosteroids, and jasmonic acid contribute to the resistance of cotton to *Verticillium dahliae*. Mol. Cell. Proteomics 12, 3690–3703.
Goda, H., Shimada, Y., Asami, T., Fujioka, S., Yoshida, S., 2002. Microarray analysis of brassinosteroid-regulated genes in Arabidopsis. Plant Physiol. 130, 1319–1334.
González-Lamothe, R., El Oirdi, M., Brisson, N., Bouarab, K., 2012. The conjugated auxin indole-3-acetic acid-aspartic acid promotes plant disease development. Plant Cell 24, 762–777.
Grant, M.R., Jones, J.D.G., 2009. Hormone (dis)harmony moulds plant health and disease. Science 324, 750–752.
Grosskinsky, D.K., Naseem, M., Abdelmohsen, U.R., Plickert, N., Engelke, T., Griebel, T., Zeier, J., Novak, O., Strnad, M., Pfeifhofer, H., van der Graaff, E., Simon, U., Roitsch, T., 2011. Cytokinins mediate resistance against *Pseudomonas syringae* in tobacco through increased antimicrobial phytoalexin synthesis independent of salicylic acid signaling. Plant Physiol. 157, 815–830.
Hamann, T., 2012. Plant cell wall integrity maintenance as an essential component of biotic stress response mechanisms. Front. Plant Sci. 3, 77.
Hao, J.J., Yin, Y.H., Fei, S.Z., 2013. Brassinosteroid signaling network: implications on yield and stress tolerance. Plant Cell Rep. 32, 1017–1030.
Hernandez-Blanco, C., Feng, D.X., Hu, J., Sanchez-Vallet, A., Deslandes, L., Llorente, F., Berrocal-Lobo, M., Keller, H., Barlet, X., Sanchez-Rodriguez, C., Anderson, L.K., Somerville, S., Marco, Y., Molina, A., 2007. Impairment of cellulose synthases required for Arabidopsis secondary cell wall formation enhances disease resistance. Plant Cell 19, 890–903.
Huckelhoven, R., 2007. Cell wall-associated mechanisms of disease resistance and susceptibility. Annu. Rev. Phytopathol. 45, 101–127.
Hwang, I., Sheen, J., Müller, B., 2012. Cytokinin signaling networks. Annu. Rev. Plant Biol. 63, 353–380.
Iwai, H., Ishii, T., Satoh, S., 2001. Absence of arabinan in the side chains of the pectic polysaccharides strongly associated with cell walls of *Nicotiana plumbaginifolia* non-organogenic callus with loosely attached constituent cells. Planta 213, 907–915.
Joubert, D.A., Slaughter, A.R., Kemp, G., Becker, J.V., Krooshof, G.H., Bergmann, C., Benen, J., Pretorius, I.S., Vivier, M.A., 2006. The grapevine polygalacturonase-inhibiting protein (VvPGIP1) reduces *Botrytis cinerea* susceptibility in transgenic tobacco and differentially inhibits fungal polygalacturonases. Transgenic Res. 15, 687–702.
Jung, K.W., Oh, S.I., Kim, Y.Y., Yoo, K.S., Cui, M.H., Shin, J.S., 2008. Arabidopsis histidine-containing phosphotransfer factor 4 (AHP4) negatively regulates secondary wall thickening of the anther endothecium during flowering. Mol. Cells 25, 294–300.
Kazan, K., Manners, J.M., 2009. Linking development to defense: auxin in plant–pathogen interactions. Trends Plant Sci. 14, 373–382.
Khripach, V., Zhabinskii, V., De Groot, A., 2000. Twenty years of brassinosteroids: steroidal plant hormones warrant better crops for the XXI century. Ann. Bot. 86, 441–447.
Kinoshita, T., Caño-Delgado, A., Seto, H., Hiranuma, S., Fujioka, S., Yoshida, S., Chory, J., 2005. Binding of brassinosteroids to the extracellular domain of plant receptor kinase BRI1. Nature 433, 167–171.
Korolev, N., David, D.R., Elad, Y., 2008. The role of phytohormones in basal resistance and *Trichoderma*-induced systemic resistance to *Botrytis cinerea* in *Arabidopsis thaliana*. Biocontrol 53, 667–683.
Kukavica, B.M., Veljovic-Jovanovic, S.D., Menckhoff, L., Luthje, S., 2012. Cell wall-bound cationic and anionic class III isoperoxidases of pea root: biochemical characterization and function in root growth. J. Exp. Bot. 63, 4631–4645.
L'Haridon, F., Besson-Bard, A., Binda, M., Serrano, M., Abou-Mansour, E., Balet, F., Schoonbeek, H.J., Hess, S., Mir, R., Leon, J., Lamotte, O., Metraux, J.P., 2011. A permeable cuticle is associated with the release of reactive oxygen species and induction of innate immunity. PLoS Pathog. 7.
Laluk, Mengiste, 2010. Necrotroph attack on plants: wanton destruction or covert extortion? Arabidopsis book, e0136.
Lamb, C., Dixon, R.A., 1997. The oxidative burst in plant disease resistance. Annu. Rev. Plant Phys. 48, 251–275.
Laskowski, M., Biller, S., Stanley, K., Kajstura, T., Prusty, R., 2006. Expression profiling of auxin-treated Arabidopsis roots: toward a molecular analysis of lateral root emergence. Plant Cell Physiol. 47, 788–792.
Lee, C.W., Efetova, M., Engelmann, J.C., Kramell, R., Wasternack, C., Ludwig-Müller, J., Hedrich, R., Deeken, R., 2009. *Agrobacterium tumefaciens* promotes tumor induction by modulating pathogen defense in *Arabidopsis thaliana*. Plant Cell 21, 2948–2962.
Limam, F., Chahed, K., Ouelhazi, N., Ghrir, R., Ouelhazi, L., 1998. Phytohormone regulation of isoperoxidases in *Catharanthus roseus* suspension cultures. Phytochemistry 49, 1219–1225.
Limberg, G., Korner, R., Buchholt, H.C., Christensen, T.M.I.E., Roepstorff, P., Mikkelsen, J.D., 2000. Analysis of pectin structure part 1 – analysis of different de-esterification mechanisms for pectin by enzymatic fingerprinting using endo-pectin lyase and endopolygalacturonase II from *A. niger*. Carbohyd. Res. 327, 293–307.
Lionetti, V., Raiola, A., Camardella, L., Giovane, A., Obel, N., Pauly, M., Favaron, F., Cervone, F., Bellincampi, D., 2007. Overexpression of pectin methylesterase inhibitors in Arabidopsis restricts fungal infection by *Botrytis cinerea*. Plant Physiol. 143, 1871–1880.
Llorente, F., Muskett, P., Sanchez-Vallet, A., Lopez, G., Ramos, B., Sanchez-Rodriguez, C., Jorda, L., Parker, J., Molina, A., 2008. Repression of the auxin response pathway increases Arabidopsis susceptibility to necrotrophic fungi. Mol. Plant 1, 496–509.
Lopez, M.A., Bannenberg, G., Castresana, C., 2008. Controlling hormone signaling is a plant and pathogen challenge for growth and survival. Curr. Opin. Plant Biol. 11, 420–427.
Luna, E., Pastor, V., Robert, J., Flors, V., Mauch-Mani, B., Ton, J., 2011. Callose deposition: a multifaceted plant defense response. Mol. Plant Microbe Interact. 24, 183–193.
Malinovsky, F.G., Fangel, J.U., Willats, W.G., 2014. The role of the cell wall in plant immunity. Front. Plant Sci. 5, 178.
McQueen-Mason, S., Durachko, D.M., Cosgrove, D.J., 1992. Two endogenous proteins that induce cell wall extension in plants. Plant Cell 4, 1425–1433.
Monaghan, J., Zipfel, C., 2012. Plant pattern recognition receptor complexes at the plasma membrane. Curr. Opin. Plant Biol. 15, 349–357.
Mur, L.A., Mandon, J., Persijn, S., Cristescu, S.M., Moshkov, I.E., Novikova, G.V., Hall, M.A., Harren, F.J., Hebelstrup, K.H., Gupta, K.J., 2013. Nitric oxide in plants: an assessment of the current state of knowledge. AoB Plants 5, pls052.
Mussig, C., Fischer, S., Altmann, T., 2002. Brassinosteroid-regulated gene expression. Plant Physiol. 129, 1241–1251.
Nafisi, M., Stranne, M., Zhang, L., van Kan, J., Sakuragi, Y., 2014. The endo-arabinanase BcAra1 is a novel host-specific virulence factor of the necrotic fungal phytopathogen Botrytis cinerea. Mol. Plant Microbe Interact..
Nakashita, H., Yasuda, M., Nitta, T., Asami, T., Fujioka, S., Arai, Y., Sekimata, K., Takatsuto, S., Yamaguchi, I., Yoshida, S., 2003. Brassinosteroid functions in a broad range of disease resistance in tobacco and rice. Plant J. 33, 887–898.
Ng, L.M., Melcher, K., Teh, B.T., Xu, H.E., 2014. Abscisic acid perception and signaling: structural mechanisms and applications. Acta Pharmacol. Sin. 35, 567–584.
Nguema-Ona, E., Moore, J.P., Fagerstrom, A.D., Fangel, J.U., Willats, W.G., Hugo, A., Vivier, M.A., 2013. Overexpression of the grapevine PGIP1 in tobacco results in compositional changes in the leaf arabinoxyloglucan network in the absence of fungal infection. BMC Plant Biol. 13, 46.
O'Brien, J.A., Daudi, A., Finch, P., Butt, V.S., Whitelegge, J.P., Souda, P., Ausubel, F.M., Bolwell, G.P., 2012. A peroxidase-dependent apoplastic oxidative burst in cultured Arabidopsis cells functions in MAMP-elicited defense. Plant Physiol. 158, 2013–2027.
Oide, S., Bejai, S., Staal, J., Guan, N., Kaliff, M., Dixelius, C., 2013. A novel role of PR2 in abscisic acid (ABA) mediated, pathogen-induced callose deposition in *Arabidopsis thaliana*. New Phytol. 200, 1187–1199.
Orozco-Cardenas, M., Ryan, C.A., 1999. Hydrogen peroxide is generated systemically in plant leaves by wounding and systemin via the octadecanoid pathway. Proc. Natl. Acad. Sci. U.S.A. 96, 6553–6557.
Pacoda, D., Montefusco, A., Piro, G., Dalessandro, G., 2004. Reactive oxygen species and nitric oxide affect cell wall metabolism in tobacco BY-2 cells. J. Plant Physiol. 161, 1143–1156.
Pagant, S., Bichet, A., Sugimoto, K., Lerouxel, O., Desprez, T., McCann, M., Lerouge, P., Vernhettes, S., Hofte, H., 2002. KOBITO1 encodes a novel plasma membrane protein necessary for normal synthesis of cellulose during cell expansion in Arabidopsis. Plant Cell 14, 2001–2013.
Pan, X.Q., Welti, R., Wang, X.M., 2008. Simultaneous quantification of major phytohormones and related compounds in crude plant extracts by liquid chromatography–electrospray tandem mass spectrometry. Phytochemistry 69, 1773–1781.
Paque, S., Mouille, G., Grandont, L., Alabadi, D., Gaertner, C., Goyallon, A., Muller, P., Primard-Brisset, C., Sormani, R., Blazquez, M.A., Perrot-Rechenmann, C., 2014. AUXIN BINDING PROTEIN1 links cell wall remodeling, auxin signaling, and cell expansion in Arabidopsis. Plant Cell 26, 280–295.
Passardi, F., Penel, C., Dunand, C., 2004. Performing the paradoxical: how plant peroxidases modify the cell wall. Trends Plant Sci. 9, 534–540.
Perez, A.C., Goossens, A., 2013. Jasmonate signalling: a copycat of auxin signalling? Plant, Cell Environ. 36, 2071–2084.
Pieterse, C.M.J., Van der Does, D., Zamioudis, C., Leon-Reyes, A., Van Wees, S.C.M., 2012. Hormonal modulation of plant immunity. Annu. Rev. Cell Dev. Biol. 28, 489–521.
Powell, A.L., van Kan, J., ten Have, A., Visser, J., Greve, L.C., Bennett, A.B., Labavitch, J.M., 2000. Transgenic expression of pear PGIP in tomato limits fungal colonization. Mol. Plant Microbe Interact. 13, 942–950.
Raiola, A., Camardella, L., Giovane, A., Mattei, B., De Lorenzo, G., Cervone, F., Bellincampi, D., 2004. Two *Arabidopsis thaliana* genes encode functional pectin methylesterase inhibitors. FEBS Lett. 557, 199–203.
Raiola, A., Lionetti, V., Elmaghraby, I., Immerzeel, P., Mellerowicz, E.J., Salvi, G., Cervone, F., Bellincampi, D., 2011. Pectin methylesterase is induced in

Arabidopsis upon infection and is necessary for a successful colonization by necrotrophic pathogens. Mol. Plant Microbe Interact. 24, 432–440.
Ranocha, P., Denance, N., Vanholme, R., Freydier, A., Martinez, Y., Hoffmann, L., Kohler, L., Pouzet, C., Renou, J.P., Sundberg, B., Boerjan, W., Goffner, D., 2010. Walls are thin 1 (WAT1), an Arabidopsis homolog of *Medicago truncatula* NODULIN21, is a tonoplast-localized protein required for secondary wall formation in fibers. Plant J..
Ranocha, P., Dima, O., Nagy, R., Felten, J., Corratge-Faillie, C., Novak, O., Morreel, K., Lacombe, B., Martinez, Y., Pfrunder, S., Jin, X., Renou, J.P., Thibaud, J.B., Ljung, K., Fischer, U., Martinoia, E., Boerjan, W., Goffner, D., 2013. Arabidopsis WAT1 is a vacuolar auxin transport facilitator required for auxin homoeostasis. Nat. Commun. 4.
Rasul, S., Dubreuil-Maurizi, C., Lamotte, O., Koen, E., Poinssot, B., Alcaraz, G., Wendehenne, D., Jeandroz, S., 2012. Nitric oxide production mediates oligogalacturonide-triggered immunity and resistance to *Botrytis cinerea* in *Arabidopsis thaliana*. Plant Cell Environ. 35, 1483–1499.
Ridley, B.L., O'Neill, M.A., Mohnen, D., 2001. Pectins: structure, biosynthesis, and oligogalacturonide-related signaling. Phytochemistry 57, 929–967.
Rubio, S., Rodrigues, A., Saez, A., Dizon, M.B., Galle, A., Kim, T.H., Santiago, J., Flexas, J., Schroeder, J.I., Rodriguez, P.L., 2009. Triple loss of function of protein phosphatases type 2C leads to partial constitutive response to endogenous abscisic acid. Plant Physiol. 150, 1345–1355.
Ryu, H., Hwang, I., 2013. Brassinosteroids in plant developmental signaling networks. J. Plant Biol. 56, 267–273.
Sanchez-Vallet, A., Lopez, G., Ramos, B., Delgado-Cerezo, M., Riviere, M.P., Llorente, F., Fernandez, P.V., Miedes, E., Estevez, J.M., Grant, M., Molina, A., 2012. Disruption of abscisic acid signaling constitutively activates Arabidopsis resistance to the necrotrophic fungus *Plectosphaerella cucumerina*. Plant Physiol. 160, 2109–2124.
Savatin, D.V., Ferrari, S., Sicilia, F., De Lorenzo, G., 2011. Oligogalacturonide-auxin antagonism does not require posttranscriptional gene silencing or stabilization of auxin response repressors in Arabidopsis. Plant Physiol. 157, 1163–1174.
Sharon, A., Elad, Y., Barakat, R., Tudzynski, P., 2007. Phytohormones in Botrytis–Plant Interactions. Springer.
Siemens, J., Keller, I., Sarx, J., Kunz, S., Schuller, A., Nagel, W., Schmulling, T., Parniske, M., Ludwig-Muller, J., 2006. Transcriptome analysis of Arabidopsis clubroots indicate a key role for cytokinins in disease development. Mol. Plant Microbe Interact. 19, 480–494.
Somerville, C., Bauer, S., Brininstool, G., Facette, M., Hamann, T., Milne, J., Osborne, E., Paredez, A., Persson, S., Raab, T., Vorwerk, S., Youngs, H., 2004. Toward a systems approach to understanding plant cell walls. Science 306, 2206–2211.
Sun, Y., Veerabomma, S., Abdel-Mageed, H.A., Fokar, M., Asami, T., Yoshida, S., Allen, R.D., 2005. Brassinosteroid regulates fiber development on cultured cotton ovules. Plant Cell Physiol. 46, 1384–1391.
Swartzberg, D., Kirshner, B., Rav-David, D., Elad, Y., Granot, D., 2008. Botrytis cinerea induces senescence and is inhibited by autoregulated expression of the IPT gene. Eur. J. Plant Pathol. 120, 289–297.
Talboys, P.J., Zhang, H.M., Knox, J.P., 2011. ABA signalling modulates the detection of the LM6 arabinan cell wall epitope at the surface of *Arabidopsis thaliana* seedling root apices. New Phytol. 190, 618–626.
Thomas, J., Ross, C.W., Chastain, C.J., Koomanoff, N., Hendrix, J.E., Vanvolkenburgh, E., 1981. Cytokinin-induced wall extensibility in excised cotyledons of radish and cucumber. Plant Physiol. 68, 107–110.
Ton, J., Mauch-Mani, B., 2004. Beta-amino-butyric acid-induced resistance against necrotrophic pathogens is based on ABA-dependent priming for callose. Plant J. 38, 119–130.
Turner, S.R., Somerville, C.R., 1997. Collapsed xylem phenotype of Arabidopsis identifies mutants deficient in cellulose deposition in the secondary cell wall. Plant Cell 9, 689–701.
Ulvskov, P., Wium, H., Bruce, D., Jorgensen, B., Qvist, K.B., Skjot, M., Hepworth, D., Borkhardt, B., Sorensen, S.O., 2005. Biophysical consequences of remodeling the neutral side chains of rhamnogalacturonan I in tubers of transgenic potatoes. Planta 220, 609–620.
Vance, C.P., Kirk, T.K., Sherwood, R.T., 1980. Lignification as a mechanism of disease resistance. Annu. Rev. Phytopathol. 18, 259–288.
Verhertbruggen, Y., Marcus, S.E., Chen, J.S., Knox, J.P., 2013. Cell wall pectic arabinans influence the mechanical properties of *Arabidopsis thaliana* inflorescence stems and their response to mechanical stress. Plant Cell Physiol. 54, 1278–1288.
Voigt, C.A., 2014. Callose-mediated resistance to pathogenic intruders in plant defense-related papillae. Front. Plant Sci. 5, 168.
Vorwerk, S., Somerville, S., Somerville, C., 2004. The role of plant cell wall polysaccharide composition in disease resistance. Trends Plant Sci. 9, 203–209.
Wang, D., Pajerowska-Mukhtar, K., Culler, A.H., Dong, X.N., 2007. Salicylic acid inhibits pathogen growth in plants through repression of the auxin signaling pathway. Curr. Biol. 17, 1784–1790.
Wang, H., Nagegowda, D.A., Rawat, R., Bouvier-Nave, P., Guo, D.J., Bach, T.J., Chye, M.L., 2012. Overexpression of *Brassica juncea* wild-type and mutant HMG-CoA synthase 1 in Arabidopsis up-regulates genes in sterol biosynthesis and enhances sterol production and stress tolerance. Plant Biotechnol. J. 10, 31–42.
Wang, X., Kota, U., He, K., Blackburn, K., Li, J., Goshe, M.B., Huber, S.C., Clouse, S.D., 2008. Sequential transphosphorylation of the BRI1/BAK1 receptor kinase complex impacts early events in brassinosteroid signaling. Dev. Cell 15, 220–235.
Williamson, B., Tudzynski, B., Tudzynski, P., Van Kan, J.A.L., 2007. Botrytis cinerea: the cause of grey mould disease. Mol. Plant Pathol. 8, 561–580.
Windram, O., Madhou, P., McHattie, S., Hill, C., Hickman, R., Cooke, E., Jenkins, D.J., Penfold, C.A., Baxter, L., Breeze, E., Kiddle, S.J., Rhodes, J., Atwell, S., Kliebenstein, D.J., Kim, Y.S., Stegle, O., Borgwardt, K., Zhang, C.J., Tabrett, A., Legaie, R., Moore, J., Finkenstadt, B., Wild, D.L., Mead, A., Rand, D., Beynon, J., Ott, S., Buchanan-Wollaston, V., Denby, K.J., 2012. Arabidopsis defense against *Botrytis cinerea:* chronology and regulation deciphered by high-resolution temporal transcriptomic analysis. Plant Cell 24, 3530–3557.
Wolf, S., Mravec, J., Greiner, S., Mouille, G., Hofte, H., 2012. Plant cell wall homeostasis is mediated by brassinosteroid feedback signaling. Curr. Biol. 22, 1732–1737.
Woodward, A.W., Bartel, B., 2005. Auxin: regulation, action, and interaction. Ann. Bot. 95, 707–735.
Xie, L.Q., Yang, C.J., Wang, X.L., 2011. Brassinosteroids can regulate cellulose biosynthesis by controlling the expression of CESA genes in Arabidopsis. J. Exp. Bot. 62, 4495–4506.
Xiong, J., An, L., Lu, H., Zhu, C., 2009. Exogenous nitric oxide enhances cadmium tolerance of rice by increasing pectin and hemicellulose contents in root cell wall. Planta 230, 755–765.
Yin, Y.H., Wang, Z.Y., Mora-Garcia, S., Li, J.M., Yoshida, S., Asami, T., Chory, J., 2002. BES1 accumulates in the nucleus in response to brassinosteroids to regulate gene expression and promote stem elongation. Cell 109, 181–191.
Yoda, S., 1958. Auxin action and pectin enzyme. Bot. Mag. Tokyo 71, 207–213.
Zhang, Z.Y., Wang, H.H., Wang, X.M., Bi, Y.R., 2011. Nitric oxide enhances aluminum tolerance by affecting cell wall polysaccharides in rice roots. Plant Cell Rep. 30, 1701–1711.
Zhao, M.R., Han, Y.Y., Feng, Y.N., Li, F., Wang, W., 2012. Expansins are involved in cell growth mediated by abscisic acid and indole-3-acetic acid under drought stress in wheat. Plant Cell Rep. 31, 671–685.
Zhao, Q., Dixon, R.A., 2014. Altering the cell wall and its impact on plant disease: from forage to bioenergy. Annu. Rev. Phytopathol..
Zhao, Y., 2010. Auxin biosynthesis and its role in plant development. Annu. Rev. Plant Biol. 61, 49–64.