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Review

Roles of plant hormones in the regulation of host–virus interactions

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SUMMARY

Hormones are tuners of plant responses to biotic and abiotic stresses. They are involved in various complicated networks, through which they modulate responses to different stimuli. Four hormones primarily regulate plant defence to pathogens: salicylic acid (SA), jasmonic acid (JA), ethylene (Et) and abscisic acid (ABA). In susceptible plants, viral infections result in hormonal disruption, which manifests as the simultaneous induction of several antagonistic hormones. However, these antagonistic hormones may exhibit some sequential accumulation in resistant lines. Virus propagation is usually restricted by the activation of the small interfering RNA (siRNA) antiviral machinery and/or SA signalling pathway. Several studies have investigated these two systems, using different model viruses. However, the roles of hormones other than SA, especially those with antagonistic properties, such as ABA, have been neglected. Increasing evidence indicates that hormones control components of the small RNA system, which regulates many processes (including the siRNA antiviral machinery and the microRNA system) at the transcriptional or post-transcriptional level. Consequently, cross-talk between the antagonistic SA and ABA pathways modulates plant responses at multiple levels. In this review, we summarize recent findings on the different roles of hormones in the regulation of plant–virus interactions, which are helping us to elucidate the fine tuning of viral and plant systems by hormones.

Keywords: defence pathways, host–virus interaction, plant hormones, plant virus.

INTRODUCTION

Being immobilized organisms, plants are particularly vulnerable to climatic and environmental changes, and thus have had to develop cost-effective adaptive mechanisms. On detection of stress, plants stimulate a response in distal parts through the release of small chemical molecules, called hormones. Hormones are signal molecules that rove all around the plant to stimulate responses to different environmental stresses. Several hormones have long been known for their roles in tuning plant responses to biotic stresses, such as salicylic acid (SA), jasmonic acid (JA) and ethylene (Et). Others, which are mostly known for their roles in plant growth and development, have recently been found to play a role in plant–pathogen interactions, such as auxins (Auxs), brassinosteroids (BRs), cytokinins (CKs) and abscisic acid (ABA) (Denance *et al*., 2013; Pieterse *et al*., 2009; Santner *et al*., 2009).

Hormones have antagonistic or synergistic inter-relations, through which certain hormones can prevail over others under specific circumstances. SA, JA and Et, which regulate defence pathways, exhibit antagonistic interactions. For example, the induction of the SA signalling pathway can repress the JA/Et pathway through *NPR1* (*NONEXPRESSER OF PATHOGENESIS-RELATED GENE 1*) and *WRKY70*, and the ABA pathway through *NPR1* or its downstream elements (Bari and Jones, 2009; Koornneef and Pieterse, 2008; Spoel *et al*., 2003; Yasuda *et al*., 2008). Conversely, induction of the JA/Et pathway represses the expression of certain genes downstream of SA signalling via *MAPK4* (*MITOGEN-ACTIVATED PROTEIN KINASE 4*) and *JIN2* (Kachroo and Kachroo, 2007; Koornneef and Pieterse, 2008). Several abiotic stress responses, such as responses to drought or cold, are mediated primarily by ABA, which strongly antagonizes many hormone pathways, including the SA pathway (Soosaar *et al*., 2005; Yasuda *et al*., 2008), the Et pathway (Cheng *et al*., 2009; Ghassemian *et al*., 2000) and the synergized dual Et/JA pathway (Broekaert *et al*., 2006). However, ABA seems to positively regulate JA biosynthesis and signalling during necrotrophic infection (Adie *et al*., 2007; Fan *et al*., 2009) or stomatal closure (Hossain *et al*., 2011; Munemasa *et al*., 2007). Finally, ABA, CKs and Et have antagonistic effects on gibberellic acid (GA) during several developmental processes, whereas Aux interacts positively with GA (Greenboim-Wainberg *et al*., 2005; Jasinski *et al*., 2005; Weiss and Ori, 2007).

ROLES OF HORMONES IN PLANT–VIRUS INTERACTIONS

Salicylic acid

SA is a phenolic compound synthesized by plants in response to a wide range of pathogens, and is essential for the establishment of

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local and systemic resistance (Loake and Grant, 2007; Vlot *et al*., 2009). The importance of SA arises from its role in the mediation of resistance (*R*)-gene resistance and basal immune responses, and from the positive link between SA-mediated defence and the small interfering RNA (siRNA) antiviral machinery (Alamillo *et al*., 2006; Baebler *et al*., 2014; Hunter *et al*., 2013).

SA biosynthesis and signalling are activated on recognition of viral effectors by *R* gene products, which conditions incompatible interaction. Activation of the incompatible interaction results in several responses to limit viral propagation at the infection site, including the accumulation of reactive oxygen species (ROS) and pathogenesis-related (PR) proteins, induction of the hypersensitive response (HR), callose deposition, tissue disorganization, changes in the size and shape of chloroplasts, nuclear and nucleolar degradation, and programmed cell death (PCD) (Baebler *et al*., 2014; Dinesh-Kumar *et al*., 2000). SA is also responsible for the activation of systemic acquired resistance (SAR) in distal tissues, which lessens the effects of secondary attacks.

The literature contains well-studied examples of incompatible plant–virus interactions. For example, infection with *Tobacco mosaic virus* (TMV) results in a significant increase in SA in the inoculated and systemic leaves of resistant tobacco plants. In parallel, the expression of *PR* genes is strongly increased at both sites (reviewed in Vlot *et al*., 2009). Similar observations were made in *Ny-1*-resistant potatoes after infection with *Potato virus Y* (PVY) (Baebler *et al*., 2014). In the context of the incompatible interaction between resistant tobacco and *Tomato ringspot virus* (ToRSV), or between resistant potato and PVY, introduction of the *NahG* gene results in the degradation of SA into catechol, the negation of plant defences and a resulting increase in lesion size, thereby allowing the virus to accumulate substantially and move systemically (Baebler *et al*., 2014; Jovel *et al*., 2011). Similarly, the *eds5* (*enhanced disease susceptibility 5*) mutation and the *NahG* transgene partially negated the resistance of *Col-24-C* to *Cucumber mosaic virus* strain-Y (CMV-Y) (Takahashi *et al*., 2004). In both *Ny-1*-resistant and *NahG*-transgenic-*Ny-1* potato plants, genes encoding PRs or components involved in the production of ROS are induced at early stages of infection [1–3 days post-infection (dpi)]. However, genes encoding enzymes involved in cell wall rearrangement and the synthesis of secondary metabolites were up-regulated only in the resistant line (Baebler *et al*., 2014).

Mutations in the SA pathway impair plant defence, thereby rendering plants susceptible to viral infection, even in the presence of relevant *R* genes (Baebler *et al*., 2014; Dinesh-Kumar *et al*., 2000; Lewsey *et al*., 2008; Takahashi *et al*., 2004). The absence of specific *R* genes also makes plants vulnerable to infection; such an interaction is defined as compatible, and is characterized by weak defence responses to infection. The overexpression of SA biosynthesis genes or application of SA or its analogues often improves plant basal immunity by delaying the onset of viral infection and disease establishment (Ishihara *et al*., 2008; Mayers *et al*., 2005; Peng *et al*., 2013).

SA also controls extreme resistance (ER), characterized by the absence of necrotic lesions in plants with *R* genes. This resistance results in almost complete elimination of the virus without visual symptoms. ER, which conceptually resembles effector-triggered immunity (ETI), can be observed in the resistance of tobacco plants to *Tomato bushy stunt virus* (TBSV) (Sansregret *et al*., 2013), the *Tm-2*^{2}-mediated resistance to TMV or *Tomato mosaic virus* (ToMV) (Zhang *et al*., 2013) and the soybean *Rsv1*-mediated resistance to *Soybean mosaic virus* (SMV) (Zhang *et al*., 2012). Sansregret *et al*. (2013) found that TBSV resistance in tobacco requires an intact SA pathway. Although TBSV does not accumulate in wild-type (WT) *Nicotiana tabacum* Xanthi, *NahG* lines of the same background are susceptible to infection. In tobacco, ER is triggered by P19, the TBSV viral suppressor of RNA silencing (VSR); however, constitutive expression of P19 induces HR-like necrosis. It has been rationalized that resistant tobacco plants can sense small amounts of P19, and subsequently trigger ER. When this response is disrupted by impairment of the VSR functionality of P19, TBSV may accumulate to levels sufficient to trigger HR (Sansregret *et al*., 2013). Interestingly, the VSR function of P19 was found to be necessary, but insufficient, for ER, based on findings that the constitutive expression of mutant versions of P19 that lack VSR activity failed to induce HR or PR genes, and that competition with other VSRs for siRNA reduces the P19-mediated HR. This indicates that plants can perceive P19–siRNA complex formation, enabling ER initiation via downstream cascades (Sansregret *et al*., 2013).

Arabidopsis exhibits a compatible interaction with CMV-Y or *Oilseed rape mosaic virus* (ORMV); both viruses accumulate similarly in the SA mutants *npr1*, *sid2* (*salicylic acid induction deficient2*), *eds5* and *pad4* (*phytoalexin deficient 4*) when compared with the WT *Col-0* at 5 dpi. It was concluded that SA is less likely to be induced by CMV or ORMV infection (Huang *et al*., 2005). However, a later study compared the levels of coat proteins (CPs) of CMV and *Turnip crinkle virus* (TCV) in *eds5* and *NahG* mutants at different time points; although CP levels in these mutants were almost identical up to 5 dpi, similar to the findings of Huang *et al*. (2005), substantial differences were observed between mutants at 10 dpi. CPs increased to high levels, before gradually decreasing after 15 dpi to levels similar to those of the WT for both viruses (Wang *et al*., 2011a). Similarly, *Bamboo mosaic virus* (BaMV) RNA levels were greater in the SA mutants *eds1* (Alazem *et al*., 2014) and *sid2-1* (M. Alazem and N-S. Lin, unpublished data) at 10 dpi. The ability of mutants to discern differences in viral levels leads us to suggest that the viral incubation period in Arabidopsis should be examined using time-course methodology. In contrast, *NahG* potato plants exhibit faster onset of PVY^{NTN} viral infection and develop stronger PVY symptoms, when compared with non-transgenic lines. However, this difference was observed only in inoculated leaves at early stages of infection, and diminished with

infection progression. In systemic leaves, no difference was observed between *NahG* and WT potato plants, in terms of viral titre or expression of defence-related genes (Baebler *et al*., 2011).

The exogenous application of SA to susceptible plants improves their resistance to different viruses. For example, SA treatment has been reported to reduce the CP levels of TMV and *Potato virus X* (PVX) during their compatible interactions with *N. benthamiana* plants (Lee *et al*., 2011). Furthermore, spraying *NahG*-transgenic potato with 2,6-dichloroisonicotinic acid (INA), an SA analogue, restored the asymptomatic phenotype of PVY^{NTN} infection (Baebler *et al*., 2011). *CPR1* (*CONSTITUTIVE EXPRESSER OF PR GENES 1*) encodes an F-box protein which negatively regulates SA in Arabidopsis (Bowling *et al*., 1994; Gou *et al*., 2009). The *cpr-1* and *cpr-5* mutations rendered plants resistant to certain DNA viruses, including *Cabbage leaf curl virus* (CaLCuV), as a result of the constitutive elevation of SA and its related genes (Ascencio-Ibanez *et al*., 2008). The effects of SA on plant defence seem to be diverse, and depend on both the host and infecting virus. In *N. tabacum* and *Arabidopsis thaliana*, SA-induced resistance to CMV infection inhibits viral systemic movement. However, SA-mediated resistance in *Cucurbita pepo* results from decreased viral accumulation in inoculated tissues, implying that SA affects cell-to-cell, rather than systemic, movement. Therefore, different hosts may use alternative approaches to resist the same virus (Mayers *et al*., 2005). In addition, SA-mediated resistance to several viruses (such as PVX and TMV) is affected by the capacity of the alternative respiratory pathway. Notably, resistance to both PVX and TMV is increased when the capacity of the alternative respiratory pathway is reduced, but is decreased when the capacity is enhanced (Lee *et al*., 2011). However, SA treatment does not always improve resistance. For instance, exogenous SA treatment did not affect levels of *Bean pod mottle virus* (BPMV) in inoculated or systemic leaves of soybean at 3 or 7 dpi, respectively (Singh *et al*., 2011). One study (to date) has reported that SA actually increases the susceptibility of pea cultivar to *Clover yellow vein virus* (ClYVV). The *CYN1* resistance gene controls systemic cell death on ClYVV infection. Although treatment of resistant pea plants with benzothiadiazole, an analogue of SA and inducer of SAR, augmented resistance, benzothiadiazole treatment of *cyn1*-susceptible peas enhanced ClYVV symptoms. Although the response was different at the symptomatic level, there was no significant difference between the two cultivars in terms of viral titre. It remains unclear how SA enhanced the virulence in susceptible plants (Atsumi *et al*., 2009).

SA repression of viral replication has been suggested to be partially mediated through the siRNA pathway, and evidence for positive cross-talk between SA and siRNA antiviral defences is accumulating (Alamillo *et al*., 2006; Campos *et al*., 2014; Hunter *et al*., 2013; Jovel *et al*., 2011; Yu *et al*., 2003). For example, accumulation of *Plum Pox virus* (PPV)-derived small RNAs was reduced in *NahG* transgenic plants, and transgenic lines over-expressing the P1/helper component-proteinase (HC-Pro) suppressor exhibited reduced SA-mediated defence and PPV-derived siRNA levels (Alamillo *et al*., 2006). In addition, P1/HC-Pro tobacco plants carrying *NahG* accumulated ToRSV-derived small RNAs only in lesions that accumulated viral RNA, but not in systemic tissues (Jovel *et al*., 2011). This evidence strongly suggests that SA enhances RNA-silencing antiviral defence in tobacco plants (Alamillo *et al*., 2006). Of note, SA treatment increased *RDR1* (*RNA-DEPENDENT RNA POLYMERASE 1*) levels in both *N. tabacum* and *A. thaliana* (Alamillo *et al*., 2006; Hunter *et al*., 2013; Jovel *et al*., 2011; Ying *et al*., 2010; Yu *et al*., 2003). However, genes encoding dicer-like proteins (*DCL*s; proteins involved in small RNA production) seem to be independent of SA-induced resistance in Arabidopsis, as SA treatment is able to reduce CMV and TMV titres in *dcl2*, *dcl3* and *dcl4* mutants (Lewsey and Carr, 2009). The authors suggested that SA may trigger several redundant mechanisms, some of which are independent of *DCL*s (Lewsey and Carr, 2009). Interestingly, *DCL1*, *DCL2*, *RDR1* and *RDR2* were all found to be induced by SA and ToMV infection in tomato plants (Campos *et al*., 2014). This probably gives SA more means to positively regulate the siRNA system in such a host. SA has also been reported to act against VSRs; for example, levels of CMVΔ2b virus (lacking the CMV2b suppressor) were higher in *NahG* transgenic than in WT plants, but lower than the level of CMV in WT plants. This implies that reduced SA may partially compensate for the 2b defect (Ji and Ding, 2001). SA may act upstream of the siRNA pathway, thereby amplifying the siRNA response (Fig. 1). On the basis of this hypothesis, defective SA biosynthesis would weaken siRNA biogenesis in a similar manner to VSR. The current evidence suggests strong links between SA biosynthesis and siRNA pathways, but it is unclear whether downstream components of SA are also involved in the stimulation of the siRNA system. On the basis of the reported relationships between these processes, it is not surprising to observe interference of the SA pathway by viral VSRs. For example, 2b VSR of CMV and Tobamovirus replicase affect the regulation of SA-responsive genes, and the deletion of 2b enhances the sensitivity of CMVΔ2b to SA, thereby reducing symptoms in *N. glutinosa* (Ji and Ding, 2001; Lewsey *et al*., 2010; Pruss *et al*., 2004; Shams-Bakhsh *et al*., 2007). Similarly, P6 VSR of *Cauliflower mosaic virus* (CaMV) suppresses SA signalling responses and modulates JA responses by interacting with NPR1 in the cytosol, the intersection between the SA and JA pathways (Laird *et al*., 2013; Love *et al*., 2012). Moreover, the 1a subdomain of P6 represses SA responses; deletion of this subdomain restores the ability of P6 to down-regulate PR-1a levels (Laird *et al*., 2013).

Abscisic acid

ABA, a sesquiterpene compound resulting from the cleavage of γ-carotene, regulates numerous developmental processes and adaptive stress responses in plants. It strongly regulates several

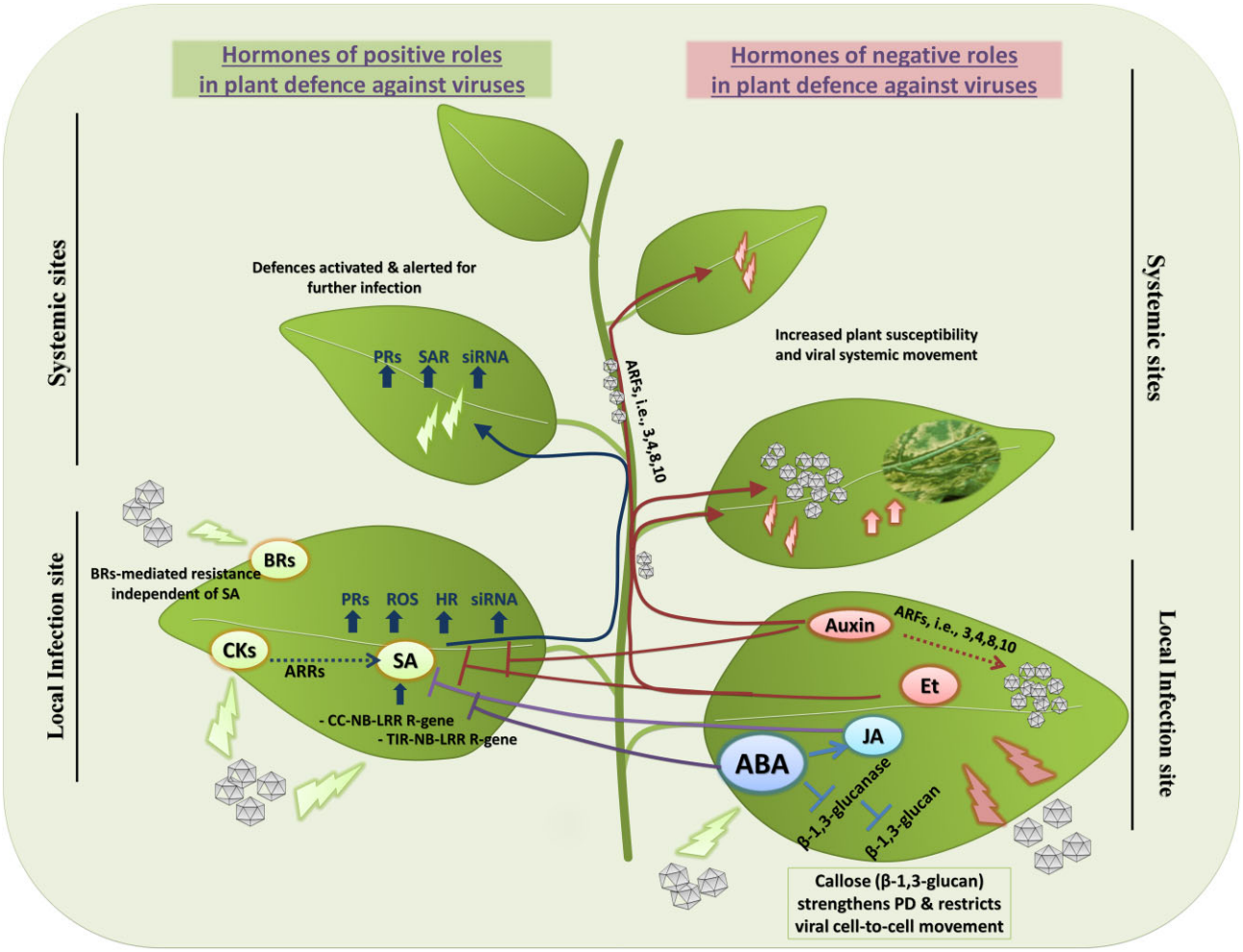

Fig. 1 Hormone–virus inter-relations: general effects of hormones on plant defence against viruses. Hormones in light green circles have positive effects on defence against viruses. Salicylic acid (SA) signalling, which is tightly connected to the majority of *NB-LRR* (*NUCLEOTIDE-BINDING-LEUCINE-RICH REPEAT*) genes, constitutes the major defensive pathway against viruses. The recognition of viral effectors by R proteins initiates defensive pathways, including the activation of SA and small interfering RNA (siRNA) pathways, induction of reactive oxygen species (ROS) and the hypersensitive response (HR) (Baebler *et al*., 2014). These responses limit viral spread in necrotic lesions, and activate siRNA antiviral mechanisms. SA activates systemic acquired resistance (SAR) and siRNA machinery at distal sites (Alamillo *et al*., 2006; Loake & Grant, 2007; Vlot *et al*., 2009). Cytokeratins (CKs) improve plant defences to biotrophs in an SA-mediated manner. CK-responsive factors, such as *ARR3* (ARABIDOPSIS RESPONSE REGULATOR 3), *ARR4*, *ARR5*, *ARR6*, *ARR8* and *ARR9*, are involved in the cross-talk between SA and CKs (Argueso *et al*., 2012). Brassinosteroids (BRs) act independently of SA in enhancing resistance to biotrophs. Hormones in red circles have primarily negative effects on plant defences to viruses. Auxin is known to antagonize the SA pathway, and a subset of Auxin response factors (*ARF*s) is important for the replication and movement of certain viruses, such as *Tobacco mosaic virus* (TMV) (Padmanabhan *et al*., 2008, 2005). Ethylene (Et) also antagonizes the pathway downstream of SA signalling, and is involved in symptom development on *Cauliflower mosaic virus* (CaMV) infection, systemic movement of TMVcg and the formation of necrotic lesions following infection by other viruses (Chen *et al*., 2013, Geri *et al*., 2004). Jasmonis acid (JA) and abscisic acid (ABA) have both positive and negative effects on defence against viruses; JA seems to support plant defence at early stages of infection, but, if it is induced or applied at later stages, it decreases plant resistance (Garcia-Marcos *et al*., 2013, Pacheco *et al*., 2012). ABA has multifaceted roles in plant defence; on the one hand, it increases callose deposition on plasmodesmata (PD) and restricts cell-to-cell movement of viruses, such as TMV and *Tobacco necrosis virus* (TNV), whereas, on the other, it antagonizes the SA pathway and reduces resistance at local sites of infection by repressing HR induction, decreasing the production of ROS and SA, and weakening distal SAR and siRNA systems (Alazem *et al*., 2014, Iriti & Faoro, 2008, Whenham *et al*., 1986).

developmental stages, including seed germination and fruit ripening, and is considered as the key hormone in the modulation of plant responses to many abiotic stresses (Atkinson and Urwin, 2012; Rajjou *et al*., 2012; Sung and Luan, 2012). In addition to its antagonistic roles in defence hormone pathways, such as SA and JA/Et, ABA appears to have multifaceted roles against the same pathogen, depending on the stage of infection. ABA can positively regulate plant defence at the early stages of infection by the mediation of stomatal closure against invaders, or induction of callose deposition if the pathogen evades the first line of defence.

However, if activated at later stages, ABA can suppress ROS induction and SA or JA signalling transduction, thereby negating defences controlled by these two pathways (Asselbergh *et al*., 2008; Ton *et al*., 2009). Although the involvement of ABA in biotic stress has been studied extensively, the roles of ABA in virus replication and movement are not well characterized. ABA–virus interaction was first studied in the context of the effect of TMV on ABA accumulation in *N. tabacum* and tomato, which revealed that ABA increases callose deposition and limits virus movement (Fraser and Whenham, 1989; Whenham *et al*., 1986). In *Phaseolus vulgaris* infected with *Tobacco necrosis virus* (TNV), ABA restricts viral movement by priming callose deposition. Exogenous ABA application attenuated symptoms and reduced viral titre, and the effects of ABA were diminished when *P. vulgaris* plants were treated with nordihydroguaiaretic acid (NDGA), an ABA inhibitor (Iriti and Faoro, 2008). However, ABA was not induced in response to *White clover mosaic virus* (WClMV) infection in the same host (Clarke *et al*., 1998). The ABA response to infection varied in plants harbouring specific resistance genes. ABA levels did not differ between uninfected or infected potato cultivar resistant to PVY (Kovac *et al*., 2009), but tomato plants expressing the *Tm-1* gene (conferring resistance to TMV) contained elevated levels of ABA when compared with susceptible lines (Fraser and Whenham, 1989).

The strong antagonism with SA suggests that either could prevail under certain circumstances. The SA pathway is induced to various levels under both compatible and incompatible interactions with many viral infections. However, ABA is also induced during some viral infections. Simultaneous up-regulation of ABA and SA pathways has been reported for TMV and BaMV infections (Alazem *et al*., 2014; Fraser and Whenham, 1989). Other works have reported the induction of either pathway without masking the other (Flors *et al*., 2009; van Loon *et al*., 2006; Whenham *et al*., 1986; Yalpani *et al*., 1993). This particular phenomenon, in which these two antagonistic pathways are induced following infection of certain RNA viruses, may be a common occurrence. Indeed, both SA and ABA levels [and the related marker genes, *PR-1a* and *NCED3* (*NINE-CIS-EPOXYCAROTENOID DIOXYGENASE 3*)] are elevated in the ascorbic acid-deficient mutant *vtc1*; the mutant line was resistant to infection by two different types of pathogen (Barth *et al*., 2004). Perhaps certain viruses dysregulate the function of *VTC1* (*VACUOLAR TRANSPORTER CHAPERONE 1*) or related genes, allowing such simultaneous induction.

The defensive role of ABA against viruses is mediated through inhibition of the basic β-1,3-glucanase, which is responsible for the degradation of β-1,3-glucan (callose) (Fig. 1). The subsequent release of β-1,3-glucan (callose) is deposited on plasmodesmata and strengthens them against virus movement (Mauch-Mani and Mauch, 2005). We have reported previously that exogenous application of ABA increases plant resistance to BaMV (Alazem *et al*., 2014). Our observations were corroborated by the finding that mutants of the ABA pathway [*aao3* (*abscisic aldehyde oxidase 3*), *abi1-1* (*abscisic acid insensitive 1-1*), *abi3-1* and *abi4-1*] are susceptible to BaMV infection. The ABA biosynthesis gene *ABA2* and, to a lesser extent, *NCED3* are of particular importance for BaMV, as the mutants *nced3* and *aba2-1* exhibited reduced BaMV titres. Furthermore, the accumulation of (–)RNA was decreased dramatically in *aba2-1*. These findings imply that the product of *ABA2* is essential during the early steps of replication, possibly because it is incorporated into a regulatory protein complex required by BaMV. Similarly, CMV also failed to replicate in the *aba2-1* mutant (Alazem *et al*., 2014). Although our findings establish that ABA is active against BaMV in inoculated leaves of *A. thaliana*, such an effect was only evident in systemic leaves against TMVcg (crucifier-infecting strain of TMV) (Chen *et al*., 2013). Although the *aba1*, *aba2* and *aba3* mutations enhanced plant susceptibility and systemic movement of TMVcg, the transcript levels of their genes were not induced in the WT after infection. In addition, *aba2* did not have different effects on TMVcg relative to WT plants. This study also reported that *WRKY8* positively regulates *ABI4* by mediating transcription via binding to W-boxes in its promoter region. *ABI4* failed to accumulate to WT levels in different mutant alleles of *wrky8*, and these mutants were more susceptible to TMVcg infection (Chen *et al*., 2013).

ABA may have an important role in incompatible interactions with viruses. A recent study has proposed a role for ABA in controlling the localization of temperature-sensitive *R* genes (Mang *et al*., 2012). ABA deficiency promoted the activity and nuclear localization of temperature-sensitive *SNC1* (*SUPPRESSOR OF NPR1-1, CONSTITUTIVE 1*) and *RPS4* (*RESISTANCE TO PSEUDOMONAS SYRINGAE 4*) *R* genes, which function against *Pseudomonas syringae*. Such localization is essential for these proteins to function at low and high temperatures, whereas, in WT plants, these proteins function only at low temperatures. Thus, ABA deficiency enhanced the resistance mediated by the two *R* genes. Interestingly, the effect of ABA deficiency on the nuclear localization of both proteins was not mediated by SA (Mang *et al*., 2012). A few antiviral *R* genes, such as the tobacco *N* gene, are also temperature sensitive. Nevertheless, the role of ABA in R-mediated defence against viruses has not been studied. It is possible that ABA deficiency may negate temperature sensitivity and enhance antiviral *R*-gene performance against viral infection, but this awaits further investigation.

ABA also affects plant defences at the level of the RNA silencing machinery, which is considered to be a broader defence system against viruses when compared with *R*-gene-specific resistance. RNA silencing affects both the local accumulation and systemic movement of a wide range of viruses, and is considered to be the cause of non-host resistance for some viruses, such as PVX (Jaubert *et al*., 2011; Lewsey *et al*., 2008; Ruiz-Ferrer and Voinnet, 2009). ABA seems to have direct and indirect links with this system. For example, ABA partially controls *ARGONAUTE1* (*AGO1*)

levels, which are significantly increased in *aba1-5* (Li *et al*., 2012). Furthermore, *miR168a*, which regulates AGO1 protein and is specifically induced in infected tissues, contains ABA-responsive elements in its promoter, and its levels are positively correlated with ABA levels (Laubinger *et al*., 2010; Li *et al*., 2012; Liu *et al*., 2008). In addition, mutants of the microRNA (miRNA) and siRNA pathways (such as *dcl1-11*, HUA ENHANCER 1 (*hen1*), *dcl2*, *dcl3* and *dcl4*) are hypersensitive to ABA during germination (Zhang *et al*., 2008). Moreover, ABA hypersensitivity was observed in certain mutants of RNA processing, including mutants of mRNA cap binding protein 80 (*CBP80*) and *CBP20*, which are involved in miRNA processing. ABA has been reported to stabilize CBP80 and CBP20 proteins through a post-translational mechanism (Kim *et al*., 2008; Kuhn *et al*., 2008; Papp *et al*., 2004; Pieczynski *et al*., 2013). In summary, ABA appears to influence viral defence at several levels, including mRNA processing, siRNA and miRNA biogenesis, and hormone-regulated defence pathways, such as SA (Fig. 2). Although earlier work partially elucidated the roles of ABA after virus infection, its roles in RNA silencing require further investigation. Modulation of disease resistance by ABA is a complex process, and earlier reports of diverse regulatory effects of ABA on defence responses are insufficient to provide us with clear-cut models of how ABA affects disease resistance.

Jasmonic acid

JA is an oxygenated fatty acid (oxylipin) involved in resistance to necrotrophic pathogens and insect infestation (Thaler *et al*., 2004). Together with Et, JA regulates induced systemic resistance (ISR), which is invoked by non-pathogenic microbes, such as rhizobacteria. A study has shown that rhizobacterium-mediated induction of JA reduces the symptoms of CMV infection in *Col-0* (Ryu *et al*., 2004). Several later studies supported the positive roles of JA in compatible interactions, but in a phase-specific mode. For example, co-infection with PVY and PVX, or infection with PVY carrying HC-Pro from a potyvirus (PPV), induced oxylipin biosynthesis genes at early stages of infection, and PCD (Garcia-Marcos *et al*., 2013; Pacheco *et al*., 2012). Both studies showed that knocking down *COI1* (*CORONATINE-INSENSITIVE 1*), a gene involved in the JA signalling pathway, accelerated the development of symptoms and accumulation of viral titres at early stages of infection. However, both WT and knock-down lines showed similar symptoms as infection progressed (Garcia-Marcos *et al*., 2013; Pacheco *et al*., 2012). JA treatment at early stages of PVY–PVX double infection enhanced resistance, but later application increased susceptibility, probably as a result of the antagonistic effect of JA on SA (Garcia-Marcos *et al*., 2013). Similar studies have shown that JA-responsive genes are modulated at early stages of infection, e.g. in CaMV in *A. thaliana* and in *Panicum mosaic virus* and its satellite virus in the monocot plant *Brachypodium distachyon* (Love *et al*., 2005, 2012; Mandadi and Scholthof, 2012). Recently, Zhu *et al*. (2014) have shown that the treatment of *N. benthamiana* plants with JA or SA enhances systemic resistance to TMV, and that resistance is further enhanced by pretreatment with JA followed by SA. Remarkably, plants impaired in the JA pathway failed to accumulate SA in the leaves or phloem, and exhibited increased susceptibility, whereas impairment of the SA pathway did not affect JA levels, but increased susceptibility (Zhu *et al*., 2014). JA may modulate early components of the SA pathway, but it is unknown how JA regulates SA biosynthesis and resistance in compatible interactions.

The C2 proteins of a few geminiviruses have been shown to down-regulate JA-responsive genes by interfering with SCF complexes (Skp, Cullin, F-box-containing complexes), thereby affecting certain hormonal responses (JA, ABA or Aux) via ubiquitination (Lozano-Duran *et al*., 2011). The same study also showed that continuous treatment with JA (every other day) decreased the DNA titres of *Beet curly top virus* (BCTV) (Lozano-Duran *et al*., 2011).

Compared with its known roles in compatible interactions, the roles of JA in incompatible interactions are more controversial. JA has been shown to act against *N*-mediated resistance to TMV in tobacco; *N*-mediated resistance to TMV was enhanced in the Nt*COI1*-RNAi line, indicating that *COI* negatively affects resistance (Oka *et al*., 2013). It has also been reported that silencing of *AOS* (*ALLENE OXIDE SYNTHASE*), a JA biosynthesis gene, enhanced resistance, and exogenous application of methyljasmonate (MeJA) reduced local resistance to TMV and permitted systemic movement, implying that such treatment abolished *N* resistance to TMV. The authors also found that the enhanced resistance of the *Nt-COI1*-RNAi line was partially a result of elevated SA levels in the *COI1*- or *AOS*-silenced plants (Oka *et al*., 2013).

It remains unclear why *COI-1* knock-down had different effects on compatible and incompatible viruses. More examples are required to confirm the roles of JA in incompatible interactions. Furthermore, unravelling the effects of JA on compatible and incompatible interactions requires further kinetic analyses involving other antagonistic/synergistic defence hormones, which may be involved in such regulation at the initial phase of infection.

Ethylene

Et is involved in certain developmental stages, such as senescence, as well as in the defence response to necrotrophic pathogens (van der Ent and Pieterse, 2012; Graham *et al*., 2012). Et does not appear to be essential for plant resistance against viruses, with only a few studies describing an involvement of Et in symptom development. Geri *et al*. (2004) used mutagenesis screening of a transgenic line of P6 (which is solely responsible for stunting and chlorosis symptoms in Arabidopsis infected with CaMV) to identify mutants in which the P6 phenotype was suppressed; symptoms were milder and delayed when compared with those of infected

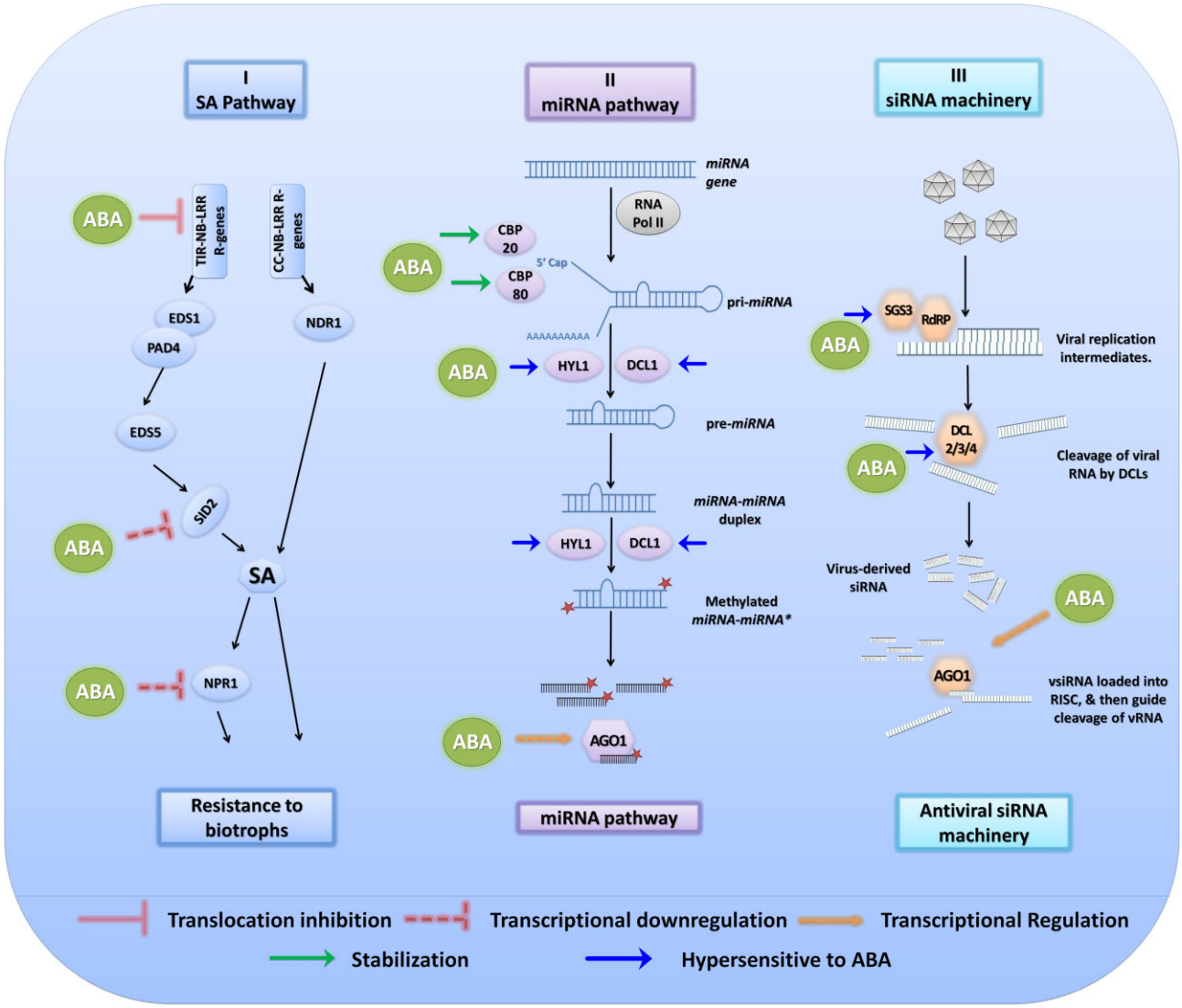

Fig. 2 Roles of abscisic acid (ABA) in modulating plant defence and hormone responses. I. SA pathway: ABA suppresses the salicylic acid (SA) pathway. Certain temperature-sensitive *R* proteins [Toll/interleukin-1 receptor-nucleotide-binding site-leucine-rich repeat (TIR-NBS-LRR)-type] translocate from the cytosol to the nucleus at low temperatures. In ABA-deficient mutants, such proteins lose their temperature sensitivity, localize in the nucleus and act against their specific pathogens (Mang *et al*., 2012). Pretreatment with ABA suppresses certain genes involved in SA biosynthesis and signalling (e.g. *SID1* and *NPR1*), and thereby negates SA-induced resistance against biotrophic pathogens (Yasuda *et al*., 2008). II. miRNA pathway: ABA affects microRNA (miRNA) biosynthesis at several levels. Certain mutants, such as *hyl1* (HYPONASTIC LEAVES 1) and *dcl1* (DICER-LIKE 1), are hypersensitive to ABA, whereas *AGO1* homeostasis is regulated by ABA through *miR168a*. At the protein level, ABA stabilizes CBP20 and CBP80 (CAP-BINDING PROTEINS 20 and 80), which are required for capping of the 5' untranslated region (UTR) of synthesized miRNAs (Kim *et al*., 2008; Li *et al*., 2012). III. siRNA machinery: ABA indirectly affects the small interfering RNA (siRNA) pathway. The mutants *dcl2*, *dcl3*, *dcl4* and *sgs3* (SUPPRESSOR OF GENE SILENCING 3) are hypersensitive to ABA (Zhang *et al*., 2008).

WT plants. Although infected WT plants were partially Et insensitive, the P6 transgenic line was almost completely Et insensitive. The authors deduced that the symptoms of CaMV infection in Arabidopsis depend on interactions between P6 and certain components of the Et pathway (Geri *et al*., 2004). Et has been reported previously to be responsible for symptom development in cucumber infected with CMV (Marco and Levy, 1979). A recent study has revealed that mutants of the Et pathway [such as *acs1* (*1-aminocyclopropane-1-carboxylate synthase*), *erf106* (*ethylene-responsive transcription factor 106*) and *ein2* (*ethylene insensitive 2*)] are resistant to TMVcg. In addition, 1-aminocyclopropane-1-carboxylic acid (ACC) application enhanced TMVcg accumulation in treated plants (Chen *et al*., 2013). The study also showed that *ACS6* and *ERF104* are significantly up-regulated in *wrky8* mutants. It was found that *WRKY8* negatively regulates these genes by binding to W-box clusters within their promoter (Chen *et al*.,

2013). Although Et may support symptom development in the case of CaMV infection and systemic movement in the case of TMVcg infection, an interesting, opposing study demonstrated the importance of Et to the ER against TBSV in tobacco plants. TBSV accumulates in tobacco plants insensitive to Et (ETR line), but not in WT plants (Sansregret *et al*., 2013). It remains to be determined how Et positively regulates ER in response to TBSV in this case.

In *N. tabacum* plants resistant to TMV or TNV, the precursor of Et, ACC, accumulates locally around necrotic areas, indicating a possible contribution to lesion formation (Delaat and Vanloon, 1983; Ohtsubo *et al*., 1999). However, spraying plants with ACC prior to infection prevented lesion formation (Delaat and Vanloon, 1983; Knoester *et al*., 2001; Ohtsubo *et al*., 1999). Similarly, spraying *P. vulgaris* with ACC before WClMV infection reduced viral titres. In addition, spraying with JA and SA helped to reduce viral levels (Clarke *et al*., 1998). Although endogenous JA and Et have antagonistic effects on SA-mediated defences against viruses, these findings imply that the timing of treatment greatly affects plant defence against viral infection.

Auxins

Auxs play a key role in plant growth and development by maintaining apical dominancy, and mutants in the Aux signalling pathway or responsive factors display an aberrant growth phenotype (Benjamins and Scheres, 2008). Many viral infections result in aberrant phenotypes, such as stunting, leaf curl and loss of apical dominance, which resemble those of mutants with compromised Aux biosynthesis and/or signalling (Kazan and Manners, 2009). For example, CMV and ToMV infections of tomato cause tomato shoestring mosaic disease (Andrade *et al*., 1981; Pratap *et al*., 2012), which has symptoms that resemble the phenotype of *WIRY* mutants. *WIRY* genes were subsequently found to be involved in siRNA biogenesis. In *WIRY* mutants, levels of trans-acting (ta)-siRNAs that regulate the Aux response factors *ARF3* and *ARF4* were reduced, whereas levels of their target *ARF*s were elevated. These findings suggest that failure to negatively regulate *ARF3* and *ARF4* underlies the wiry phenotype (Yifhar *et al*., 2012).

The manipulation of specific ARFs by viruses affects symptom development. For example, TMV replicase interacts directly with Arabidopsis *PAP1* (*PHYTOCHROME-ASSOCIATED PROTEIN 1*)/*IAA26* (*INDOLE-3-ACETIC ACID INDUCIBLE 26*), *IAA18* and *IAA27* proteins through the helicase domain. A helicase-mutated TMV (TMV-V1087I) that cannot bind these factors does not accumulate to levels comparable with those of WT-TMV, and fails to induce stunting symptoms in older plants (10–12 weeks); however, it continues to replicate and move normally in younger plants (4–6 weeks) (Padmanabhan *et al*., 2005, 2008). The interaction between TMV replicase and certain AUX/IAA proteins selectively enhances TMV pathogenicity. TMV disrupts these factors, thereby reprogramming the cellular machinery for enhanced replication and movement. In contrast, *ARF8* is the major cause of developmental defects in TuMV-infected plants and in the HcPro-transgenic line (HcPro is the VSR of TuMV), based on findings that the *arf8* mutant alleviates the developmental defects induced by HcPro. The *arf8* mutant does not affect RNA silencing, suppression by VSRs or the virulence/accumulation of TuMV. Disruption of the regulation of *ARF8* alone underlies most defects caused by VSR expression in infected WT and transgenic plants (Jay *et al*., 2011). Thus, some viruses, such as TMV, partially hijack the Aux signalling pathway and some of its responsive factors. In addition, if a biotrophic infection induces Aux, the HR is usually down-regulated and the SA signalling pathway is repressed, which indicates a possible repressive role of Aux on SA (Benjamins and Scheres, 2008; Robert-Seilaniantz *et al*., 2007).

In summary, some viruses interfere with certain Aux factors involved in apical dominance, and manipulate their functions and subcellular localization as a means to promote their own replication and dissemination (Fig. 1). The manipulation of ARFs by viruses accounts for the phenotypic defects observed after viral infection. Dysfunctions in such ARFs often affect symptom severity and delay systemic spread. Remarkably, virus-induced symptoms mediated by Aux may not be associated with viral titres (Jay *et al*., 2011; Satoh *et al*., 2011).

Cytokinins

CKs are mainly produced in the meristemic zones of shoots and translocated to actively growing areas. They promote cell proliferation and elongation, and are involved in various developmental processes, including transduction of nutritional signals and delay of senescence (Aloni *et al*., 2005; Sakakibara, 2006). In addition, some bacterial and fungal pathogens produce CKs. Much like Auxs, CKs suppress defence responses (such as HR) to *Pseudomonas savastanoi* (Robert-Seilaniantz *et al*., 2007). However, this does not seem to be the case with biotrophs, such as viruses, which do not produce CKs. For example, knock-down of *S*-adenosylhomocysteine hydrolase (*SAHH*), which mediates the methylation of the 5' end of some viral RNAs and is a prerequisite for the replication of such viruses, enhanced plant resistance to viruses requiring *SAHH*, such as PVX, CMV and TMV (Choi *et al*., 2011; Masuta *et al*., 1995). Interestingly, PVY, which does not require *SAHH*, was also unable to replicate in these transgenic lines. Many of these transgenic plants exhibited a stunted phenotype, accompanied by an increase of approximately three-fold in CKs in root exudates, when compared with WT plants. This finding led the authors to infer that CKs may play a role in plant resistance to viruses (Choi *et al*., 2011; Masuta *et al*., 1995).

Together with SA, plant-derived CKs stimulate defence responses to biotrophs. CKs activate the transcriptional regulator *ARR2* (*ARABIDOPSIS RESPONSE REGULATOR 2*), which positively modulates SA signalling by interacting with the SA-responsive

factor TGA3 (TGA1A-RELATED GENE 3) (Choi *et al*., 2011). Indeed, ARR2 binds directly to the promoters of *PR-1* and *PR-2* to induce their transcription. Consistent with this finding, impairment of the SA pathway in *npr1-1* or *NahG* lines failed to mediate the CK induction of *ARR2*. Over-expression of *ARR2* increased the transcription of genes involved in SA-biosynthesis and signalling (*SID1*, *SID2*, *PR-1* and *PR-5*) in plants challenged with the biotroph *P. syringae* pv. *tomato* (*Pst* DC3000). Accordingly, the effect of CK is not observed in SA signalling mutants. Thus, CKs can act synergistically on the SA signalling pathway. Treatment with the CK dihydrozeatin (DHZ, 50 nM/L) reduced viral RNA and CP levels, but did not significantly affect the level of SA-responsive genes, such as *PR-1* and *NPR1*, in WClMV-infected *P. vulgaris*, whereas 1 mM/L SA reduced levels of WClMV RNA and CP. The suppressive effect of DHZ on WClMV infection lasted until only 9 dpi (Galis *et al*., 2004), in contrast with earlier results showing that SA or DHZ conferred full resistance to WClMV (Clarke *et al*., 1998). In addition, WClMV infection specifically decreased the active forms of CK during the first days of infection; the authors proposed that production of the inactive form of 9-glucoside was a direct response to WClMV infection (Clarke *et al*., 1999). Higher concentrations of DHZ may have more profound and prolonged antiviral effects. The antibiotrophic effect of CKs largely depends on SA biosynthesis, and is probably dose dependent (Argueso *et al*., 2012) (Fig. 1). Argueso *et al*. (2012) suggested a model of plant defence in which CK levels help to determine the amplitude of SA-related immunity, which is regulated in part by type-A *ARRs* (*ARR3*, *ARR4*, *ARR5*, *ARR6*, *ARR8* and *ARR9*). Most previous studies of virus–CK interactions have investigated compatible interactions for only a few viruses, and the role of CKs in resistant plants with the *R* gene has not been addressed.

Gibberellic acid

GA induces seed germination, promotes stem elongation and modulates flowering (Sun and Gubler, 2004). This hormone promotes plant growth by inhibiting DELLA proteins, which are negative regulators of plant growth (Robert-Seilaniantz *et al*., 2007). GA seems to have a negative role in plant defence. Loss-of-function mutants of DELLA increase plant resistance to biotrophs, such as *Pst* DC3000, but exhibit hypersusceptibility to infection with necrotrophs. GA may serve to facilitate defences to biotrophs or necrotrophs by partially modulating the balance between SA- and JA/Et-mediated signalling pathways (Robert-Seilaniantz *et al*., 2007).

Ent-kaurene oxidase, a key factor in the biosynthesis of gibberellins (Helliwell *et al*., 1998), interacts with the P2 outer capsid protein of *Rice dwarf virus* (RDV) (Zhu *et al*., 2005). Rice plants infected with RDV exhibited a dwarf phenotype and reduced levels of ent-kaurene oxidase and GA1, but these defects were rescued by exogenous application of GA3. The interaction between P2 and ent-kaurene oxidase-type proteins may interfere with the biosynthesis of phytoalexins, thus promoting viral replication, but this needs to be proven experimentally (Pallas and Garcia, 2011; Zhu *et al*., 2005). Similarly, TuMV infection of non-heading Chinese cabbage decreased GA accumulation (Wang *et al*., 2011b).

Brassinosteroids

BRs are a class of polyhydroxysteroids that affect many cellular processes, including elongation, proliferation, differentiation, membrane polarization and proton pumping (Clouse and Sasse, 1998; Xia *et al*., 2010). They also affect disease resistance at several levels in tobacco and rice (Nakashita *et al*., 2003). In potato, BRs can reduce viral infection in starting plant materials at various stages of development until the second tuber generation. In addition, BR treatment decreased levels of TMV and other biotrophs in tobacco plants (Hayat *et al*., 2011). A BR receptor, the leucine-rich repeat receptor-like kinase (LRR-RLK) Brassinosteroid Insensitive-1 (BRI1), and several pattern recognition receptors (PRRs) interact with the co-receptor BRI1-associated kinase 1 (BAK1) in a ligand-dependent manner. BAK1 has been characterized as a general regulator of plant immunity against certain biotrophs, as well as hemibiotrophs, such as *Hyaloperonospora arabidopsidis* and *P. syringae* (Liebrand *et al*., 2014; Roux *et al*., 2011). BAK1 was also found to be essential for plant basal immunity during compatible interactions with RNA viruses. For example, TCV, ORMV and TMV accumulated to higher levels in the *bak1-4* and *bak1-5* mutants than in WT plants (Korner *et al*., 2013). Notably, BR-induced defence to biotrophs seems to be independent of SA. Treatment of plants with BR did not affect the expression of SAR marker genes (*PR-1*, *PR-2* and *PR-5*) or the levels of free or total SA (Nakashita *et al*., 2003). In a model of TMV infection, the average lesion size was smaller in BR-treated *NahG*-transgenic plants than in water-treated controls. Furthermore, SAR development was enhanced by BR treatment 24 h before TMV infection in resistant tobacco harbouring the *N* gene. Similar findings were obtained in a model of *Pst* infection (Nakashita *et al*., 2003). SA-independent, BR-induced defence is of particular interest. Studies using additional viruses may unveil novel strategies by which plants tolerate or resist viral infections.

CONCLUDING REMARKS

Viral infections disrupt many processes, resulting in temporal changes in hormone signalling and responses, metabolites, and transcriptomic and small RNA profiles. The affected networks are wide and overlapping, and careful elucidation of their interactions is required to fully understand the interplay between host and virus. Elucidation of cellular rearrangements at very early steps of viral infection, during which cell conditions are altered (to induce

resistance or to favour virus multiplication and spread) requires further investigation. Future identification of the roles of hormones in plant virus interactions and cross-talk among hormone pathways will help to determine the molecular mechanisms by which plants resist infection. In addition, our understanding of plant–virus interactions has primarily been obtained from work on dicot plants. As such, much less is known about monocots. Work on monocots has focused on breeding for resistance, leaving the underlying mechanisms largely unexplored (Mandadi and Scholthof, 2013). A few steps have been taken in the latter direction, especially following the sequencing of the genome of *B. distachyon* (International Brachypodium, 2010). Such work uncovered some of the mechanisms underlying *Bsr1* (*Barley stripe mosaic virus resistance 1*)-mediated resistance to *Barley stripe mosaic virus* (Lee *et al*., 2012). The need to understand various molecular mechanisms in economically important monocot crops will encourage the use of *B. distachyon* as an alternative model plant for further research.

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