MICROREVIEW

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Manipulation of auxin signalling by plant viruses

Maximilian Müllender[1] (iD) | Mark Varrelmann[1] | Eugene I. Savenkov[2] (iD) | Sebastian Liebe[1] (iD)

[1]Department of Phytopathology, Institute of Sugar Beet Research, Göttingen, Germany

[2]Department of Plant Biology, Uppsala BioCenter SLU, Swedish University of Agricultural Sciences, Linnean Center for Plant Biology, Uppsala, Sweden

Correspondence
Sebastian Liebe, Department of Phytopathology, Institute of Sugar Beet Research, Göttingen, Germany.
Email: liebe@ifz-goettingen.de

Abstract

Compatible plant–virus interactions result in dramatic changes of the plant transcriptome and morphogenesis, and are often associated with rapid alterations in plant hormone homeostasis and signalling. Auxin controls many aspects of plant organogenesis, development, and growth; therefore, plants can rapidly perceive and respond to changes in the cellular auxin levels. Auxin signalling is a tightly controlled process and, hence, is highly vulnerable to changes in the mRNA and protein levels of its components. There are several core nuclear components of auxin signalling. In the nucleus, the interaction of auxin response factors (ARFs) and auxin/indole acetic acid (Aux/IAA) proteins is essential for the control of auxin-regulated pathways. Aux/IAA proteins are negative regulators, whereas ARFs are positive regulators of the auxin response. The interplay between both is essential for the transcriptional regulation of auxin-responsive genes, which primarily regulate developmental processes but also modulate the plant immune system. Recent studies suggest that plant viruses belonging to different families have developed various strategies to disrupt auxin signalling, namely by (a) changing the subcellular localization of Aux/IAAs, (b) preventing degradation of Aux/IAAs by stabilization, or (c) inhibiting the transcriptional activity of ARFs. These interactions perturb auxin signalling and experimental evidence from various studies highlights their importance for virus replication, systemic movement, interaction with vectors for efficient transmission, and symptom development. In this microreview, we summarize and discuss the current knowledge on the interaction of plant viruses with auxin signalling components of their hosts.

KEYWORDS
Aux/IAA, auxin response factor, phytohormone

1 | INTRODUCTION

Plant viruses are of great importance to agriculture as they constantly threaten crop production by causing major economic losses in yield and quality of harvested tissue (Rybicki, 2015; Scholthof et al., 2011). The interaction of viruses with their host plants is often associated with rapid alterations in phytohormone homeostasis and signalling, which is an important aspect in plant–virus interactions as highlighted in a recent review (Zhao & Li, 2021). Plant defence hormones, namely, salicylic acid (SA), jasmonic acid (JA), and ethylene

(ET), are important for mounting the primary defence responses to the pathogen attack, whereas growth-related phytohormones, including auxin, cytokinins, brassinosteroids, abscisic acid, and gibberellins, can modulate the plant immune system (Han & Kahmann, 2019; Islam et al., 2019). Auxin controls a multitude of cellular and developmental processes including cell division and enlargement, differentiation, vascular tissue formation, tropic responses to light and gravity, apical dominance, and organ development (Abas et al., 2006; Dharmasiri et al., 2005b; Friml et al., 2002; Gray et al., 2001; Ori, 2019). The major natural auxin occurring in plants is indole-3-acetic acid (IAA), and plants have universally conserved machinery for its synthesis.

Alterations in host auxin metabolism are important for plant–microbe interactions as these changes stimulate plant cell growth, modulate defence responses, and alter host physiology. Bacterial and fungal plant pathogens can interfere with auxin metabolism by pathogen-produced enzymes that either synthesize or inactivate auxin (Kunkel & Harper, 2018; Ludwig-Müller, 2015). Plant viruses do not encode such enzymes owing to the limitations imposed by the small size of their genomes. The small genome size of plant viruses means that most viral proteins are multifunctional, suggesting that some viral proteins might subvert phytohormone-mediated responses (e.g., through direct interaction with signalling components) for the virus' benefit. Indeed, research over the last decade, mostly on RNA viruses, has established that plant viruses are able to manipulate auxin signalling of their hosts for their own advantage.

2 | MECHANISM OF AUXIN SENSING IN PLANTS

Auxin response factors (ARFs) and auxin/indole acetic acid (Aux/IAA) proteins are key components in the regulation of auxin signalling events. Members of the ARF transcription factor (TF) family across plant species share four highly conserved domains. ARFs bind as dimers to auxin-responsive elements (AuxREs) in the promoters of auxin-regulated genes via an N-terminal B3-type DNA-binding domain (DBD). The variable middle region of ARF proteins functions as either activation or repression domain for auxin-responsive genes. The C-terminal dimerization domain (CTD) contains a Phox/Bem1p (PB1) domain that mediates homo- and heterodimerization, as well as heterodimerization with Aux/IAA proteins under low auxin concentrations (Figure 1a) (Chandler, 2016; Guilfoyle, 2015; Guilfoyle & Hagen, 2007; Piya et al., 2014). Aux/IAA proteins represent key regulators in auxin-mediated signalling as they are able to respond to the auxin levels in the cells.

Aux/IAAs are short-lived, small (18–36 kDa) proteins with four highly conserved domains (Abel & Theologis, 1996; Oeller et al., 1993). The N-terminal domain I is characterized by the presence of the consensus sequence LxLxL (where L refers to leucine and x to any amino acid residue), a conserved ethylene response factor-associated amphiphilic repression (EAR) motif (Tiwari et al., 2004). At low auxin concentrations, this domain is responsible for the dominant repressive activity of Aux/IAA proteins as it binds to tetramers
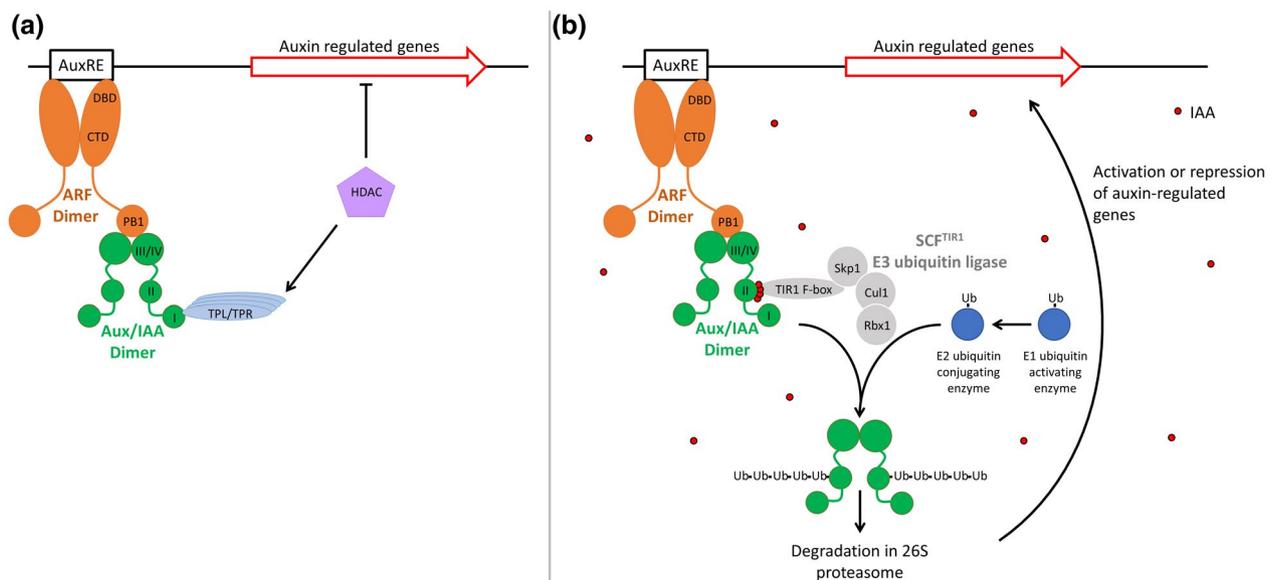

FIGURE 1 (a) The state of the auxin signalling pathway under low auxin conditions. Auxin responsive factors (ARFs) are bound as dimers (CTD, C-terminal dimerization domain) to auxin-responsive elements (AuxREs) on the DNA with their B3-type DNA-binding domain. Aux/IAA dimers are bound via their domain III/IV to a type I/II Phox/Bem1p (PB1) protein–protein interaction domain. Domain I of Aux/IAAs interacts with TOPLESS and TOPLESS-RELATED corepressors (TPL/TPR) that recruit a histone deacetylase (HDAC). Resulting modifications of the DNA lead to down-regulation of the transcriptional activity of auxin-regulated genes. (b) The state of the auxin signalling pathway under high auxin conditions. Auxin acts as molecular glue between domain II of Aux/IAA proteins and the SCF^{TIR1} E3 ubiquitin ligase complex (subunits: Skp1, S-phase kinase-associated protein 1; Rbx1, RING-box protein 1; Cul1, Cullin 1; TIR1 F-box, F-box protein). Ubiquitin (Ub) is first activated by the E1 ubiquitin-activating enzyme and then bound to domain II of Aux/IAA proteins via an E2 ubiquitin-conjugating enzyme and the Rbx1 subunit of the SCF^{TIR1} E3 ubiquitin ligase. The ubiquitinated Aux/IAA proteins are degraded in the 26S proteasomes and are no longer bound to ARF dimers. ARF dimers are released and can now operate as transcriptional activators or repressors

of the corepressors TOPLESS (TPL) and TOPLESS-RELATED (TPR) (Szemenyei et al., 2008). TPL/TPR corepressors harbour WD40 repeats, which recruit chromatin-modifying enzymes such as histone deacetylases (HDACs). HDACs modify chromatin to be transcriptionally inactive, leading to repression of auxin-responsive genes (Causier et al., 2012; Ke et al., 2015; Kieffer et al., 2006). Domain II contains the primary degron sequence QV**VGWPP**VRSY**RK**N (highly conservative residues are in bold and underlined) that mediates the auxin responsiveness (Song & Xu, 2013). The C-terminal domains III and IV of Aux/IAAs are similar to the PB1 domains of ARFs that allow interactions among these TFs and, hence, suppress the regulatory activities of ARFs (Dinesh et al., 2015; Guilfoyle, 2015; Guilfoyle & Hagen, 2012; Korasick et al., 2015; Tiwari et al., 2004).

When the auxin concentration increases (Figure 1b), Aux/IAA proteins are ubiquitinated by a ubiquitin SCF-type E3 ligases (E3) via an E1/E2 enzyme system and degraded by the 26S proteasome (Dharmasiri et al., 2005a; Hershko & Ciechanover, 1998; Leyser, 2018; Pickart, 2001; Tan et al., 2007; Thelander et al., 2019). Auxin acts as a molecular glue and connects leucine-rich repeats of F-box proteins with the conserved degron motif (domain II) of Aux/IAAs (Tan et al., 2007). As part of the SCF-type E3 ligases, the F-box protein conveys the substrate specificity to the Aux/IAAs (Hayashi et al., 2008; Ruegger et al., 1998). SCF-type E3 ligases are named after their three subunits: S-PHASE KINASE-ASSOCIATED PROTEIN 1 (SKP1), a RING-box protein 1 (RBX1)–CULLIN 1 (CUL1) dimer, and an F-box protein (Deshaies, 1999). The F-box protein is a member of the auxin-perceiving coreceptor family TRANSPORT INHIBITOR RESPONSE 1/AUXIN SIGNALLING F-BOX 1-5 (TIR1/AFB) (Kepinski & Leyser, 2005; Tan et al., 2007). The RBX1–CUL1 dimer catalyses ubiquitin polymerization and is responsible for ubiquitination of the target proteins. The multiprotein complex responsible for the auxin-dependent interaction and subsequent degradation of Aux/IAAs is called SCF^{TIR1} (Dharmasiri et al., 2005b; Prigge et al., 2016; Ruegger et al., 1998).

Upon degradation of Aux/IAAs, ARFs can act as TFs regulating the expression of primary auxin-responsive genes. The gene families *Small Auxin Up-regulated RNA* (*SAUR*), *Gretchen Hagen 3* (*GH3*), and *Lateral Organ Boundaries Domain* (*LBD*) are often part of an early auxin response (Catalá et al., 2000; Fan et al., 2012; Hagen & Guilfoyle, 1985; Knauss et al., 2003). *Aux/IAAs* are also primary auxin-responsive genes whose expression is rapidly elevated shortly after auxin application (Abel & Theologis, 1996; Li et al., 2016; Theologis et al., 1985).

3 | PLANT VIRUS INFECTIONS INDUCE CHANGES IN AUXIN METABOLISM

Auxin metabolism comprises biosynthesis, conjugation, and degradation (Casanova-Sáez et al., 2021). It is now well established that IAA is mainly synthesized from tryptophan via the indole-3-pyruvic acid (IPyA) pathway (Chen et al., 2020; Woodward, 2005; Zhao, 2001; Zheng et al., 2013), whereas several other redundant pathways

function in parallel, including auxin production via tryptamine (TRA) (Facchini et al., 2000; Hull et al., 2000; Mikkelsen et al., 2000; Pollmann et al., 2002, 2003). The inactivation of auxin is important to maintain auxin homeostasis in plants (Ljung, 2013). Metabolic inactivation of IAA is performed through oxidation and conjugation processes. Whereas auxin-inducible acyl amino synthetases of the *GH3* gene family convert IAA to IAA–amino acid conjugates (Staswick et al., 2005), uridine diphosphate glucosyltransferase oxidizes IAA to 2-oxindole-3-acetic acid (Peer et al., 2013; Pěnčík et al., 2013).

Viral infections are often accompanied by changes in the expression of the key genes of these pathways leading to either an increase in accumulation or a decrease in the cellular levels of auxin. In rice plants infected with rice black streaked dwarf virus (RBSDV; genus *Fijivirus*, family *Reoviridae*), the concentration of the main active form of IAA gradually decreases whereas the amount of the intermediate degradation product, IAA–aspartate, sharply increases (Huang et al., 2018). This coincides with the down-regulation of auxin-biosynthesis genes and a strong up-regulation of the *GH3.8* gene, which encodes an IAA–amino synthetase responsible for the synthesis of IAA–aspartate conjugate (Zhang et al., 2019). In contrast, sugar beet plants infected with beet necrotic yellow vein virus (BNYVV; genus *Benyvirus*, family *Benyviridae*) are characterized by elevated auxin levels (Pollini et al., 1990). Furthermore, in those plants, the *GH3.1* gene, involved in auxin conjugation and inactivation, is strongly down-regulated (Gil et al., 2020). Similarly, rice dwarf virus (RDV; genus *Phytoreovirus*, family *Reoviridae*) triggers auxin biosynthesis in rice (Qin et al., 2020).

In *Arabidopsis thaliana*, the expression of HC-Pro, a viral suppressor of RNA silencing of tobacco vein banding mosaic virus (TVBMV; genus *Potyvirus*, family *Potyviridae*), decreases the DNA methylation in the promoters of the *YUCCA* genes of the IPyA pathway, leading to transcriptional activation of these genes and ultimately to elevated auxin levels (Yang et al., 2020). Moreover, transcriptional changes in auxin-responsive genes have also been reported for many other plant–virus pathosystems (Li et al., 2017; Liu et al., 2019; Padmanabhan et al., 2019; Pierce & Rey, 2013; Zhou et al., 2016), and therefore seem to be a general response of plants to virus infection.

4 | PLANT VIRUSES DISRUPT AUXIN SENSING BY TARGETING Aux/IAA PROTEINS: CASE STUDIES

4.1 | Tobacco mosaic virus

The interaction between a viral protein and a plant Aux/IAA was first described for the *A. thaliana*–tobacco mosaic virus (TMV; genus *Tobamovirus*, family *Virgaviridae*) pathosystem (Padmanabhan et al., 2005). IAA26 was found to interact with the helicase domain of the TMV replicase (Figure 2a). The nuclear localization of IAA26 was disrupted by coexpression with the TMV replicase, leading to a cytoplasmic distribution of IAA26. Therefore, it was hypothesized
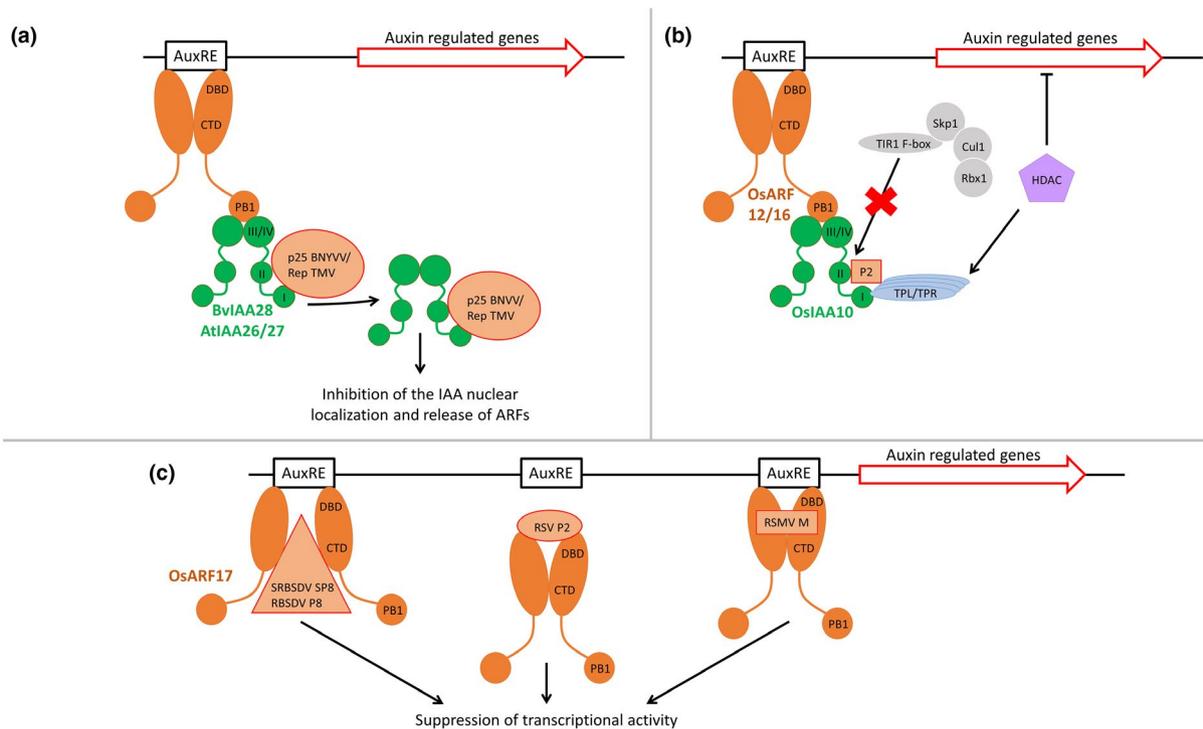
FIGURE 2 (a) Interaction of the pathogenicity factors p25 from beet necrotic yellow vein virus (BNYVV) and replicase (Rep) from tobacco mosaic virus (TMV) with the Aux/IAA proteins BvIAA28 from sugar beet (*Beta vulgaris*) and AtIAA26/27 from *Arabidopsis thaliana*. This interaction inhibits the nuclear localization of Aux/IAAs and suppresses their regulatory properties, leading to the release of the auxin response factors (ARFs), which can then take up their roles as transcription factors again. (b) Interaction of rice dwarf virus (RDV) with the auxin signalling pathway of rice (*Oryza sativa*). OsIAA10 is stabilized by the viral protein P2 in a dose-dependent manner. An interaction of the SCF$^{TIR1/AFBs}$ complex with domain II of OsIAA10 is prevented by P2 even under high auxin concentrations. OsARF12 and 16 are still suppressed by OsIAA10 and genes involved in early synthesis of indole acetic acid (IAA) as well as auxin-responsive genes are down-regulated during infection. (c) Interaction of the rice-infecting viruses southern rice black streaked dwarf virus (SRBSDV, SP8), rice black streaked dwarf virus (RBSDV, P8), rice stripe virus (RSV, P2), and rice stripe mosaic virus (RSMV, M protein) with ARFs. SP8 from SRBSDV and P8 from RBSDV were found to specifically interact with the C-terminal dimerization domain (CTD) of OsARF17, preventing its dimerization. The RSV protein P2 was found to interact with the DNA-binding domain (DBD) of OsARF17, which impedes the interaction with AuxREs, and the M protein from RSMV interacts with the MR-CTD of OsARF17. All these interactions lead to suppression of transcriptional activity of OsARF17

that translocation of IAA26 to the cytoplasm impairs its putative function as a transcriptional regulator of auxin-responsive genes in the nucleus (Padmanabhan et al., 2005, 2006). Indeed, this hypothesis was supported by changes in the transcript levels of auxin-responsive genes in TMV-infected plants. Furthermore, transgenic plants silenced for *IAA26* showed TMV-like symptoms. Additionally, a TMV mutant (TMV-V1087I) expressing an altered replicase with a single amino acid substitution (V1087I) was incapable of interacting with IAA26. This did not lead to a change of the subcellular localization of IAA26 and induced only attenuated developmental symptoms in the infected plants. The TMV-V1087I mutant replicated and spread in young leaf tissue similar to the wild-type (wt) virus, but the virus accumulation was reduced in older tissue (Padmanabhan et al., 2008). The protein levels of IAA26 were found to be higher in mature tissue, and therefore it was concluded that the interaction of TMV replicase with IAA26 is crucial for supporting virus replication in older leaves. Consequently, the accumulation of the TMV-V1087I mutant was further reduced in transgenic *A. thaliana* plants expressing a degradation-resistant variant of IAA26 (Padmanabhan et al.,

2008). Later, it was shown that IAA26 is predominantly expressed in vascular tissue and its nuclear localization is disrupted by TMV in companion cells of the vascular bundle (Collum et al., 2016). The ability of wt TMV to interact with Aux/IAAs resulted in an increased ability for phloem loading and systemic spread in mature tissue compared to the TMV-V1087I mutant.

Interestingly, the expression levels of *pectin methylesterase 5* (*PME5*), *microtubule end-binding 1a* (*EB1a*), *PD-located protein 3* (*PDLP3*), and members of the *β-1,3-glucanase* gene family were altered in transgenic plants overexpressing a degradation-resistant IAA26 variant (Collum et al., 2016). It is assumed that these genes are involved in cell-to-cell movement of TMV. Additionally, the expression levels of defence-related genes were changed, suggesting that the interaction of TMV with IAA26 is also important for mounting an antiviral defence. The interaction of TMV with IAA26 seems to be mediated by a highly conserved domain of IAA26 because the orthologous proteins from tomato and *Nicotiana benthamiana* also interact with the TMV replicase, leading to a disruption of their nuclear localization (Collum et al., 2016; Padmanabhan et al., 2008).

Knockdown of *IAA26* expression in tomato resulted in a phenotype similar to that of TMV-infected plants. Besides IAA26, two other *A. thaliana* Aux/IAA proteins, namely, IAA27 and IAA18, were found to interact with TMV replicase, but with lower affinity compared to IAA26 (Padmanabhan et al., 2006). Furthermore, upon TMV infection, only the nuclear localization of IAA27 was disrupted, whereas the localization of IAA18 to the nucleus was not affected. So far, the role of IAA27 and IAA18 in TMV pathogenesis remains elusive.

4.2 | Rice dwarf virus

The mechanism by which plant viruses manipulate auxin signalling has also been well characterized for RDV, which causes dwarfism in rice. Genes involved in early synthesis of IAA as well as auxin-responsive genes are down-regulated during RDV infection (Satoh et al., 2011). The RDV P2 protein interacts with domain II of OsIAA10, which impedes the interaction of OsIAA10 with OsTIR1 (Jin et al., 2016) (Figure 2b). Moreover, OsIAA10 is stabilized by P2 in a dose-dependent manner and its degradation through auxin perception by the SCF$^{TIR1/AFBs}$ complex is prevented. Transgenic rice plants overexpressing *OsIAA10* develop an auxin-resistant phenotype that resembles symptoms of RDV-infected rice plants including stunting, higher number of tillers, shorter crown roots, and lower seed fertility. Moreover, these transgenic plants display more severe symptoms after natural RDV infection whereas knockout of *OsIAA10* reduces virus replication and symptom severity. These findings highlight the important role of the interaction between P2 and OsIAA10 for enhancing virus infection.

The active role of auxin in the defence against RDV was addressed in a recent study (Qin et al., 2020). Two ARF proteins, namely, OsARF12 and OsARF16, were identified as interaction partners of OsIAA10, which positively regulates rice antiviral defence against RDV. Moreover, the OsWRKY13 TF was identified as a target of OsARF12 as OsARF12 binds to an AuxRE element in the promoter of *OsWRKY13* to activate transcription of the gene. Knockout of *OsWRKY13* increases virus accumulation and symptom severity. Consequently, the increase of auxin content in RDV-infected rice plants leading to degradation of OsIAA10 and transcription activation of *OsWRKY13* by OsARF12 appears to be a part of an auxin-mediated defence response against RDV (Qin et al., 2020). However, RDV has developed a counter-defence strategy by stabilizing OsIAA10, which leads to repression of OsARF12 and OsARF16 and dampening of OsARF12- and OsARF16-mediated antiviral responses (Jin et al., 2016). Interestingly, P2 is targeted for degradation by the rice E3 ubiquitin ligase OsRFPH2-10 as part of an antiviral defence at the early stages of infection (Liu et al., 2014).

Besides the auxin signalling pathway, RDV can hijack signalling pathways of other phytohormones to enhance infection and virus multiplication. P2 interacts with *ent*-kaurene oxidases, leading to reduced accumulation of GA, which, in turn, results in a dwarf phenotype of RDV-infected rice plants (Zhu et al., 2005). Furthermore, the RDV-encoded protein Pns11 interacts with OsSAMS1 and enhances its enzymatic activity, leading to higher ethylene levels, which in turn

result in enhanced severity of the virus symptoms in RDV-infected rice plants (Zhao et al., 2017). Thus, collectively the disease symptoms induced by RDV are probably the result of disrupting signalling pathways of several phytohormones.

4.3 | Beet necrotic yellow vein virus

Another plant virus known to interfere with auxin signalling pathways is BNYVV, which causes rhizomania disease in sugar beet. The taproot of BNYVV-infected sugar beet plants is characterized by massive lateral root (LR) formation, which requires the presence of the P25 virulence factor (Tamada et al., 1999). LR formation is a developmental process governed by auxin and specific Aux/IAA–ARF modules (Trinh et al., 2018). The taproot of infected sugar beet plants undergoes comprehensive transcriptional reprogramming of auxin-regulated pathways (Gil et al., 2018, 2020; Schmidlin et al., 2008). This includes the up-regulation of *LBD* TFs and *EXPANSINSs* (*EXPs*), both of which are crucial for LR development. *LBD* TFs are directly activated by ARFs and can activate the expression of *EXP* genes (Lee & Kim, 2013; Lee et al., 2013; Okushima et al., 2007), which encode cell wall-loosening proteins needed for cell elongation during LR formation (Cosgrove, 2015). Additionally, genes involved in auxin biosynthesis via the IPyA and TRA pathways are also strongly activated during BNYVV infection (Gil et al., 2020), which is in accordance with the observation of higher auxin levels in BNYVV-infected taproots (Pollini et al., 1990). However, recently, elevated levels of the conjugated inactive form of auxin IAA–Ala were detected in BNYVV-infected sugar beet plants, suggesting a compensatory plant response to maintain auxin homeostasis (Webb et al., 2020).

A sugar beet cDNA library was screened using a yeast two-hybrid assay to identify host proteins that interact with the P25 virulence factor (Thiel & Varrelmann, 2009). The screen yielded IAA28 as a P25 interaction partner (Gil et al., 2018; Thiel & Varrelmann, 2009). IAA28–P25 interaction occurs via IAA28 domains I and II (Gil et al., 2018). Subcellular localization of coexpressed P25 and IAA28 revealed that P25 inhibits IAA28 nuclear localization similarly to TMV as described above (Figure 2a). Interestingly, BNYVV-infected sugar beet plants characterized by massive LR formation resemble the appearance of tomato plants silenced for Aux/IAA genes (Bassa et al., 2012). By contrast, suppression of LR formation and extreme stunting of the plants is a typical phenotype of the Aux/IAA-overexpressing lines of *A. thaliana* (Fukaki et al., 2002; Rogg et al., 2001). Thus, P25 presumably inactivates the transcriptional repressor activity of IAA28 through the disruption of its nuclear localization, again a mechanism that seems to be similar to the interaction of TMV with auxin signalling described above. Alternatively, P25 may trigger 26S proteasome-mediated degradation of IAA28, but this hypothesis needs to be addressed in future experiments.

The interaction of the P25 virulence factor with auxin signalling pathways seems to occur via signalling components sharing some level of conservation between sugar beet (a host for BNYVV) and *A. thaliana* (a nonhost for BNYVV) as transgenic *A. thaliana* plants

expressing P25 are characterized by increased auxin content, abnormal root branching, and differential expression of auxin-responsive genes (Peltier et al., 2011). Additionally, these transgenic *A. thaliana* plants are more sensitive to treatment with the synthetic auxin 2,4-dichlorophenoxyacetic acid, supporting the idea that P25 increases auxin sensitivity by disrupting the transcriptional activity of Aux/IAA proteins via yet unknown mechanisms. In contrast to sugar beet (*Beta vulgaris* subsp. *vulgaris*) and *A. thaliana*, the experimental host *N. benthamiana* and the crop's wild relative subspecies *B. vulgaris* subsp. *macrocarpa* display stunting, leaf curling, and root developmental defects after BNYVV infection. These symptoms resemble an auxin-insensitive phenotype, suggesting that in these particular species P25 might stabilize IAA28 (or/and other Aux/IAA proteins) similarly to RDV P2–IAA10 interactions described above. These questions require further investigation. However, additional alternatives deserve consideration as small RNA sequencing and subsequent validation of the data revealed an up-regulation of miR396 (in both species in question), resulting in down-regulation of the TIR1 auxin receptor transcript, the cleavage target of miR396 (Fan et al., 2015; Liu et al., 2020). The repression of the auxin response by reducing the expression of the auxin receptor may indicate a host-specific effect of BNYVV on the auxin signalling pathway in hosts other than sugar beet.

5 | PLANT VIRUSES DISRUPT TRANSCRIPTIONAL ACTIVITY OF ARFs

Besides interaction with Aux/IAA proteins, plant viruses are also able to target ARF TFs and disrupt their transcriptional activity (Figure 2c). A comprehensive study (Zhang et al., 2020) investigated the interaction of the rice-infecting viruses southern rice black streaked dwarf virus (SRBSDV; genus *Fijivirus*, family *Reoviridae*), RBSDV (genus *Fijivirus*, family *Reoviridae*), rice stripe virus (RSV; genus *Tenuivirus*, family *Phenuiviridae*), and rice stripe mosaic virus (RSMV; genus *Cytorhabdovirus*, family *Rhabdoviridae*) with ARFs. The two related proteins SP8 from SRBSDV and P8 from RBSDV were found to specifically interact with the CTD of OsARF17, preventing its dimerization and leading to suppression of its activity as a TF. Furthermore, overexpression of *OsARF17* reduced accumulation of both viruses, whereas virus accumulation and symptom severity were enhanced in the knockout mutant rice lines. In the same study the P2 protein of the distantly related RSV was found to interact with the DBD of OsARF17, which impeded its interaction with AuxREs in the promoters and therefore the transcription activation of auxin response genes. Similarly to SRBSDV and RBSDV, the accumulation of RSV and symptom severity were reduced in the transgenic rice lines overexpressing *OsARF17*. Finally, the authors showed that the M protein from the cytorhabdovirus RSMV interacts with the MR-CTD of OsARF17 and represses its transcriptional activity. Overexpression of *OsARF17* resulted in reduced virus accumulation, similarly to the aforementioned viruses. Thus, OsARF17 is important for antiviral defence in rice and several plant viruses have

independently evolved strategies aiming at disrupting the transcriptional activity of this protein.

6 | CONCLUSION

As described above, plant viruses have developed diverse strategies to disrupt auxin signalling by (a) changing the subcellular localization of Aux/IAAs, (b) preventing degradation of Aux/IAAs by stabilization, or (c) inhibiting the transcriptional activity of ARFs. This leads to either activation (a) or suppression (b and c) of auxin signalling. Overall, these changes result in virus-mediated transcriptional reprogramming of auxin-regulated pathways, which ultimately can lead to a suppression of plant defence, efficient virus movement, and symptom development. As shown for TMV, the interaction with Aux/IAAs can help viruses to replicate and move better in older leaf tissue, where Aux/IAAs are present at higher levels. Thus, it has been speculated that disruption of auxin signalling reprogrammes older tissues to make them more compatible with virus replication and movement (Padmanabhan et al., 2008). The formation of an auxin gradient either by local synthesis or by polar transport is crucial to drive plant growth and development. It is possible that the disruption of auxin signalling might help viruses to cope with local auxin maxima in developing tissue. Whether the disruption of auxin signalling also activates a negative or positive feedback loop leading to suppression or activation of auxin biosynthesis remains unclear.

The effects of virus infections on the expression of genes involved in auxin metabolism and the alteration of cellular auxin levels cannot be separated from the host responses. Plants constantly have to adjust catabolic and anabolic auxin pathways acting together with auxin carriers to regulate cellular auxin homeostasis and to respond to developmental and environmental cues (Rosquete et al., 2012). Furthermore, auxin is also in close crosstalk with stress-related hormones, including SA, JA, and ET, which collectively also affect its homeostasis (Naseem et al., 2015; Robert-Seilaniantz et al., 2011; Yang et al., 2019). The defence-related phytohormone SA represses auxin signalling (Wang et al., 2007; Yuan et al., 2017), whereas JA signalling can induce auxin synthesis (Hentrich et al., 2013).

Auxin is of similar importance in plant interactions with bacteria and fungi. For example, *Botrytis cinerea* and *Pseudomonas syringae* induce the accumulation of the conjugated form IAA–Asp in *A. thaliana*, which enhances disease development due to inactivation of auxin (González-Lamothe et al., 2012). In contrast, *Fusarium oxysporum* requires functional auxin signalling and transport to promote disease susceptibility (Kidd et al., 2011). Recent studies support the dual role of auxin during infection, either by enhancing disease susceptibility (Djami-Tchatchou et al., 2020; Fu & Wang, 2011; Mutka et al., 2013) or increasing resistance (Llorente et al., 2008). There is very little evidence whether bacterial and fungal pathogens directly target key regulators of the auxin signalling pathway. To the best of our knowledge, so far, there was only one study demonstrating that the type III effector AvrRpt2 from *P. syringae* stimulates the degradation of the Aux/IAA protein AXR2, which is a negative regulator

in auxin signalling in *A. thaliana* (Cui et al., 2013). The degradation of AXR2 promotes pathogenicity, but it remains to be shown whether AXR2 directly interacts with AvrRpt2.

To sum up, it has become evident that successful virus infection results from compatible interplay between plant viruses and phytohormones, including auxin. Some viruses, such as TMV, RDV, and BNYVV, inactivate negative regulators of auxin signalling, whereas other viruses, such as SRBSDV, RBSDV, RSMV, and RSV, target positive regulators (transcriptional activators) of auxin signalling. Only very recently, it has been found that the P22 protein from tomato chlorosis virus binds to the C-terminal part of SKP1.1 and destabilizes SCF^{TIR1} complex assembly, resulting in suppression of Aux/IAA degradation and promoting virus infection (Liu et al., 2021). This finding adds a new molecular mechanism as the SCF^{TIR1} complex mediating protein degradation via the ubiquitin pathway is targeted by a plant virus to disrupt auxin signalling. As indicated above, transcriptional changes in auxin-responsive genes have also been observed in other plant–virus pathosystems for which a direct interaction between viral proteins and regulators of auxin signalling have not been elucidated yet. Therefore, how viral infections precisely reprogramme and regulate auxin-mediated responses is far from being understood, which represents one of the important future research directions. The main obstacle to finding putative interactions is, on the one hand, the diversity of viral proteins and, on the other hand, the large number of plant proteins involved in auxin signalling, which results in a high number of theoretically possible interactions. This problem can be overcome by comprehensive protein–protein interaction screening. Elucidation of the exact roles of auxin signalling pathways in the host defence response and mechanisms of their subversion by viruses for their own benefit will improve our understanding of plant–virus interactions and assist in the development of novel antiviral strategies, for example, identification of the key residues in the host protein interacting domains for genetic intervention (gene editing, plant breeding). It has been shown for some of the aforementioned viruses that loss of the interaction with the components of auxin signalling correlates with increased host resistance. Engineering recessive resistance using the CRISPR/Cas9 technology to prevent the interaction by modifications of key auxin regulators could be helpful in developing virus control strategies.

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DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analysed.

ORCID

Maximilian Müllender (iD) https://orcid.org/0000-0001-5278-3674

Eugene I. Savenkov (iD) https://orcid.org/0000-0002-5802-5089
Sebastian Liebe (iD) https://orcid.org/0000-0002-4728-9548

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