SCIENCE CHINA PRESS

SCIENCE CHINA Life Sciences
life.scichina.com link.springer.com
https://doi.org/10.1007/s11427-024-2680-3

REVIEW

Crop antiviral defense: Past and future perspective

Zhirui Yang[1,2], Guangyao Li[1,3], Yongliang Zhang[2], Fangfang Li[4], Tao Zhou[5], Jian Ye[6], Xianbing Wang[2], Xiaoming Zhang[7], Zongtao Sun[8], Xiaorong Tao[9], Ming Wu[3], Jianguo Wu[3] & Yi Li[1,3*]

[1]The State Key Laboratory of Protein and Plant Gene Research, School of Life Sciences, Peking University, Beijing 100871, China;
[2]State Key Laboratory of Plant Environmental Resilience, Frontiers Science Center for Molecular Design Breeding, College of Biological Sciences, China Agricultural University, Beijing 100193, China;
[3]State Key Laboratory for Ecological Pest Control of Fujian and Taiwan Crops, Vector-borne Virus Research Center, College of Plant Protection, Fujian Agriculture and Forestry University, Fuzhou 350002, China;
[4]State Key Laboratory for Biology of Plant Diseases and Insect Pests, Institute of Plant Protection, Chinese Academy of Agricultural Sciences, Beijing 100193, China;
[5]State Key Laboratory for Agro-Biotechnology and Department of Plant Pathology, China Agricultural University, Beijing, 100193, China;
[6]State Key Laboratory of Plant Genomics, Institute of Microbiology, Chinese Academy of Sciences, Beijing, 100101, China;
[7]State Key Laboratory of Integrated Management of Pest Insects and Rodents, Institute of Zoology, Chinese Academy of Sciences; CAS Center for Excellence in Biotic Interactions, University of Chinese Academy of Sciences, Beijing 100049, China;
[8]State Key Laboratory for Managing Biotic and Chemical Threats to the Quality and Safety of Agro-products, Key Laboratory of Biotechnology in Plant Protection of MARA and Zhejiang Province, Institute of Plant Virology, Ningbo University, Ningbo 315211, China;
[9]Department of Plant Pathology, The Key Laboratory of Plant Immunity, Nanjing Agricultural University, Nanjing 210095, China

*Corresponding author (email: liyi@pku.edu.cn)

Received 29 April 2024; Accepted 9 July 2024; Published online 15 August 2024

Viral pathogens not only threaten the health and life of humans and animals but also cause enormous crop yield losses and contribute to global food insecurity. To defend against viral pathogens, plants have evolved an intricate immune system to perceive and cope with such attacks. Although most of the fundamental studies were carried out in model plants, more recent research in crops has provided new insights into the antiviral strategies employed by crop plants. We summarize recent advances in understanding the biological roles of cellular receptors, RNA silencing, RNA decay, hormone signaling, autophagy, and ubiquitination in manipulating crop host-mediated antiviral responses. The potential functions of circular RNAs, the rhizosphere microbiome, and the foliar microbiome of crops in plant–virus interactions will be fascinating research directions in the future. These findings will be beneficial for the development of modern crop improvement strategies.

crops | antiviral defense | RNA silencing | autophagy | ubiquitin proteasome system

Introduction

In the future, significant increases in crop yields should be achieved to meet increasing challenges raised by climate change, rapid growth of world's population, and economic development (Li and Wang, 2019; Nicaise, 2014; Ning et al., 2017; Ramirez-Prado et al., 2018). Among the environmental limitations that affect plant growth, viral pathogens cause enormous losses in crop yield and quality and are serious threats to food security (Li et al., 2022a; Wei and Li, 2016; Wu et al., 2022a). As sessile organisms, plants coexist in intimate relationships with dynamic microbial communities in their natural environment. In the long-term arms race between plants and viral pathogens, plants have developed intricate multilayer defenses, including inducible and tightly regulated immune defense systems that mediate interactions with different pathogens including viruses (Calil and Fontes, 2017; Jones and Dangl, 2006). The outcome of these complex multidimensional interactions is one of the determinants of plant survival and fitness. Therefore, there is a great need for future agricultural development to understand how plant defense networks defend against viral pathogens and to improve crops to achieve both biotic stress resistance and high yields from genetics and genomics.

In addition, viruses are excellent entities for elucidating host-microbe interactions because of their simple DNA or RNA genomes and few proteins, and their multiplication and movement are exclusively dependent on host cellular metabolism (García and Pallás, 2015; Wang, 2015). In recent decades, successful viral infection has been shown to overcome complex defense systems comprising multiple layers (Ismayil et al., 2020a; Li and Wang, 2019; de Ronde et al., 2014).

In this review, we summarize the molecular mechanisms underlying antiviral immune responses in crop plants and highlight the latest advances in crop plant defense against viruses. The key aspects covered include but are not limited to intracellular immune receptor-mediated immune responses, RNA silencing or interference, RNA decay, hormone signaling, and autophagy pathways.

Cellular immune receptor-mediated immunity

The dominant resistance (*R*) genes, which have been identified in plant–virus interactions, mainly encode nucleotide binding site-leucine-rich repeat (NBS-LRR)-containing proteins to specifically recognize virus-encoded avirulence (Avr) proteins (Boualem et al., 2016; DeYoung and Innes, 2006; Jones et al., 2024; Nicaise, 2014; de Ronde et al., 2014). Disease resistance mediated by *R* genes typically triggers a robust host response, often culminating in programmed cell death known as the hypersensitive response (HR) (Boualem et al., 2016; Chakraborty et al., 2018). NBS-LRR

proteins can be categorized into three groups according to their N-terminal structure: TIR-NBS-LRR (TNL) with a Toll–interleukin-1 receptor (TIR) domain, CC-NBS-LRR (CNL) with a coiled-coil (CC) domain, or CC_R-NBS-LRR (RNL) with a POWDERY MILDEW 8-like CC (CC_R) domain (Duxbury et al., 2021; de Ronde et al., 2014; Sett et al., 2022). The tobacco *N* gene is the first *NBS-LRR* gene cloned from plants that mediates antiviral defense against tobacco mosaic virus (TMV) (Whitham et al., 1994). Since then, many dominant antiviral *R* genes have been cloned in model plants and crops, primarily in dicot plants (Table 1).

An illustrative example is the potato (*Solanum tuberosum*) *Rx1* gene, which encodes a typical CC-NBS-LRR protein that confers resistance to potato virus X (PVX). The interaction between the CC domain of Rx1 and cellular ranGTPase-activating protein 2 (ranGAP2) is required for Rx1 function. Furthermore, the NB domain of Rx1 is sufficient to elicit an HR (Rairdan et al., 2008). Ry_{sto}, a TNL immune receptor, can recognize the potato virus Y (PVY) coat protein (CP). The central 149-amino acid domain of the PVY CP acts as the core region influencing Ry_{sto}-mediated cell death. Viruses sharing a structural pattern similar to that of PVY CP are suppressed in Ry_{sto} plants. The functionality of Ry_{sto} makes it feasible to achieve durable and broad-spectrum resistance in crops (Grech-Baran et al., 2022; Grech-Baran et al., 2020). *SlSw5a*, an *NLR* gene that confers resistance against tomato leaf curl New Delhi virus (ToLCNDV), has been identified in the ToLCNDV-resistant tomato cultivar H-88-78-1. SlSw5a directly interacts with the AC4 protein encoded by ToLCNDV to trigger HR and induce reactive oxygen species (ROS) generation at infection sites to limit virus spread (Sharma et al., 2021). The pepper (*Capsicum chinense*) NLR Tsw is characterized by an unusually large leucine-rich repeat (LRR) domain resembling hormone receptors such as COI1, TIR1, or MAX2. The tomato spotted wilt orthotospovirus (TSWV) effector nonstructural protein NSs can target COI1, TIR1, or MAX2 by enhancing the interaction of the hormone receptor with TROSINTE BRANCHED/CYCLOIDEA/PCF (TCP) 21 to facilitate viral infection. However, the pepper NLR Tsw can monitor this progress and convert it to a defense against TSWV infection, demonstrating the evolution of countervirulence strategies in plants and making pathogen invasion more challenging (Chen et al., 2023). The tomato *Sw-5b* gene confers resistance to American-type tomato-infecting tospoviruses. The ARC-LRR domain of *Sw-5b* directly interacts with the conserved 21-amino acid region in the viral movement protein NSm (NSm^{21}) (Zhu et al., 2017). The CC domain of Sw-5b directly interacts with a subunit of the mediator complex, Mediator 10b (MED10b), interfering with the interaction between MED10b and MED7, thus derepressing the expression of antiviral defense genes (Wu et al., 2023b). $Tm\text{-}2^2$, a plasma membrane (PM)-localized CNL, confers resistance against TMV (Wang et al., 2020). Recent findings revealed that the CC domain is the signaling domain of $Tm\text{-}2^2$ that requires PM localization and self-association upon TMV infection. The NB-ARC domain of $Tm\text{-}2^2$ is crucial for self-interaction and regulates the activation of the CC domain (Wang et al., 2020). *Rsc4-3*, encoded by the soybean (*Glycine max*) cultivar Dabaima, is a dominant cell wall-localized NLR protein that recognizes soybean mosaic virus (SMV). Knocking down *Rsc4-3* in the resistant cultivar Dabaima weakens resistance to SMV (Yin et al., 2021). A recent study showed that viruses have evolved counterdefensive strategies for more sustainable invasion by suppressing NLR-mediated resistance. BPMV disrupted Rsv3-mediated resistance upon soybean infection by impairing the downstream defense system of the *R* gene (Alazem et al., 2023).

There are numerous reports regarding the function of NLR-mediated resistance against viruses in dicotyledonous plants but few in monocotyledonous plants. *Brachypodium barley stripe resistance 1* (BSR1) is the first cloned NLR virus resistance gene in monocots, shedding light on the antiviral mechanism of crops. The gene-for-gene hypothesis was validated by BSR1 recognizing the triple gene block 1 (TGB1) effector encoded by barley stripe mosaic virus (BSMV) upon viral infection (Wu et al., 2022b). The *CC-NBS-LRR* gene has been finely mapped as the best candidate for *RYMV3*, a locus related to rice resistance against rice yellow mottle virus (RYMV) (Pidon et al., 2017). Recently, the *Ym2* allele, which is associated with resistance against wheat yellow mosaic virus (WYMV), was identified as the *CDS618* gene, which encodes a CC-NBS-LRR protein (Mishina et al., 2023). However, the functions of these *CC-NBS-LRR* genes remain to be further validated. Obviously, there are two open questions. First, although numerous *R* genes from dicotyledonous plants against viruses and from monocotyledonous plants against bacterial and fungal pathogens (Table 1) are reported, few such *R* genes have been identified against different viral pathogens in monocotyledonous crops. Second, although the critical role of the NLR resistosome against bacterial pathogens has been established in plants and mammals (Bi et al., 2021; Duxbury et al., 2021; Wang et al., 2019), the existence of a similar NLR "resistosome" (plasma membrane-localized) in plants against viral effectors inside cells remains to be determined.

Increasing evidence shows that micro(mi)RNA-mediated *R* gene turnover is a protective mechanism utilized by plants to avoid autoimmunity in the absence of pathogens (Jin et al., 2021; Li et al., 2011). A recent study also showed that viral pathogens promote infection via interference with the expression of *R* genes as well as the turnover of *R* genes mediated by miRNA (Cui et al., 2020a). For example, miR1885, a 22-nt miRNA found in *Brassica*, directly targets BraTNL1 for silencing, thereby negatively regulating BraTNL1-mediated antiviral immunity. Under natural conditions, miR1885 is maintained at low levels to support regular development but is induced in response to turnip mosaic virus (TuMV) infection (Cui et al., 2020a). The increased levels of miR1885 subsequently lead to the turnover of the *BraTNL1* and *BraTIR1* genes. Furthermore, miR1885 triggers the production of trans-acting small interfering RNA (tasiRNA)-like phasiRNAs from the *BraTIR1* locus, further contributing to the silencing of *BraCP24*, a photosynthesis-related gene involved in plant growth and developmental regulation. Notably, the suppression of *R* genes by miR1885 is counteracted by the transcriptional upregulation of these *R* genes (Cui et al., 2020a). This work underscores the precise regulation between viruses and host plants.

Recessive resistance

Given that viruses rely on host factors to complete their biological cycle, the absence of appropriate host factors results in recessive resistance to diverse viruses (Wu et al., 2024). Eukaryotic initiation factors (eIFs), belonging to the eIF4E/4G family, play a role in the successful infection of diverse viruses, including *Potyvirus*, *Bymovirus*, *Carmovirus*, *and Fijivirus*. To date, many recessive *R* genes have been identified and cloned in model plants

Table 1. Genetically dominant antiviral *R* genes cloned from crops and model plants

Plant species	*R* gene	Type	Virus targets	Virus genus	Avr	Inoculate methods	References
Arabidopsis thaliana	*HRT*	CC-NB-LRR	TCV (*Turnip crinkle virus*)	*Carmovirus*	CP	*In vitro* transcription	Cooley et al., 2000
	RCY1	CC-NB-LRR	CMV (*Cucumber mosaic virus*)	*Cucumovirus*	CP	Infectious cloned	Takahashi et al., 2001
	RTM1	Jacalin-like	TEV (*Tobacco etch virus*)	*Potyirus*	CP	Artist's airbrush	Chisholm et al., 2000
	JAX1	Jacalin-like	Broad resistance against potexviruses	*Potexvirus*	Unknown	Mechanically inoculated	Bijaisoradat and Kuhn, 1985
Brassica campestris Field mustard	*BcTuR3*	TIR-NBS-LRR	TuMV (*Turnip mosaic virus*)	*Potyvirus*	Unknown	Mechanically inoculated	Ma et al., 2010
Nicotiana tabacum	*N*	TIR-NB-LRR	TMV (*Tobacco mosaic virus*) ToMV (*Tomato mosaic virus*)	Tobamovirus	p50	Mechanically inoculated	Whitham et al., 1994
Pepper (*Capsicum annuum*)	*L-locus: L1, L2, L3, L4*	CC-NB-LRR	*Tobamoviruses*	*Tobamovirus*	CP	Mechanically inoculated	Tomita et al., 2011
Tomato (*Solanum peruvianum*)	*Sw5b*	CC-NB-LRR	TSWV (*Tomato spotted wilt virus*) and other Tospoviruseses	*Tospovirus*	NSm	Mechanical inoculations	Spassova et al., 2001
	Tm-2	CC-NB-LRR	TMV (*Tobacco mosaic virus*) and other Tobamoviruseses	*Tobamovirus*	30 kD MP	Mechanical inoculations	Meshi et al., 1989
	Tm-2²	CC-NB-LRR	TMV and other Tobamoviruses	*Tobamovirus*	30 kD MP	Mechanical inoculations	Lanfermeijer et al., 2005
Pepper (*Capsicum chinense*)	*Tsw*	CC-NB-LRR	TSMV (*Tomato spotted wilt virus*)	*Tospovirus*	NSs	Mechanically inoculated	Chen et al., 2023
Tomato (*Solanum lycopersicum*)	*Ty-2*	CC-NBS-LRR	TYLCV (*Tomato yellow leaf curl virus*)	*Begomovirus*	Rep/C1	Agrobacterium-mediated inoculation	Shen et al., 2020
Tomato (*Solanum chilense*)	*Ty-1*	RDR	TYLCV	*Begomovirus*	Unknown	Whitefly mediated inoculation; Infectious TYLCV-IL clone	Verlaan et al., 2013
Tomato (*Solanum lycopersicum*)	*Sw5a*	CC-NBS-LRR	ToLCNDV (*Tomato leaf curl New Delhi virus*)	*Geminiviridae*	AC4	Agroinoculation	Sharma et al., 2021
Tomato (*Solanum hirsutum*)	*Tm-1*	TIM-barrel-like domain protein	ToMV (*Tomato mosaic virus*)	Tobamovirus	Replicase Helicase domain	Mechanically inoculated	Ishibashi et al., 2007
Potato (*Solanum tuberosum*)	*Rx1*	CC-NB-LRR	PVX (*Potato virus X*) and other Potex viruses	*Potexvirus*	CP	Mechanically inoculated	Baurès et al., 2008
	Rx2	CC-NB-LRR	PVX (*Potato virus X*)	*Potexvirus*	CP	Mechanically inoculated	Bendahmane et al., 2000
	Y-1	TIR-NB-LRR	PVY (*Potato virus Y*)	*Potyvirus*	Unknown	Sap was rubbed	Vidal et al., 2002
	Ry_{sto}	TIR-NB-LRR	PVY (*Potato virus Y*)	*Potyvirus*	CP	Sap was rubbed	Grech-Baran et al., 2022
Soybean (*Glycine max*)	*Rsv1*	CC-NB-LRR	SMV (*Soybean mosaic virus*)	*Potyvirus*	P3 and HC-Pro	Infectious cDNA clones	Wen et al., 2013
	Rvs4	dsRNase	SMV (*Soybean mosaic virus*)	*Potyvirus*	P3 and NIb	Rubbed softly	Ishibashi et al., 2019
	Rsc4-3	CC-NB-LRR	SMV (*Soybean mosaic virus*)	*Potyvirus*	Cylindrical inclusion (CI)	Rubbed softly	Yin et al., 2021
Kidney bean (*Phaseolus vulgaris*)	*I*	TIR-NB-LRR	BCMV (*Bean common mosaic virus*) and other Potyviruses	*Potyvirus*	Unknown	Mechanically inoculated	Vallejos et al., 2006
Phaseolus vulgaris	*PvVTT1*	TIR-NB-LRR	BDMV (*Bean dwarf mosaic virus*)	*Begomovirus*	BV1 (NSP)	Infectious cloned	Seo et al., 2007
	PvRT4-4	TIR-NB-LRR	CMV (*Cucumber mosaic virus*)	*Cucumovirus*	2a	Sap inoculation	Seo et al., 2006
Rice (*Oryza sativa*)	*STV11*	sulphotransferase	RSV (*Rice stripe virus*)	Tenuivirus	Unknown	Viruliferous planthoppers	Wang et al., 2014
Maize (*Zea mays*)	*ZmABP1*	Auxin binding protein 1	SCMV (*Sugarcane mosaic virus*) and other potyviruses	*Potyvirus*	Unknown	Virus-containing sap	Ruffel et al., 2005
	ZmTrxh	Thioredoxin	SCMV (*Sugarcane mosaic virus*)	*Potyvirus*	Unknown	Virus-containing sap	Liu et al., 2017
Brachypodium sylvaticum	*BSR1*	CC-NBS-LRR	BSMV (*Barley stripe mosaic virus*)	Hordeivirus	Unknown	*Agrobacterium*-mediated inoculation	Wu et al., 2022b

and crops (Table 2), suggesting a broad mechanism of virus infection. Mutations or silencing of *eIF4E*, *eIF4E isoform* (*eIF(iso)4E*), or *eIF4G* mediate resistance against viruses in tomato, soybean, pepper, rice, barley, and wheat (Albar et al., 2006; Gao et al., 2020b; Gao et al., 2004; Kan et al., 2023; Stein et al., 2005; Wang et al., 2021a; Yang et al., 2013b). In addition to eIFs, other crop host proteins also contribute to recessive resistance to viral pathogens. For example, the natural variant of *protein disulfide isomerase like 5-1* (*HvPDIL5-1*), which was identified in barley at the recessive *resistance to yellow mosaic*

Table 2. Antiviral recessive *R* genes cloned from crops and model plants

Plant species	*R* gene	Type	Virus targets	Virus genus	Avr	Inoculate methods	References
Capsicum annuum	*pvr1*/*pvr2*	eIF4E	PVY; TEV (*Tobacco etch virus*)	*Potyvirus*	VPg	Mechanically inoculated	Kang et al., 2005
	pvr2 and *pvr6*	eIF(iso)4E	PVMV (*Pepper veinal mottle virus*)	*Potyvirus*	Unknown	Rubbed manually	Ruffel et al., 2006
Solanum lycopersicum	*pot-1*	eIF4E	PVY; TEV (*Tobacco etch virus*)	*Potyviruse*	VPg	Mechanically inoculated	Ruffel et al., 2005
Pisum sativum	*cyv1*; *cyv2*	eIF4E	ClYVV (*Clover yellow vein virus*)	*Potyvirus*	Unknown	Infectious cDNA	Andrade et al., 2009
	sbm2	eIF4E	PSbMV (*Pea seed-borne mosaic virus*)	*Potyvirus*	Unknown	Infectious clones	Gao et al., 2004
	sbm1	eIF4E	PSbMV (*Pea seed-borne mosaic virus*); BYMV (*Bean yellow mosaic virus*)	*Potyvirus*	Unknown	Infectious clones	
Phaseolus vulgaris	*bc-3*	eIF4E	BCMV (*Bean common mosaic virus*)	*Potyvirus*	Unknown	Mechanically inoculated	Naderpour et al., 2010
Lactuca sativa	$mo1^1$/$mo1^2$	eIF4E	LMV (*Lettuce mosaic virus*)	*Potyvirus*	Unknown	Rub inoculate	Nicaise et al., 2003
Cucumis melo	*Nsv*	eIF4E	MNSV (*Melon necrotic spot virus*)	*Carmovirus*	Unknown	Infectious clones	Nieto et al., 2006
Rice (*Oryza sativa*)	*rymv1*	eIF(iso)4G	RYMV (*Rice yellow mottle virus*)	*Sobemovirus*	Unknown	Mechanically inoculated	Albar et al., 2006
Hordeum vulgare	*rym7*	eIF4E	BaYMV (*Barley yellow mosaic virus*)	*Bymovirus*	Unknown	Leaf-sap	Yang et al., 2013b
	rym4/*5*	eIF4E	BaMMV (*Barley mild mosaic virus*); BaYMV (*Barley yellow mosaic virus*)	*Bymovirus*	Unknown	Mechanically inoculated	Stein et al., 2005
Triticum aestivum	*eIF4E*	eIF4E	WYMV (*Wheat yellow mosaic virus*); CWMV (*Chinese wheat mosaic virus*)	*Bymovirus*	Unknown	Mechanically inoculated	Kan et al., 2023
Hordeum vulgare	*rym11*	PDI	BaMMV; BaYMV	*Bymovirus*	Unknown	Mechanically inoculated	Yang et al., 2014
Maize (*Zea mays*)	*qMrdd1*	RabGDIα	RBSDV (*Rice black-steaked virus*)	Fijivirus	P7	Viruliferous planthoppers	Liu et al., 2020
Solanum lycopersicum	*Ty-5*	Poleta	TYLCV	*Begomovirus*	Unknown	Infectious clones	Ren et al., 2022
			BCTV (*Beet curly top virus*)	*Curtovirus*	Unknown	Infectious clones	

disease 11 (*rym11*) resistance locus, causes naturally occurring resistance to multiple strains of *Bymoviruses* (Yang et al., 2014). More recently, the *qMrdd1* locus, which confers maize host resistance to rice black-streaked dwarf virus (RBSDV), was finely mapped to the candidate gene *Rab GDP dissociation inhibitor alpha* (*RabGDIα*) (Liu et al., 2020). Natural helitron insertion impairs the interaction between RBSDV-encoded P7-1 and RabGDIα, thus decreasing the ability of P7-1 to recruit RabGDIα-hel, leading to recessive resistance to RBSDV (Liu et al., 2020). *StEXA1*, an ortholog of the susceptibility gene in *Arabidopsis* named *Essential for PoteXvirus Accumulation 1* (*EXA1*), encodes a glycine-tyrosine-phenylalanine (GYF) domain-containing protein. *StEXA1* knockdown reduces the accumulation of PVY in potato (Chen et al., 2022). These findings provide more potential targets for crop viral disease resistance breeding.

RNA silencing

RNA interference (RNAi) or RNA silencing-based antiviral defense is one of the most evolutionarily conserved primary defense mechanisms in plants (Chen and Ding, 2020a; Islam et al., 2018; Jin et al., 2021; Li and Wang, 2019; Yang and Li, 2018; Zhu and Guo, 2012). Over the past few decades, many efforts have been made to dissect the roles of antiviral RNAi in the interactions between plant hosts and viruses, particularly in model plants such as *Arabidopsis thaliana*. In general, antiviral RNA silencing progresses through three steps: initiation, amplification, and signaling spreading. Antiviral RNA silencing is triggered by the recognition of viral double-stranded RNAs by plant dicer-like (DCL) proteins (Guo et al., 2019; Jin et al., 2021; Li and Wang, 2019; Yang and Li, 2018). DCL proteins process dsRNA into virus-derived small interfering RNAs (vsiRNAs). These vsiRNAs are then recruited by plant ARGONAUTE (AGO) proteins to form RNA-induced silencing complexes (RISCs), which subsequently target the same or another sequence of complementary viral RNAs (Guo et al., 2019; Li and Wang, 2019; Yang and Li, 2018). The amplification stage involves the activity of RNA-dependent RNA polymerases (RDRs), which utilize single-stranded (ss) RNA to synthesize long, perfect double-stranded (ds) RNA. These dsRNAs serve as substrates for the DCL-dependent formation of secondary vsiRNAs. This second pool of vsiRNAs contributes to systemic silencing signal transduction (Guo et al., 2019; Jin et al., 2021; Li and Wang, 2019; Yang and Li, 2018). Furthermore, virus infection is often accompanied by wounding caused by insect feeding or mechanical damage, which activates RNAi-related gene expression through calcium signaling in model plants (Wang et al., 2021b).

Crop plants possess more complex genomes and encode a greater number of components involved in the RNA silencing pathway than the model plant *Arabidopsis thaliana* (Table 3) (Akond et al., 2020; Kapoor et al., 2008; Liu et al., 2014b; Qian et al., 2011). Recent research in crop plants has made significant strides in understanding antiviral RNAi, especially concerning the regulation of core components (Figure 1, Table 4) (Urayama et al., 2010; Wang et al., 2016; Wu et al., 2017b; Wu et al., 2015; Yang et al., 2020; Yang and Li, 2018; Yao et al., 2019; Zhang et al., 2016).

The rice genome encodes eight DCL proteins, among which OsDCL2 has been found to positively affect *Oryza sativa* endornavirus (OsEV) maintenance (Urayama et al., 2010). Notably, single-knockdown rice mutants, including *dcl1*, *dcl2*, *dcl3a*, *dcl3b*, or *dcl4*, are more sensitive to rice stripe virus (RSV) infection than wild-type rice plants (Yang and Li, 2018). Further research is needed to elucidate the unique molecular mechanisms associated with different DCL proteins.

The AGO18 subclade has been exclusively identified in grass genomes. Previous studies have demonstrated that rice AGO18 is highly induced by infection with different viruses and functions as a miRNA sequester to enhance rice antiviral defense (Wu et al., 2015). OsAGO18 can compete with miR168 and miR528,

Table 3. Comparison of RNAi pathway components (AGO, RDR, DICER) in crops with those in the model plants *Arabidopsis* and tobacco

Plant species	AGO	RDR	DCL	References
Arabidopsis thaliana	10	6	4	Fang and Qi, 2016
Tobacco (*Nicotiana benthamiana*)	9	3	4	Nakasugi et al., 2013
Soybean (*Glycine max*)	21	7	7	Liu et al., 2014b
Rice (*Oryza sativa*)	19	5	8	Kapoor et al., 2008
Maize (*Zea mays*)	18	5	5	Qian et al., 2011
Wheat (*Triticum aestivum L.*)	39	16	7	Akond et al., 2020
Sorghum (*Sorghum vulgare*)	14	7	3	Liu et al., 2014b
Tomato (*Solanum lycopersicum*)	15	6	7	Bai et al., 2012
Barley (*Hordeum vulgare*)	11	7	5	Hamar et al., 2020
Pepper (*Capsicum Annuum L.*)	12	6	4	Qin et al., 2018
Brassica.napus (*Brassica* species)	27	16	8	Cao et al., 2016

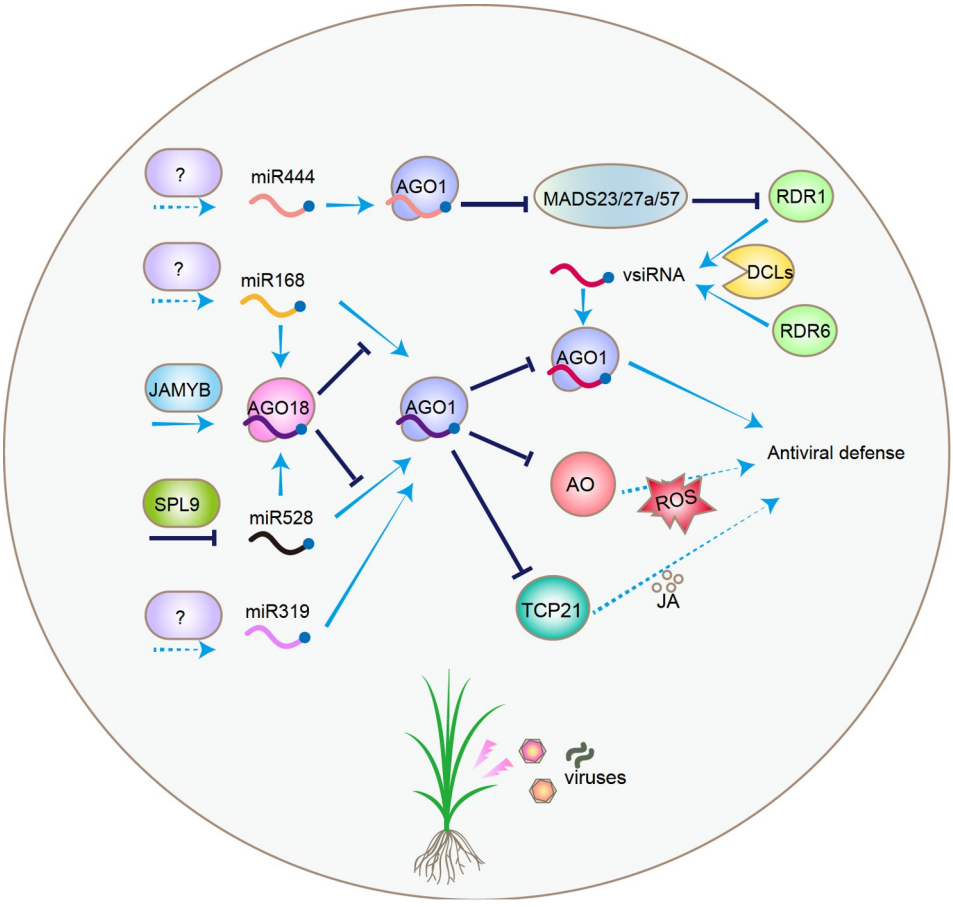

Figure 1. RNAi-mediated antiviral defense signaling in rice. During viral infection, RNA-silencing components are transcriptionally regulated by different transcription factors. JAMYB transcriptionally activates AGO18 expression. AGO18 further sequesters miR168 and miR528, subsequently releasing AGO1 and AO, respectively. MiR444 targets the MADS23/27a/57 transcription factors to upregulate RDR1. RDR1 and RDR6 provide additional substate double-stranded RNAs (dsRNAs) for DCLs to generate vsiRNAs. AGO1 recruits vsiRNAs to repress virus infection. AO mediates antiviral defense by increasing ROS accumulation. MiR319 targets TCP21 to inhibit JA signaling, thus repressing rice antiviral defense.

thereby releasing their targets, *OsAGO1* and *L-ascorbic acid oxidase* (*AO*), respectively. OsAGO1 further orchestrates antiviral defense through the vsiRNA pathway (Wu et al., 2015). A more recent study revealed that the transcription of *OsAGO18*, the core component of rice antiviral RNAi, is positively regulated by the jasmonic acid (JA)-responsive transcription factor JAMYB upon viral infection (Yang et al., 2020). Notably, the overexpression of virus CP activates the JA signaling pathway and upregulates *OsAGO18* expression through JAMYB (Yang et al., 2020). This finding marks the first instance of synergistic regulation of a core component of antiviral RNAi through a primary plant hormone.

Among the six *RDR* genes in *Arabidopsis*, the functions of RDR1, RDR2, and RDR6 have been well demonstrated (Garcia-Ruiz et al., 2010; Wang et al., 2010). In the rice genome, five

Table 4. RNAi components with antiviral activity

RNAi component	Virus	Host	References
AGO1	RDV/RSV	*Oryza sativa*	Wu et al., 2015
AGO18	RDV/RSV	*Oryza sativa*	Wu et al., 2015
RDR1	RSV	*Oryza sativa*	Wang et al., 2016
RDR6	RDV/RSV	*Oryza sativa*	Hong et al., 2015; Jiang et al., 2012
OsDCL2	dsRNA virus *Oryza sativa* endornavirus (OsEV)	*Oryza sativa*	Urayama et al., 2010
RNase P	TMV/CMV	*Arabidopsis thaliana*	Gobert et al., 2021

RDR genes have been annotated. RDR1 orthologs confer basal resistance to several viruses by participating in the biogenesis of virus-derived secondary siRNAs (Diaz-Pendon et al., 2007; Garcia-Ruiz et al., 2010; Qi et al., 2009; Wang et al., 2010). The expression levels of both *RDR1* in *Arabidopsis* and *NtRDR1*, the ortholog of AtRDR1 in *Nicotiana tabacum*, have been shown to be induced by viral infection or salicylic acid (SA) treatment (Xie et al., 2001; Yu et al., 2003). In *Arabidopsis*, RDR1 is known to be responsible for the accumulation of a distinct class of virus-activated host siRNAs (vasiRNAs), which directly suppress host genes to confer broad-spectrum antiviral activity (Cao et al., 2014). Interestingly, in *Nicotiana benthamiana* (*N. benthamiana*), NtRDR1 has been reported to play dual roles during viral infection. On the one hand, NtRDR1 mediates RNA silencing-based antiviral defense elicited by SA, on the other hand, it enhances viral infection through suppressing RDR6-mediated antiviral defense (Ying et al., 2010). These findings reflect the complicated functions of antiviral RNAi components during plant-virus interactions. Rice plants with the Tos17 insertion *rdr1* exhibit increased sensitivity to RSV infection, indicating that rice RDR1 plays important roles in rice virus resistance (Wang et al., 2016). Further studies are required to address whether OsRDR1 exerts antiviral effects in a manner similar to that of the RDR1 ortholog in *Arabidopsis* or whether it has different functions in antiviral defense. Rice RDR6 has also been reported to confer resistance to both RSV and rice dwarf virus (RDV) (Hong et al., 2015; Jiang et al., 2012). Since a deficiency in OsRDR6 could cause a severe developmental phenotype, RNAi knockdown mutants were used for functional investigations. The abundance of vsiRNAs significantly decreased in virus-infected *OsRDR6* knockdown rice plants compared to wild-type rice plants (Jiang et al., 2012), indicating that OsRDR6 participated in amplifying viral siRNAs upon viral infection.

In addition to vsiRNA-mediated antiviral RNAi, increasing evidence suggests that miRNAs are involved in regulating plant antiviral immunity (Wang et al., 2016; Wu et al., 2017b; Wu et al., 2015; Zhang et al., 2016). Previous studies have revealed that monocot-specific miR444 can be induced by RSV infection, thereby upregulating *OsRDR1* expression by diminishing the repressive effects of MIKCC-type MADS box transcription factors (OsMADS23, OsMADS27a and OsMADS57) on *OsRDR1* (Wang et al., 2016). Another monocot-specific miRNA, miR528, has been shown to negatively regulate rice antiviral defense against RSV by downregulating the target *AO* gene (Wu et al., 2017b). A recent study revealed that rice SQUAMOSA PROMOTER BINDING PROTEIN-LIKE 19 (OsSPL9) is responsible for the down-regulation of miR528 during RSV infection (Yao et al., 2019). OsSPL9 displays high-affinity binding to specific "GTAC" motifs within the promoter region of miR528, activating the expression of the miR528 gene *in vivo*. Loss-of-function mutations in OsSPL9 caused a significant reduction in miR528 accumulation, enhancing plant resistance to RSV (Yang et al., 2019b; Yao et al., 2019). This study identified a new regulatory layer to the miR528-AO antiviral defense pathway. Another study showed that infection with rice ragged stunt virus (RRSV) and RBSDV induces the accumulation of miR319 in rice and wheat (*Triticum aestivum*), respectively (Zhang et al., 2016). Overexpression of miR319 in rice downregulates the expression of *TCP21*, a target of miR319, resulting in a significantly greater susceptibility to RRSV than in wild-type plants (Zhang et al., 2016). The RNAi-mediated antiviral pathway plays a fundamental role in plant–virus interactions, and resolving the antiviral-RISC structure in crops could be an interesting avenue for future research.

To overcome the host immune response, plant viruses have evolved to express a variety of viral suppressors of RNA silencing (VSRs) aimed at suppressing host antiviral RNA silencing (Cao et al., 2005; Díaz-Pendón and Ding, 2008; Gao et al., 2020a; Guo et al., 2019; Li and Wang, 2019; Ren et al., 2010; Wu et al., 2017a). VSRs are multifunctional factors that target various steps in the antiviral RNA silencing pathway, including blocking vsiRNA biogenesis, preventing AGO-vsiRNA silencing complex formation and suppressing the spread of silencing signals (Gao et al., 2020a; Guo et al., 2019; Li and Wang, 2019; Ren et al., 2010).

RNA decay

In addition to RNA silencing, RNA decay or exonucleolytic RNA turnover has emerged as another crucial strategy for antiviral immunity in plants (Cui et al., 2020a; Garcia et al., 2014; Jin et al., 2021; Li and Wang, 2019; Li and Wang, 2018). The RNA decay machinery utilizes exonucleases to degrade aberrant RNAs, initiating the decay process through deadenylation, followed by exosome complex-mediated 3′–5′ cleavage or decapping and subsequent exoribonuclease (XRN4)-mediated 5′–3′ decay (Stoecklin and Mühlemann, 2013; Zhang et al., 2015; Zhang and Guo, 2017). RNA decay and RNA silencing appear to synergistically inhibit viral infection, and their activities partially overlap. A recent study showed that key cytoplasmic 5′-3′ RNA decay pathway gene-encoding proteins (5′RDGs), such as DCP1, DCP2, XRN4, and PARN, mediate the suppression of viral RNA accumulation via the RNA decay pathway when RNA silencing is compromised upon TuMV infection (Li and Wang, 2018). To counter this defense, VPg and HC-Pro, two known VSRs of TuMV, bind to DCP2 and XRN4, respectively, suppressing their antiviral functions (Li and Wang, 2018). These findings suggest that VSRs target not only antiviral RNAi but also RNA decay to ensure successful infection.

Endogenous mRNAs targeted for 5′ decay are typically deadenylated by the carbon catabolite repression (CCR4)-NOT

complex (Zhang and Guo, 2017). Furthermore, RNA quality control systems, which are coupled with RNA decay, primarily include nonsense-mediated decay (NMD), nonstop decay (NSD), and no-go decay (NGD) in plants; these systems have also been revealed to play pivotal roles in limiting virus infection in plants (Ge et al., 2024; Ge et al., 2023; He et al., 2024). A recent study demonstrated that the barley yellow striate mosaic virus (BYSMV) P protein, which is essential for viral genomic RNA replication and mRNA transcription, recruits barley (*Hordeum vulgare*) CCR4 (HvCCR4) from processing bodies (PBs) into viroplasm-like bodies through direct interactions (Zhang et al., 2020c). The deadenylase activity of HvCCR4 enhances BYSMV minigenome RNA replication, promoting virus infection (Zhang et al., 2020c). This finding suggests that viruses can exploit the host RNA decay machinery to facilitate *cytorhabdovirus* replication in plants. In contrast, the host NMD machinery recognizes the viral genomic region of pea mosaic virus 2 (PEMV2), PVX, and turnip crinkle virus (TCV) and degrades its transcripts. To counter this response, the movement protein p26 of PEMV2 and the VSR CP of TCV protect viral transcripts from NMD (May et al., 2020; Wu et al., 2023b).

Interestingly, although increasing evidence shows that RNA decay also plays important roles in antiviral immunity, several studies have shown that some components of the RNA decay pathway counteract endogenous RNAi by competing for similar RNA substrates (Liu and Chen, 2016; Zhang et al., 2015). RISC-cleavage products serve as substrates for RDR for mediating amplification of silencing signal, or are degraded by RNA decay. In plants, generally, the vast majority of coding genes do not produce secondary siRNA, while in the defective mutants of XRN4-mediated 5′-3′ and the exosome complex-mediated 3′-5′ RNA decay pathways, RDR6-dependent 21–22 nt siRNAs from endogenous miRNA targets are detected (Zhang et al., 2015; Zhang and Guo, 2017). The accumulation of these inappropriate silencing signals may result in detrimental consequences. These studies indicate that the repression of endogenous RNAi by RNA decay may be a supervision strategy to protect plant cells from inappropriate silencing signals (Li and Wang, 2019; Szádeczky-Kardoss et al., 2018). It seems that plants have evolved surveillance strategies to distinguish foreign RNAs, like viral RNAs and transgenic RNAs, from endogenous mRNAs, and direct the RNAi and RNA decay pathways to degrade the target foreign RNAs in a hierarchical and coordinated manner.

Notably, most components of antiviral RNAi are induced during various viral infections, and it remains unclear whether the key players in the RNA decay pathway are similarly regulated.

Hormone signaling pathway

The intricate network of plant hormones plays a crucial role in reinforcing the defense system of plants in response to attacks from various pathogens (Alazem and Lin, 2017; Huot et al., 2014; Ning et al., 2017; Pasin et al., 2020; Robert-Seilaniantz et al., 2011; Zhang et al., 2017; Zhao and Li, 2021). Plant hormones, small endogenous signaling molecules, include auxin (indole-3-acetic acid, IAA), ethylene (ET), gibberellin (GA), cytokinin (CK), abscisic acid (ABA), salicylic acid (SA), jasmonic acid (JA), and brassinosteroids (BRs). The importance of crosstalk among different plant hormones and other signaling pathways in regulating plant immune responses and growth has attracted extensive attention (Chen et al., 2023; Leon-Reyes et al., 2010; Wu et al., 2023b; Xia et al., 2015; Yang et al., 2013a).

Auxin plays critical roles in regulating plant growth and developmental processes (Gallei et al., 2020; Leyser, 2018; Li et al., 2021a). Increasing evidence indicates that auxin signaling is involved in the regulation of interactions between viruses and plant hosts (Table 5) (Jin et al., 2016; Qin et al., 2020; Yang et al., 2013a). Recent studies have further revealed the underlying mechanisms of the auxin signaling-mediated antiviral immune response (Figure 2) (Jin et al., 2016; Qin et al., 2020; Zhang et al., 2020b). Auxin signaling is initiated by the perception of auxin via a coreceptor complex comprising an auxin/indole-3-acetic acid (Aux/IAA) protein and an F-box transport inhibitor response 1/auxin signaling F-box (TIR1/AFB) protein. The binding of auxin to the coreceptor complex triggers the ubiquitination and 26S proteasome degradation of Aux/IAA proteins, releasing downstream auxin response factors (ARFs) to initiate the expression of downstream auxin-responsive genes (Guilfoyle and Hagen, 2007; Leyser, 2018; Weijers and Wagner, 2016). Exogenous auxin treatment enhances the rice defense response against RDV. Consistently, knockdown of the auxin signaling suppressor *OsIAA10* increased rice resistance to RDV infection (Jin et al., 2016; Qin et al., 2020). Furthermore, OsARF12, an OsARF that interacts with OsIAA10, promotes rice antiviral defense by regulating the expression of *OsWRKY13* (Qin et al., 2020). This finding provides a working model of the auxin-IAA-ARF-mediated signaling mechanism employed by plants for defense. Interestingly, different ARFs exhibit distinct functions during virus-plant host interactions. Loss-of-function mutants of *osarf5*, *osarf12*, and *osarf16* exhibit reduced resistance, whereas the *osarf11* mutant displays enhanced resistance to RDV (Qin et al., 2020). As an auxin core transcription factor, OsARF17 also confers broad-spectrum resistance to different viruses, including RSV, RBSDV, and rice stripe mosaic virus (RSMV) (Zhang et al., 2020b). Moreover, OsARF17 is a common target of these viruses, demonstrating that manipulation of auxin signaling by viral proteins is a conserved strategy for the pathogenicity of plant RNA viruses (Zhang et al., 2020b). A recent study showed that the NSs protein encoded by TSMV could target the receptor of the auxin pathway to interfere with auxin signaling, promoting virus infection (Chen et al., 2023). This finding reveals a counter-defense mechanism for successful viral infection.

GA plays a pivotal role in regulating plant development and has also been identified as a participant in plant–virus interactions (Huang et al., 2018; Zhao and Li, 2021; Zhu et al., 2005). GA biosynthesis is inhibited by RDV or RBSDV infection (Figure 2, Table 5) (Huang et al., 2018; Zhu et al., 2005), and the application of GA has been shown to alleviate RDV-induced symptom development (Zhu et al., 2005). Further study revealed that the P2 protein encoded by RDV interacts with ent-Kaurene Oxidases (KOs) (Zhu et al., 2005), which catalyze one step of GA biosynthesis. Another study showed that viral proteins, such as southern rice black-streaked dwarf virus (SRBSDV) SP8, RSV P2, and RSMV M, interact with the DELLA protein SLR1 and enhance its degradation. Viral infection enhances the interaction between SLR1 and the GA receptor OsGID1, simultaneously restricting SLR1 function and thereby facilitating viral infection (Li et al., 2022b). The underlying mechanism of GA in crop plant antiviral defense and the crosstalk between GA and auxin signaling pathways in regulating the tradeoff between growth and defense during plant virus infection require additional in-

depth studies.

In addition to its role in regulating plant growth and development, ET plays a versatile role in the biotic stress response (Alazem and Lin, 2015; Broekgaarden et al., 2015; Cui et al., 2020b; Yang et al., 2013a). The application of 1-aminocyclopropane-1-carboxylic acid (ACC), a precursor of ET, promotes the accumulation of crucifer-infecting tobacco mosaic virus (TMV-cg), SRBSDV, and RDV in infected plants (Chen et al., 2013; Zhao et al., 2017). In rice, ET signaling is perturbed and hijacked by RDV and SRBSDV to favor better virus infection (Zhao et al., 2017). The RDV Pns11 protein enhances the ET signaling by interacting with S-adenosyl-L-methionine synthetase 1 (OsSAMS1), enhancing its enzymatic activity and leading to increases in the production of SAM, ACC, and ET (Figure 2 and Table 5) (Zhao et al., 2017). In early infection, the P6 protein encoded by SRBSDV can interact with a negative regulator of the ethylene response, OsRTH2, in the cytoplasm, activating ET signaling and promoting SRBSDV invasion. At the late stage, P6 interacts with the nuclear protein OsEIL2, suppressing ethylene signaling and subsequently attracting planthoppers to facilitate viral transmission (Zhao et al., 2022). However, the underlying mechanism(s) by which ET affects host defenses and counter-defenses in crops are not well understood and need further research.

SA plays an essential role in regulating plant development and immune defense. It has been demonstrated that SA regulates plant antiviral response through modulating multiple defense pathways, such as activating effector-triggered immunity (ETI) and systemic acquired resistance (SAR), promoting antiviral RNAi, inducing inhibition of respiration, and directly targeting GAPDH, a host factor that is required for tomato bushy stunt virus (TBSV) replication (Corina Vlot et al., 2009; Gong et al., 2023; Huot et al., 2014; Murphy et al., 2020; Tian et al., 2015). A previous study using molecular marker-assisted selection or genetic engineering identified a resistant allele of rice *STV11* (*STV11-R*), which encodes a sulfotransferase (OsSOT1) that catalyzes the conversion of SA to sulfonated SA (SSA) (Figure 2) (Wang et al., 2014). This work showed that both SA and SSA treatment could inhibit RSV replication. However, SSA was more effective than SA in conferring RSV resistance and inhibiting viral replication. Conversely, the SA levels in KK34 and transgenic plants expressing *STV11-R* were much greater than those in rice plants expressing *STV11-S*, indicating that SSA may trigger increased biosynthesis of SA through a positive feedback mechanism upon RSV infection (Wang et al., 2014). SA accumulation is significantly increased in sugarcane mosaic virus (SCMV)-infected maize plants (Yuan et al., 2019). Exogenous SA treatment inhibits SCMV accumulation and enhances maize antiviral defense. Further research demonstrated that maize phenylalanine ammonia-lyases (ZmPALs) are required for the SA-mediated immune response to SCMV (Figure 2) (Yuan et al., 2019). In addition, SA signaling appears to be linked to the RNA silencing pathway. Studies in *Arabidopsis* and tobacco indicate that SA accumulation in virus-infected plants might prime RNA silencing by enhancing the expression of RNA-silencing components such as RDRs and AGOs (Alamillo et al., 2006; Alazem et al., 2019). Recent work has shown that SA and RNA-silencing components synergistically mediate antiviral immunity in plant stem cells. In *Arabidopsis thaliana*, TuMV infection increased salicylic acid accumulation, activating the expression of RDR1 and subsequently promoting the RNAi-mediated broad-spectrum antiviral pathway, thereby protecting stem cells from pathogenic viruses (Incarbone et al., 2023). Importantly, SA can be converted into methyl-salicylate (MeSA), a volatile organic compound (VOC) that acts as a signaling molecule in plants. Plants that attacked by aphids subsequently release volatile compounds to elicit airborne defense (AD) in neighbouring plants. The mechanism underlying MeSA-elicited AD has been revealed recently (Gong et al., 2023). Aphids are horticultural pests that transmit 40% of plant viruses to infect plants and cause severe yield loss of crops. Upon aphid feeding, SA levels are elevated in plants, subsequently activating a NAC transcription factor, NAC2, which directly binds to the promoter and promotes the expression of *salicylic acid-carboxylmethyltrans-*

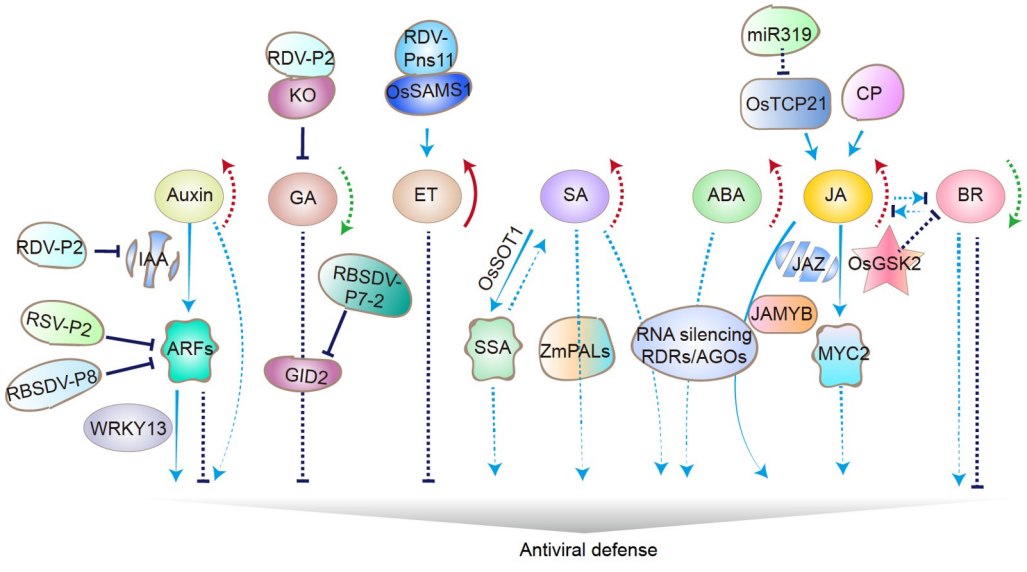

Figure 2. Role of hormone signaling during crop plant–virus interactions. Recent advances in dissecting the roles of hormones in crop antiviral immunity. The red upward arrows indicate "induction" upon viral infection. The green downward arrows indicate "reduction" upon viral infection. RDV, rice dwarf virus; RSV, rice stripe virus; RBSDV, rice black-steaked dwarf virus.

https://doi.org/10.1007/s11427-024-2680-3

ferase-1 (*SAMT1*), catalyzing the conversion of SA to MeSA. The airborne MeSA further induces neighbouring plants anti-aphid immune response leading to inhibition of virus transmission (Gong et al., 2023). To counterdefenses, viral protein CMV 1a interacts with and destabilizes NAC2 to suppress MeSA accumulation (Gong et al., 2023). This finding uncovered the complicated tripartite interaction of plant-insect vector-virus, and provided new strategies to control viruses and insect vectors. Future studies are likely to yield important insights into the crosstalk of SA and other signaling pathways in crop-virus-insect vector-environment interactions.

ABA is also involved in modulating plant resistance against viruses (Alazem et al., 2017; Pasin et al., 2020; Robert-Seilaniantz et al., 2011). Studies in *Arabidopsis* and tobacco have suggested that ABA mediates defense through multiple pathways, such as indirectly inducing the accumulation of callose (β-1,3-glucan) at plasmodesmata and activating the RNA silencing pathway (Alazem et al., 2017; Alazem and Lin, 2020; Pasin et al., 2020). An earlier study in rice cells showed that ABA treatment could induce *OsRDR3a* and *OsRDR6* expression (Figure 2) (Yang et al., 2008). However, only OsRDR6 was responsible for the observed ABA-mediated posttranscriptional control of gene silencing (Yang et al., 2008). Further research is needed to elucidate the detailed mechanisms underlying ABA-mediated antiviral defense in crops.

JA and its derivatives constitute a class of oxygenated lipid-based hormones and play critical roles in plant development and responses to abiotic or biotic stresses (Wasternack and Hause, 2013; Yang et al., 2019a; Zhou et al., 2019). A previous study showed that RRSV and RBSDV infection induce the accumulation of JA in rice and wheat, respectively (Zhang et al., 2016). This induction of JA biosynthesis occurs due to the repression of *TCP21* expression by miR319 (Figure 2) (Zhang et al., 2016). Recently, an RBSDV resistance gene named *ZmGLK36*, which encodes a G2-like transcription factor that inhibits virus infection by enhancing JA biosynthesis and JA-mediated defense responses, was identified in maize (Xu et al., 2023). Another study showed that transcriptional repressors encoded by rice viruses in the genera *Fijivirus*, *Tenuivirus*, and *Cytorhabdovirus* can attenuate the JA pathway to promote virus infection (Li et al., 2021b). These transcriptional repressors suppress the formation of the OsMED25-OsMYC complex, thus inhibiting the transcriptional activity of OsMYCs (Table 5) (Li et al., 2021b). NF-YA family genes play negative roles in antiviral defense by dissociating the OsMYC2/3-OsMED25 complex, and RSV and SRBSDV infection induce the expression of OsNF-YA family genes, repressing the JA synthesis pathway and increasing the susceptibility of rice to viral infection (Tan et al., 2022).

Importantly, recent studies have described the crosstalk between JA and other signaling pathways. A study revealed that both JA and BR signaling positively regulate rice defense against RSV (Hu et al., 2020). RSV infection induces OsGSK2, which subsequently phosphorylates OsMYC2, a master transcription factor of JA signaling. This leads to the degradation of OsMYC2, suppressing the JA-mediated RSV resistance response (Hu et al., 2020). OsGSK2 serves as a key factor linking the BR and JA signaling pathways (Hu et al., 2020). Like SA and ABA, JA is also involved in the transcriptional regulation of RNA silencing components (Figure 2) (Yang et al., 2020). AGO18 is one of the most well-studied RNA-silencing components in rice (Wu et al., 2015). The transcription of OsAGO18 is highly induced by different viruses, including RSV, RDV, and RBSDV (Wu et al., 2017b; Wu et al., 2015). A recent study suggested that JA signaling is responsible for AGO18 induction upon RSV infection (Yang et al., 2020). The JA-responsive transcription factor JAMYB can bind the promoter region of *OsAGO18* and activate its transcription. Normally, OsJAZ6 interacts with JAMYB and inhibits its transcriptional activity. Upon viral infection, the RSV coat protein (CP) induces JA accumulation. JA signaling activates JAMYB accumulation and promotes the degradation of OsJAZ6 in a COI1-dependent manner, ultimately leading to the transcriptional activation of *OsAGO18* (Yang et al., 2020). Interestingly, a recent finding also revealed that overexpression of CP activates rice jasmonic acid (JA) signaling and inhibits virus infection while attracting the insect vector (small brown planthopper) to promote virus spread (Han et al., 2020). According to these studies, more clues need to be collected to link these pathways and reveal a comprehensive regulatory network.

Other signaling pathways mediate antiviral defense in crops

Ubiquitination

A growing body of evidence indicates that the ubiquitin/26S proteasome system (UPS) degradation of viral or cellular proteins is a critical antiviral strategy for plants (Lozano-Durán et al., 2011; Shen et al., 2016). The UPS has been reported to mediate the degradation of many viral proteins, such as the movement proteins (MPs) encoded by TMV, turnip yellow mosaic virus (TYMV), and potato leafroll virus, the triple gene block protein 3 (TGBp3) of PVX, and the RNA-dependent RNA polymerase (RdRp) of TYMV (Camborde et al., 2010; Drugeon and Jupin, 2002; Vogel et al., 2007). Ubiquitination, a posttranslational modification in which ubiquitin is covalently attached via its carboxyl terminus to substrate lysine (Lys) residues, is a central process of the UPS (Miricescu et al., 2018; Zeng et al., 2006). During this process, the E3 ubiquitin ligase recruits specific ubiquitination substrates and determines the specificity of ubiquitination (Miricescu et al., 2018; Zeng et al., 2006). Rice RFPH2-10, which contains a C3H2C3-type RING-finger motif in its N-terminus, has been found to be an E3 ligase and mediate the degradation of the RDV P2 protein (Liu et al., 2014a). The overexpression of *OsRFPH2-10* enhances the rice defense response to RDV infection at the early stage of infection (Liu et al., 2014a). S-Adenosylmethionine decarboxylase 3 (SAMDC3) enhances the plant defense response to BSMV by promoting the ubiquitination and proteasomal degradation of the virus-encoded pathogenicity determinant γb (Li et al., 2022c). NtRFP1, a tobacco RING-finger protein, functions as an E3 ubiquitin ligase to mediate the ubiquitination of the βC1 protein encoded by the tomato yellow leaf curl China virus-associated betasatellite (TYLCCVB) (Shen et al., 2016). Moreover, overexpression of NtRFP1 attenuates βC1-induced symptoms by promoting βC1 degradation via the UPS (Shen et al., 2016). Ubiquitin-like protein 5 (UBL5) in both *N. benthamiana* and rice interacts with the RSV p3 (NS3) protein, which is encoded by the sense strand of RNA3, leading to its degradation through the 26S proteasome (Chen and Ding, 2020a). As a counterdefense, viruses have developed sophisticated strategies to usurp the host UPS for their own benefit. The RBSDV-encoded P5-1 protein regulates the

Table 5. The responses of plant hormone signaling mutants during virus infection in crops

Mutants	Gene	Response to virus infection	References
osarf17	*Auxin response factor (ARF) transcription factors 17*	Sensitive to RBSDV/RSMV/SRBSDV/RSV	Zhang et al., 2020b
osarf5	*Auxin response factor (ARF) transcription factors 5*	Resistant to RDV	Qin et al., 2020
osarf11	*Auxin response factor (ARF) transcription factors 11*	Resistant to RDV	
osarf12	*Auxin response factor (ARF) transcription factors 12*	Sensitive to RDV	
osarf16	*Auxin response factor (ARF) transcription factors 16*	Sensitive to RDV	
osiaa31	*Auxin/indole-3-acetic acid (Aux/IAA) protein 31*	Resistant to RBSDV	Zhang et al., 2019
osiaa20	*Auxin/indole-3-acetic acid (Aux/IAA) protein 20*	Resistant to RBSDV	
osiaa10	*Auxin/indole-3-acetic acid (Aux/IAA) protein 10*	Resistant to RDV	Jin et al., 2016
ossams1	S-adenosyl-L-methionine synthetase (*SAMS*), a key component of the ethylene synthesis pathway	Resistant to RDV	Zhao et al., 2017
osein2	Ethylene insensitive 2	Resistant to RDV	
osmyc2	*MYC2* transcription factor in the JA signalling pathway	Sensitive to RSV	Hu et al., 2020
oscoi1	*Coronatine insensitive 1*, encodes a JA receptor	Sensitive to RDV/RSV/RBSDV	Yang et al., 2020
osjaz6	Jasmonate ZIM-domain (JAZ) family, *OsJAZ6*	Resistant to RDV/RSV	
osjamyb	R2R3-type MYB transcription factor, jasmonic acid-inducible Rice *myb* gene	Sensitive to RDV/RSV	
osmyc3	MYC3 transcription factor in the JA signalling pathway	Sensitive to RBSDV/RSMV/SRBSDV/RSV	Li et al., 2021b
osnced3-2/osabaox	ABA biosynthetic genes	Resistant to RBSDV	Xie et al., 2018
osgsk2	Glycogen synthase kinase3 (GSK3)–like kinase serving as a key suppressor of BR signaling *OsGSK2*,	Sensitive to RSV, but resistant to RBSDV	Hu et al., 2020
zmpals	Maize phenylalanine ammonia-lyases (*ZmPALs*), required for SA accumulation in maize	Sensitive to SCMV	Yuan et al., 2019

ubiquitination activity of SCF E3 ligases and inhibits jasmonate signaling to promote infection in rice (He et al., 2020). The P3 protein encoded by rice grassy stunt virus (RGSV), a negative-strand RNA virus in *Bunyavirales*, induces the expression of P3IP1, a P3-inducible U-box type E3 ubiquitin ligase (Zhang et al., 2020a). P3IP1 interacts with a component of RNA-directed DNA methylation pathway, the NUCLEAR RNA POLYMERASE D1a (OsNRPD1a), which is a subunit of plant RNA polymerase IV (Pol IV), leading to ubiquitination and UPS-dependent degradation of OsNRPD1a and promoting disease symptoms (Zhang et al., 2020a). Microtubule-associated E3 ligase (MEL), a C4HC3-type E3 ligase, interacts with and ubiquitinates hydroxymethyltransferase (SHMT1), playing a crucial role in broad-spectrum plant defense. However, RSV NS3 suppresses the MEL-SHMT1 module, promoting the accumulation of SHMT1 and suppressing SHMT1-mediated disease resistance (Fu et al., 2022). A more recent study showed that RSV P2 promotes the interaction between OsNPR1 and the E3 ligase protein OsCUL3a. This CUL-ring-like E3 ligase complex accelerates the degradation of OsNPR1, increasing the sensitivity of rice to virus infection. RSV utilizes the host's UPS for successful invasion (Zhang et al., 2023b). Additionally, the infection of beet severe curly top virus (BSCTV) induces the expression of a host-imprinted E3 ligase encoding gene, *VARIANTINMETHYLATION5* (*VIM5*), leading to the UPS-mediated degradation of two DNA methyltransferases, METHYLTRANSFERASE1 (MET1) and CHROMOMETHYLASE3 (CMT3), thus escaping epigenetic modification mediated antiviral defense (Chen et al., 2020b). Regardless of whether the host's ubiquitination system is promoted or inhibited, viruses have evolved the best ways to facilitate viral infection.

Autophagy

Autophagy is an essential and conserved degradation mechanism that removes damaged or unwanted cellular materials in eukaryotes. Studies in model plants have shown the critical roles of autophagy in regulating host antiviral defense and viral pathogenesis during plant–virus interactions (Hafrén et al., 2017; Hafrén et al., 2018; Li et al., 2020; Li et al., 2018; Yang and Liu, 2022). Autophagy initiates with the formation of autophagosomes, which rely on diverse membrane factors and are mediated by a set of AUTOPHAGY (ATG) proteins (Ismayil et al., 2020b; Jia et al., 2023; Yang and Liu, 2022). Cotton leaf curl Multan virus (CLCuMuV) is associated with the disease-specific satellite DNA cotton leaf curl Multan betasatellite (CLCuMuB, βC1). The autophagic machinery can target and degrade the βC1 protein of CLCuMuB through its interaction with autophagy-related protein 8 (ATG8) (Figure 3) (Haxim et al., 2017; Ismayil et al., 2020a). The autophagy pathway is activated and enhanced by CLCuMuV infection. Silencing of *ATG5* and *ATG7* weakens plant antiviral autophagy defense and causes severe disease symptoms (Haxim et al., 2017). CLCuMuB βC1 can induce autophagy by disrupting the interaction of ATG3 with glyceraldehyde-3-phosphate dehydrogenase (GAPC) (Ismayil et al., 2020a).

As a counterdefense, viruses can also inhibit or hijack the host autophagy pathway or ATG proteins to suppress host antiviral responses. Autophagy is suppressed by BSMV infection, and overexpression of the BSMV γb protein is sufficient to inhibit autophagy. Further studies showed that the BSMV γb protein disrupts the interaction between ATG7 and ATG8 by competitively interacting with ATG7, thereby subverting autophagy-mediated antiviral defense to promote viral infection (Figure 3) (Yang et al., 2018b). Similarly, the C2 protein encoded by tomato leaf curl Yunnan virus (TLCYnV) and several other geminivirus proteins also disrupt the ATG7-ATG8 complex in *N. benthamiana* and *Solanum lycopersicum* plants by interacting with the ubiquitin-activating domain of ATG7 (Cao et al., 2023). Thus,

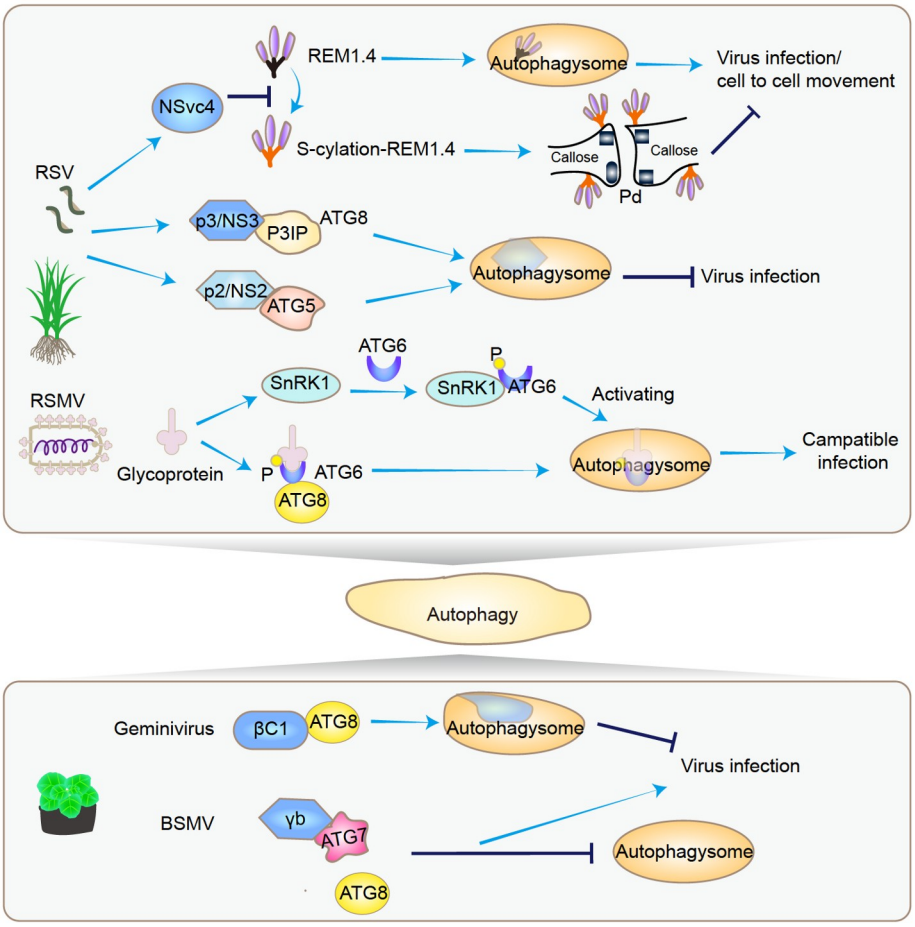

Figure 3. Role of autophagy during crop plant–virus interactions. The autophagy pathway plays a dual role in antiviral immunity in both crop plants and model plants. ATG proteins target viral proteins for degradation to enhance plant antiviral defense. In contrast, viruses can employ autophagy to degrade host antiviral factors. RSV, rice stripe virus; RSMV, rice stripe mosaic virus; BSMV, barley stripe mosaic virus.

TLCYnV and different geminiviruses have evolved this counter-defensive strategy to overcome plant antiviral autophagy (Cao et al., 2023). Additionally, the same virus can employ different autophagy-inhibition strategies, such as BSMV replicase γa, which inhibits host antiviral defense by disrupting vacuolar acidification and suppressing the degradation of autophagosome, leading to successful infection (Yang et al., 2022). The CLCuMuV C4 protein suppresses autophagy to facilitate viral infection by binding to the negative autophagy regulator eukaryotic translation initiation Factor 4A (eIF4A) to enhance the eIF4A-ATG5 interaction (Yang et al., 2023). Remorins are plant-specific membrane-associated proteins that play crucial roles in plant–pathogen interactions by controlling the size-exclusion limit of plasmodesmata. The NSvc4 protein, a movement protein of RSV, interferes with remorin protein (NbREM1) S-acylation through binding with the C-terminal domain of NbREM1 and promotes its degradation by autophagy in RSV-infected tobacco (Fu et al., 2018). Similarly, NSvc4 also impairs S-acylation of the homologous rice remorin protein (OsREM1.4) and triggers its degradation through the autophagy pathway to facilitate virus cell-to-cell movement (Figure 3) (Fu et al., 2018).

Autophagy plays antiviral and proviral roles in plant-CMV interactions. At the early stages of CMV infection, VISP1 (VIRUS-INDUCED SMALL PEPTIDE 1), a virus-induced peptide, serves as an autophagy receptor, mediating the degradation of SGS3/RDR6-bodies and thus suppressing the amplification of siRNAs and enhancing CMV infection. At late stages of CMV infection, with the accumulation of the virus, VISP1 inhibits virus invasion by accelerating the autophagic degradation of CMV protein 2b instead of SGS3/RDR6 bodies. Thus, VISP1 acts as a double-edged sword in the early and late stages of CMV infection (Tong et al., 2023; Tong et al., 2021).

Studies in crops have also validated the role of autophagy in the crop plant antiviral immune response. Rice P3IP protein was reported to mediate RSV p3 degradation by interacting with OsATG8b (Figure 3) (Jiang et al., 2020). The over-expression of OsP3IP activates autophagy and enhances resistance to RSV infection (Jiang et al., 2020). Further study revealed that OsATG5 interacts with RSV p2 and targets it for degradation through autophagy (Zhang et al., 2023c). The glycoprotein of RSMV is toxic to rice cells. Once OsSnRK1B perceives the RSMV glycoprotein, the kinase activity of OsSnRK1B on OsATG6b is promoted. The OsATG6b-mediated autophagy of the RSMV glycoprotein is an indispensable defense mechanism during RSMV infection (Huang et al., 2024). These findings provide more insights into crop antiviral defense based on autophagy regulation and targets for crop antiviral resistance breeding.

Reactive oxygen species (ROS)-mediated antiviral defense

The generation of ROS such as hydrogen peroxide (H_2O_2) or superoxide anions ($O_2^{\cdot-}$) constitutes one of the earliest cellular defense responses to pathogen infections (Mhamdi and Van Breusegem, 2018; Qi et al., 2017). Rice glycolate oxidase (GOX) has been reported to interact with the RDV P8 protein, leading to the translocation of P8 to peroxisomes (Zhou et al., 2007). A recent study showed that the BSMV γb protein interacts with GOX to inhibit GOX-derived H_2O_2 production in *N. benthamiana*, reducing ROS bursts during BSMV infection (Yang et al., 2018a). OsAO, a target of miR528, regulates the redox state of the apoplast by oxidizing apoplastic ascorbic acid (AsA), resulting in ROS accumulation to enhance rice antiviral defense. Upon viral infection, the expression of miR528 is downregulated, and mature miR528 is recruited by AGO18, which releases *AO* to increase ROS levels (Wu et al., 2017b). Further study revealed that the miR528-AO-ROS defense module is transcriptionally regulated by OsSPL9 (Yao et al., 2019). The deficiency of OsSPL9 increases miR528 expression and reduces AO accumulation, compromising rice antiviral defense (Yao et al., 2019). In addition, a recent finding showed that the transcriptional repressor Alfin-like 7 (AL7) could interact with the NLR and bind to the promoters of ROS-scavenging genes (Zhang et al., 2023a). When AL7 is phosphorylated by mitogen-activated protein kinases (MAPKs), salicylic acid-induced protein kinase (SIPK), and wound-induced protein kinase (WIPK), the interaction between AL7 and the NLR is attenuated, enhancing its DNA binding activity. This in turn promotes ROS accumulation and activates plant immunity (Zhang et al., 2023a). This result indicates that ROS bursts are likewise involved in NLR-mediated antiviral immunity.

Concluding remarks and future perspectives

Plants are the major components of the ecosystem, which must dynamically adapt to changes under environmental conditions, particularly during stress, to ensure their survival and better growth and development (Cheng et al., 2019; Fraile and García-Arenal, 2016). Plant viruses are among the most economically devastating microorganisms, leading to crop losses and serious threats to food security. Understanding how plants, especially crops, respond to viral infections in the face of rapid environmental changes remains a complex challenge (Tenllado and Canto, 2020). Most crop viruses are transmitted by insect vectors (Table 6); therefore, viral disease outbreaks often correlate with vector adaptation to the environment. In light of global climate changes, future research should increasingly consider the various environmental factors that affect plant–virus interactions and/or plant-insect vector-virus multiple interactions, which also include the endosymbionts of insect vectors with viruses. Although various mechanisms underlying virus-plant host interactions have been uncovered, there is still a need for comprehensive analyses of defense and counterdefense systems among crop plants, viruses, insect-vectors as well as endosymbionts of insect-vectors (Figure 4). The relationships among different antiviral mechanisms are not fully revealed, e.g., which mechanism/step is more upstream or downstream compared with others? Which pathways are functionally parallel? Future studies may provide more perspectives about these questions.

With the rapid advances in multiomics, bioinformatics, precise

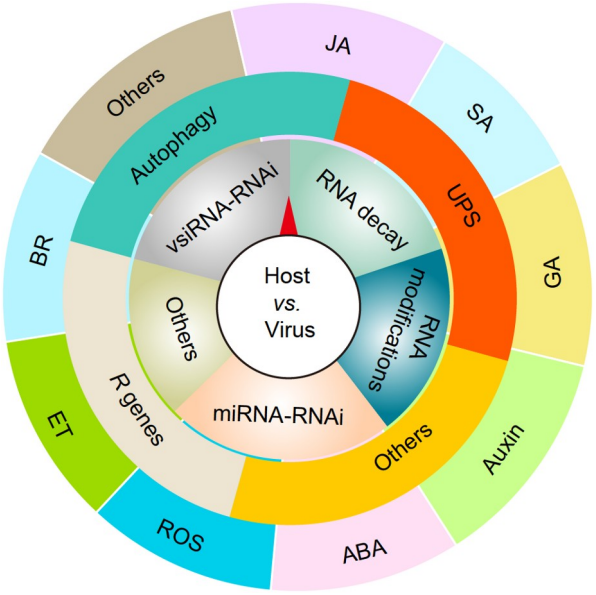

Figure 4. Schematic diagram of the complex multilayered defense system involved in plant–virus interactions. In recent decades, many pathways have been revealed to play roles in plant–virus interactions. Interestingly, many types of crosstalks between different pathways have been discovered. This diagram indicates that different pathways in different layers can cooperate with or antagonize each other. The outer layer refers to the small molecule signaling pathways mediated antiviral defense, the middle layer represents the antiviral strategies through the functional proteins, and the inner layer represents antiviral strategies at the RNA level. Additionally, many unknown mechanisms remain to be investigated, especially in crops.

genome editing, big data and AI as well other bio- and AI-technology, it is crucial to apply these new emerging technologies to address longstanding scientific questions (Wen et al., 2023). The fate of host to viral infections mainly depends on the genotypes of host, but many other bio- and abio-factors contribute to host responses to viral infections. On one hand, viral infections can reprogram host metabolism, including changes in root exudates, and the impacts of these metabolites on plant–virus interactions warrant further investigation. On the other hand, viral infection may remold the rhizosphere and foliar microbiomes of hosts, and in turn, the rhizosphere and foliar microbiomes may also affect host upon viral infections. Studying the rhizosphere and foliar microbiomes of host crops in the context of antiviral immunity represents a new research direction for the future.

Another major challenge in identifying emerging infectious diseases in the future is the timely identification of new viral pathogens. With the development of metagenomic research tools, we can discover, isolate, and evaluate new potential viral pathogens from typical field environments and propose a catalog or list of new viruses that may cause outbreaks in the future (Wang et al., 2024). The enhanced capacity for pandemic preparedness partially allows for us to proactively control viral diseases.

Meristem tip culture is an effective method for producing virus-free plants in many species. The long-standing mystery of why the shoot apical meristem (SAM) is free from viral invasion has been partially unraveled in recent research, suggesting that WUSCHEL triggers antiviral defense by impairing global protein synthesis and limiting virus replication and spread (Wu et al., 2020). Furthermore, the SA signaling and RNAi pathways are

Table 6. The transmission vectors or methods of different plant viruses

Viruses	Transmitted vectors/methods
Tobacco mosaic virus (TMV)	Aphids, flies
Potato virus X (PVX)	Aphids
Potato virus Y (PVY)	Aphids
Tomato leaf curl New Delhi virus (tolcndv)	Whitefly (*Bemisia tabaci*)
Tomato spotted wilt orthotospovirus (TSWV)	Thrips
Soybean mosaic virus (SMV)	Aphids or seed-transmitted
Barley stripe mosaic virus (BSMV)	Seed-transmitted
Rice yellow mottle virus (RYMV)	Chrysomelid beetles (Coleoptera)
Wheat yellow mosaic virus (WYMV)	Obligate soil-inhabiting fungus-like organism *Polymyxa graminis*
Turnip mosaic virus (TuMV)	Aphids
Rice black-streaked dwarf virus (RBSDV)	Small brown planthopper (*Laodelphax striatellus*)
Oryza sativa endornavirus (OsEV)	Vertical transmission through infected pollen and ova
Rice stripe virus (RSV)	Small brown planthopper (*Laodelphax striatellus*)
Rice dwarf virus (RDV)	Leafhoppers (*Nephotettix cincticeps* or *Recilia dorsalis*)
Rice ragged stunt virus (RRSV)	Brown planthopper (*Nilaparvata lugens*)
Barley yellow striate mosaic virus (BYSMV)	Small brown planthopper (*Laodelphax striatellus*)
Pea mosaic virus 2 (PEMV2)	Aphids
Turnip crinkle virus (TCV)	Flea beetles (*Psylloides* and *Phyllotreta*)
Rice stripe mosaic virus (RSMV)	Leafhoppers (*Recilia dorsalis*)
Southern rice black-streaked dwarf virus (SRBSDV)	White-backed planthopper (*Sogatella furcifera*)
Tomato bushy stunt virus (TBSV)	Soil-borne
Sugarcane mosaic virus (SCMV)	Aphids
Cucumber mosaic virus (CMV)	Aphids
Turnip yellow mosaic virus (TYMV)	Flea-beetles (*Phyllotreta and Psylliodes*), mustard beetle (*Phaedon cochleariae*)
Potato leafroll virus	Aphids
Rice grassy stunt virus (RGSV)	Brown planthopper (*Nilaparvata lugens*)
Beet severe curly top virus (BSCTV)	Leafhopper (*Circulifer tenellus*)
Cotton leaf curl Multan virus (CLCuMuV)	Whitefly (*Bemisia tabaci*)
Tomato leaf curl Yunnan virus (TLCYnV)	Whitefly (*Bemisia tabaci*)

necessary for stem cell exclusion of several unrelated RNA viruses, including TuMV (Incarbone et al., 2023). With the development of bioengineering technology, the fusion of NLR immune receptors with nanobodies is being used to confer resistance to plant pathogen infection. Additionally, artificially designed plant immune proteins can be purified from animals, and new immune receptors capable of recognizing fluorescent proteins such as GFP and mCherry can be integrated into plant immune receptor NLR proteins, offering potential strategies to combat viruses (Kourelis et al., 2023). There are some interesting but challenging questions: how do plants perceive viral infection? How do host factors control the tissue-specific response upon viral infection, especially under natural infection conditions? The utilization of single-cell sequencing, spatially resolved transcriptomic technology, and single-molecular dynamics tracking technology, which provides a powerful tool for visualizing protein dynamics and localization in living cells (Törk et al., 2023), may contribute to answering these questions.

Circular RNAs, produced from precursor mRNA backsplicing of exon(s), have been connected with innate immune responses in mammalian cells (Li et al., 2017; Liu et al., 2019). Whether circular RNAs play a role during plant virus infection remains an open question. Finally, exploring the potential use of viruses for bio-control or other beneficial purposes and finding ways to connect the intricate mechanisms underlying these interactions to achieve a comprehensive and multidimensional understanding of plant–virus interactions will be essential for future research endeavors. By providing additional multilayer interaction data on plant–virus-insect-microbiome-environmental factors, deep learning and machine learning (ML) could be very useful for providing quick and accurate estimates and managing field viral disease control.

Compliance and ethics

The authors declare that they have no conflict of interest.

Acknowledgement

We thank Prof.s Yule Liu (Tsinghua University) and Xueping Zhou (Institute of Plant Protection, Chinese Academy of Agricultural Sciences) for their valuable suggestions and critical reading of this manuscript. We thank Dr.s Guangyuan Cheng (School of Life Sciences, Peking University) and Shibo Yu (College of Plant Protection, Fujian Agriculture and Forestry University) for helping in literature collections. We apologize to those authors whose primary works could not be cited owing to space limitations. This work was funded by the National Key R&D Program of China (2023YFD1400300) to MW and National Natural Science Foundation to YL (32090010).

References

Akond, Z., Rahman, H., Ahsan, M.A., Mosharaf, M.P., Alam, M., Mollah, H. (2020). Bioinformatic analysis based genome-wide identification characterization, diversification and regulatory transcription components of RNA silencing machinery genes in wheat (*Triticum aestivum* L.). bioRxiv 218891652.

Alamillo, J.M., Saénz, P., and García, J.A. (2006). Salicylic acid-mediated and RNA-silencing defense mechanisms cooperate in the restriction of systemic spread of plum pox virus in tobacco. Plant J 48, 217–227.
Alazem, M., Bwalya, J., Hsuan, P., Yu, J., Cam Chu, H., Burch-Smith, T., and Kim, K. H. (2023). Viral synergism suppresses *R* gene-mediated resistance by impairing downstream defense mechanisms in soybean. Plant Physiol 192, 3088–3105.
Alazem, M., He, M.H., Moffett, P., and Lin, N.S. (2017). Abscisic acid induces resistance against *Bamboo Mosaic Virus* through *Argonaute 2* and *3*. Plant Physiol 174, 339–355.
Alazem, M., Kim, K.H., and Lin, N.S. (2019). Effects of abscisic acid and salicylic acid on gene expression in the antiviral RNA silencing pathway in *Arabidopsis*. Int J Mol Sci 20, 2538.
Alazem, M., and Lin, N.S. (2017). Antiviral roles of abscisic acid in plants. Front Plant Sci 8, 1.
Alazem, M., and Lin, N.-. (2015). Roles of plant hormones in the regulation of host–virus interactions. Mol Plant Pathol 16, 529–540.
Alazem, M., and Lin, N.S. (2020). Interplay between ABA signaling and RNA silencing in plant viral resistance. Curr Opin Virol 42, 1–7.
Albar, L., Bangratz-Reyser, M., Hébrard, E., Ndjiondjop, M.-., Jones, M., and Ghesquière, A. (2006). Mutations in the eIF(iso)4G translation initiation factor confer high resistance of rice to *Rice yellow mottle virus*. Plant J 47, 417–426.
Andrade, M., Abe, Y., Nakahara, K.S., and Uyeda, I. (2009). The cyv-2 resistance to Clover yellow vein virus in pea is controlled by the eukaryotic initiation factor 4E. J Gen Plant Pathol 75, 241–249.
Bai, M., Yang, G.S., Chen, W.T., Mao, Z.C., Kang, H.X., Chen, G.H., Yang, Y.H., and Xie, B.Y. (2012). Genome-wide identification of dicer-like, argonaute and RNA-dependent RNA polymerase gene families and their expression analyses in response to viral infection and abiotic stresses in *Solanum lycopersicum*. Gene 501, 52–62.
Baurès, I., Candresse, T., Leveau, A., Bendahmane, A., and Sturbois, B. (2008). The *Rx* Gene Confers Resistance to a Range of *Potexviruses* in Transgenic *Nicotiana* Plants. Mol Plant Microbe Interact 21, 1154–1164.
Bendahmane, A., Querci, M., Kanyuka, K., and Baulcombe, D.C. (2000). *Agrobacterium* transient expression system as a tool for the isolation of disease resistance genes: application to the *Rx2* locus in potato. Plant J 21, 73–81.
Bi, G., Su, M., Li, N., Liang, Y., Dang, S., Xu, J., Hu, M., Wang, J., Zou, M., Deng, Y., et al. (2021). The ZAR1 resistosome is a calcium-permeable channel triggering plant immune signaling. Cell 184, 3528–3541.e12.
Bijaisoradat, M., and Kuhn, C.W. (1985). Nature of resistance in soybean to cowpea chlorotic mottle virus. Phytopathology 75, 351–355.
Boualem, A., Dogimont, C., and Bendahmane, A. (2016). The battle for survival between viruses and their host plants. Curr Opin Virol 17, 32–38.
Broekgaarden, C., Caarls, L., Vos, I.A., Pieterse, C.M.J., and Van Wees, S.C.M. (2015). Ethylene: Traffic controller on hormonal crossroads to defense. Plant Physiol pp.01020.2015.
Calil, I.P., and Fontes, E.P.B. (2017). Plant immunity against viruses: Antiviral immune receptors in focus. Ann Bot 119, 711–723.
Camborde, L., Planchais, S., Tournier, V., Jakubiec, A., Drugeon, G., Lacassagne, E., Pflieger, S., Chenon, M., and Jupin, I. (2010). The ubiquitin-proteasome system regulates the accumulation of *Turnip yellow mosaic virus* RNA-dependent RNA Polymerase during viral infection. Plant Cell 22, 3142–3152.
Cao, B., Ge, L., Zhang, M., Li, F., and Zhou, X. (2023). Geminiviral C2 proteins inhibit active autophagy to facilitate virus infection by impairing the interaction of ATG7 and ATG8. JIPB 65, 1328–1343.
Cao, J.Y., Xu, Y.P., Li, W., Li, S.S., Rahman, H., and Cai, X.Z. (2016). Genome-wide identification of dicer-like, argonaute, and RNA-dependent RNA polymerase gene families in brassica species and functional analyses of their *Arabidopsis* homologs in resistance to *Sclerotinia sclerotiorum*. Front Plant Sci 7, 121.
Cao, M., Du, P., Wang, X., Yu, Y.Q., Qiu, Y.H., Li, W., Gal-On, A., Zhou, C., Li, Y., and Ding, S.W. (2014). Virus infection triggers widespread silencing of host genes by a distinct class of endogenous siRNAs in *Arabidopsis*. Proc Natl Acad Sci USA 111, 14613–14618.
Cao, X., Zhou, P., Zhang, X., Zhu, S., Zhong, X., Xiao, Q., Ding, B., and Li, Y. (2005). Identification of an RNA silencing suppressor from a plant double-stranded RNA virus. J Virol 79, 13018–13027.
Chakraborty, J., Jain, A., Mukherjee, D., Ghosh, S., and Das, S. (2018). Functional diversification of structurally alike NLR proteins in plants. Plant Sci 269, 85–93.
Chen, B., Lin, L., Lu, Y., Peng, J., Zheng, H., Yang, Q., Rao, S., Wu, G., Li, J., Chen, Z., et al. (2020). Ubiquitin-Like protein 5 interacts with the silencing suppressor p3 of rice stripe virus and mediates its degradation through the 26S proteasome pathway. PLoS Pathog 16, e1008780.
Chen, J., Zhao, Y., Luo, X., Hong, H., Yang, T., Huang, S., Wang, C., Chen, H., Qian, X., Feng, M., et al. (2023). NLR surveillance of pathogen interference with hormone receptors induces immunity. Nature 613, 145–152.
Chen, L., Zhang, L., Li, D., Wang, F., and Yu, D. (2013). WRKY8 transcription factor functions in the TMV-cg defense response by mediating both abscisic acid and ethylene signaling in *Arabidopsis*. Proc Natl Acad Sci USA 110, E1963–E1971.
Chen, R., Tu, Z., He, C., Nie, X., Li, K., Fei, S., Song, B., Nie, B., and Xie, C. (2022). Susceptibility factor StEXA1 interacts with StnCBP to facilitate potato virus Y accumulation through the stress granule-dependent RNA regulatory pathway in potato. Horticulture Res 9, uhac159.
Chen, Z.Q., and Ding, S.W. (2020a). Jacked responses go viral: hormonal regulation of antiviral RNAi. Cell Host Microbe 28, 7–9.
Chen, Z.Q., Zhao, J.H., Chen, Q., Zhang, Z.H., Li, J., Guo, Z.X., Xie, Q., Ding, S.W., and Guo, H.S. (2020b). DNA geminivirus infection induces an imprinted E3 ligase gene to epigenetically activate viral gene transcription. Plant Cell 32, 3256–3272.
Cheng, Y.T., Zhang, L., and He, S.Y. (2019). Plant-microbe interactions facing environmental challenge. Cell Host Microbe 26, 183–192.
Chisholm, S.T., Mahajan, S.K., Whitham, S.A., Yamamoto, M.L., and Carrington, J.C. (2000). Cloning of the *Arabidopsis RTM1* gene, which controls restriction of long-distance movement of tobacco etch virus. Proc Natl Acad Sci USA 97, 489–494.
Cooley, M.B., Pathirana, S., Wu, H.J., Kachroo, P., and Klessig, D.F. (2000). Members of the *Arabidopsis HRT/RPP8* family of resistance genes confer resistance to both viral and oomycete pathogens. Plant Cell 12, 663–676.
Vlot, A.C., Dempsey, D.M.A., and Klessig, D.F. (2009). Salicylic acid, a multifaceted hormone to combat disease. Annu Rev Phytopathol 47, 177–206.
Cui, C., Wang, J.J., Zhao, J.H., Fang, Y.Y., He, X.F., Guo, H.S., and Duan, C.G. (2020a). A brassica miRNA regulates plant growth and immunity through distinct modes of action. Mol Plant 13, 231–245.
Cui, S., Kubota, T., Nishiyama, T., Ishida, J.K., Shigenobu, S., Shibata, T.F., Toyoda, A., Hasebe, M., Shirasu, K., and Yoshida, S. (2020b). Ethylene signaling mediates host invasion by parasitic plants. Sci Adv 6, eabc2385.
DeYoung, B.J., and Innes, R.W. (2006). Plant NBS-LRR proteins in pathogen sensing and host defense. Nat Immunol 7, 1243–1249.
Díaz-Pendón, J.A., and Ding, S.W. (2008). Direct and indirect roles of viral suppressors of RNA silencing in pathogenesis. Annu Rev Phytopathol 46, 303–326.
Diaz-Pendon, J.A., Li, F., Li, W.X., and Ding, S.W. (2007). Suppression of antiviral silencing by cucumber mosaic virus 2b protein in *Arabidopsis* is associated with drastically reduced accumulation of three classes of viral small interfering RNAs. Plant Cell 19, 2053–2063.
Drugeon, G., and Jupin, I. (2002). Stability *in vitro* of the 69K movement protein of Turnip yellow mosaic virus is regulated by the ubiquitin-mediated proteasome pathway. J Gen Virol 83, 3187–3197.
Duxbury, Z., Wu, C., and Ding, P. (2021). A comparative overview of the intracellular guardians of plants and animals: NLRs in innate immunity and beyond. Annu Rev Plant Biol 72, 155–184.
Fang, X., and Qi, Y. (2016). RNAi in plants: an argonaute-centered view. Plant Cell 28, 272–285.
Fraile, A., and García-Arenal, F. (2016). Environment and evolution modulate plant virus pathogenesis. Curr Opin Virol 17, 50–56.
Fu, S., Wang, K., Ma, T., Liang, Y., Ma, Z., Wu, J., Xu, Y., and Zhou, X. (2022). An evolutionarily conserved C4HC3-type E3 ligase regulates plant broad-spectrum resistance against pathogens. Plant Cell 34, 1822–1843.
Fu, S., Xu, Y., Li, C., Li, Y., Wu, J., and Zhou, X. (2018). Rice stripe virus interferes with S-acylation of remorin and induces its autophagic degradation to facilitate virus infection. Mol Plant 11, 269–287.
Gallei, M., Luschnig, C., and Friml, J. (2020). Auxin signalling in growth: Schrödinger's cat out of the bag. Curr Opin Plant Biol 53, 43–49.
Gao, F., Zhao, S., Men, S., Kang, Z., Hong, J., Wei, C., Hong, W., and Li, Y. (2020a). A non-structural protein encoded by Rice Dwarf Virus targets to the nucleus and chloroplast and inhibits local RNA silencing. Sci China Life Sci 63, 1703–1713.
Gao, L., Luo, J., Ding, X., Wang, T., Hu, T., Song, P., Zhai, R., Zhang, H., Zhang, K., Li, K., et al. (2020b). Soybean RNA interference lines silenced for eIF4E show broad potyvirus resistance. Mol Plant Pathol 21, 303–317.
Gao, Z., Johansen, E., Eyers, S., Thomas, C.L., Noel Ellis, T.H., and Maule, A.J. (2004). The potyvirus recessive resistance gene, *sbm1*, identifies a novel role for translation initiation factor eIF4E in cell-to-cell trafficking. Plant J 40, 376–385.
Garcia-Ruiz, H., Takeda, A., Chapman, E.J., Sullivan, C.M., Fahlgren, N., Brempelis, K. J., and Carrington, J.C. (2010). *Arabidopsis* RNA-dependent RNA polymerases and dicer-like proteins in antiviral defense and small interfering RNA biogenesis during *Turnip Mosaic Virus* infection. Plant Cell 22, 481–496.
Garcia, D., Garcia, S., and Voinnet, O. (2014). Nonsense-mediated decay serves as a general viral restriction mechanism in plants. Cell Host Microbe 16, 391–402.
García, J.A., and Pallás, V. (2015). Viral factors involved in plant pathogenesis. Curr Opin Virol 11, 21–30.
Ge, L., Cao, B., Qiao, R., Cui, H., Li, S., Shan, H., Gong, P., Zhang, M., Li, H., Wang, A., et al. (2023). SUMOylation-modified Pelota-Hbs1 RNA surveillance complex restricts the infection of potyvirids in plants. Mol Plant 16, 632–642.
Ge, L., Zhou, X., and Li, F. (2024). Plant–virus arms race beyond RNA interference.

Trends Plant Sci 29, 16–19.
Gobert, A., Quan, Y., Arrivé, M., Waltz, F., Da Silva, N., Jomat, L., Cohen, M., Jupin, I., and Giegé, P. (2021). Towards plant resistance to viruses using protein-only RNase P. Nat Commun 12, 1–6.
Gong, Q., Wang, Y., He, L., Huang, F., Zhang, D., Wang, Y., Wei, X., Han, M., Deng, H., Luo, L., et al. (2023). Molecular basis of methyl-salicylate-mediated plant airborne defence. Nature 622, 139–148.
Grech-Baran, M., Witek, K., Poznański, J.T., Grupa-Urbańska, A., Malinowski, T., Lichocka, M., Jones, J.D.G., and Hennig, J. (2022). The Ry_{sto} immune receptor recognises a broadly conserved feature of potyviral coat proteins. New Phytol 235, 1179–1195.
Grech-Baran, M., Witek, K., Szajko, K., Witek, A.I., Morgiewicz, K., Wasilewicz-Flis, I., Jakuczun, H., Marczewski, W., Jones, J.D.G., and Hennig, J. (2020). Extreme resistance to *Potato virus Y* in potato carrying the Ry_{sto} gene is mediated by a TIR-NLRimmune receptor. Plant Biotechnol J 18, 655–667.
Guilfoyle, T.J., and Hagen, G. (2007). Auxin response factors. Curr Opin Plant Biol 10, 453–460.
Guo, Z., Li, Y., and Ding, S.W. (2019). Small RNA-based antimicrobial immunity. Nat Rev Immunol 19, 31–44.
Hafrén, A., Macia, J.L., Love, A.J., Milner, J.J., Drucker, M., and Hofius, D. (2017). Selective autophagy limits cauliflower mosaic virus infection by NBR1-mediated targeting of viral capsid protein and particles. Proc Natl Acad Sci USA 114, E2026–E2035.
Hafrén, A., Üstün, S., Hochmuth, A., Svenning, S., Johansen, T., and Hofius, D. (2018). Turnip mosaic virus counteracts selective autophagy of the viral silencing suppressor HCpro. Plant Physiol 176, 649–662.
Hamar, É., Szaker, H.M., Kis, A., Dalmadi, Á., Miloro, F., Szittya, G., Taller, J., Gyula, P., Csorba, T., and Havelda, Z. (2020). Genome-wide identification of RNA silencing-related genes and their expressional analysis in response to heat stress in barley (*Hordeum vulgare* L.). Biomolecules 10, 929.
Han, K., Huang, H., Zheng, H., Ji, M., Yuan, Q., Cui, W., Zhang, H., Peng, J., Lu, Y., Rao, S., et al. (2020). Rice stripe virus coat protein induces the accumulation of jasmonic acid, activating plant defence against the virus while also attracting its vector to feed. Mol Plant Pathol 21, 1647–1653.
Haxim, Y., Ismayil, A., Jia, Q., Wang, Y., Zheng, X., Chen, T., Qian, L., Liu, N., Wang, Y., Han, S., et al. (2017). Autophagy functions as an antiviral mechanism against geminiviruses in plants. eLife 6, 1–7.
He, H., Ge, L., Chen, Y., Zhao, S., Li, Z., Zhou, X., and Li, F. (2024). m6A modification of plant virus enables host recognition by NMD factors in plants. Sci China Life Sci 67, 161–174.
He, L., Chen, X., Yang, J., Zhang, T., Li, J., Zhang, S., Zhong, K., Zhang, H., Chen, J., and Yang, J. (2020). *Rice black-streaked dwarf virus*-encoded P5-1 regulates the ubiquitination activity of SCF E3 ligases and inhibits jasmonate signaling to benefit its infection in rice. New Phytol 225, 896–912.
Hong, W., Qian, D., Sun, R., Jiang, L., Wang, Y., Wei, C., Zhang, Z., and Li, Y. (2015). OsRDR6 plays role in host defense against double-stranded RNA virus, Rice Dwarf Phytoreovirus. Sci Rep 5, 11324.
Hu, J., Huang, J., Xu, H., Wang, Y., Li, C., Wen, P., You, X., Zhang, X., Pan, G., Li, Q., et al. (2020). Rice stripe virus suppresses jasmonic acid-mediated resistance by hijacking brassinosteroid signaling pathway in rice. PLoS Pathog 16, e1008801.
Huang, R., Li, Y., Tang, G., Hui, S., Yang, Z., Zhao, J., Liu, H., Cao, J., and Yuan, M. (2018). Dynamic phytohormone profiling of rice upon rice black-streaked dwarf virus invasion. J Plant Physiol 228, 92–100.
Huang, X., Wang, J., Chen, S., Liu, S., Li, Z., Wang, Z., Chen, B., Zhang, C., Zhang, Y., Wu, J., et al. (2024). Rhabdovirus encoded glycoprotein induces and harnesses host antiviral autophagy for maintaining its compatible infection. Autophagy 20, 275–294.
Huot, B., Yao, J., Montgomery, B.L., and He, S.Y. (2014). Growth–defense tradeoffs in plants: a balancing act to optimize fitness. Mol Plant 7, 1267–1287.
Incarbone, M., Bradamante, G., Pruckner, F., Wegscheider, T., Rozhon, W., Nguyen, V., Gutzat, R., Mérai, Z., Lendl, T., MacFarlane, S., et al. (2023). Salicylic acid and RNA interference mediate antiviral immunity of plant stem cells. Proc Natl Acad Sci USA 120, e2302069120.
Ishibashi, K., Masuda, K., Naito, S., Meshi, T., and Ishikawa, M. (2007). An inhibitor of viral RNA replication is encoded by a plant resistance gene. Proc Natl Acad Sci USA 104, 13833–13838.
Ishibashi, K., Saruta, M., Shimizu, T., Shu, M., Anai, T., Komatsu, K., Yamada, N., Katayose, Y., Ishikawa, M., Ishimoto, M., et al. (2019). Soybean antiviral immunity conferred by dsRNase targets the viral replication complex. Nat Commun 10, 1–3.
Islam, W., Noman, A., Qasim, M., and Wang, L. (2018). Plant responses to pathogen attack: small RNAs in focus. Int J Mol Sci 19, 515.
Ismayil, A., Yang, M., Haxim, Y., Wang, Y., Li, J., Han, L., Wang, Y., Zheng, X., Wei, X., Nagalakshmi, U., et al. (2020a). *Cotton leaf curl Multan virus* βC1 protein induces autophagy by disrupting the interaction of autophagy-related protein 3 with glyceraldehyde-3-phosphate dehydrogenases. Plant Cell 32, 1124–1135.
Ismayil, A., Yang, M., and Liu, Y. (2020b). Role of autophagy during plant-virus interactions. Semin Cell Dev Biol 101, 36–40.
Jia, D., Liang, Q., Chen, H., Liu, H., Li, G., Zhang, X., Chen, Q., Wang, A., and Wei, T. (2023). Autophagy mediates a direct synergistic interaction during co-transmission of two distinct arboviruses by insect vectors. Sci China Life Sci 66, 1665–1681.
Jiang, L., Lu, Y., Zheng, X., Yang, X., Chen, Y., Zhang, T., Zhao, X., Wang, S., Zhao, X., Song, X., et al. (2020). The plant protein NbP3IP directs degradation of *Rice stripe virus* p3 silencing suppressor protein to limit virus infection through interaction with the autophagy-related protein NbATG8. New Phytol 229, 1036–1051.
Jiang, L., Qian, D., Zheng, H., Meng, L.Y., Chen, J., Le, W.J., Zhou, T., Zhou, Y.J., Wei, C.H., and Li, Y. (2012). RNA-dependent RNA polymerase 6 of rice (*Oryza sativa*) plays role in host defense against negative-strand RNA virus, Rice stripe virus. Virus Res 163, 512–519.
Jin, L., Qin, Q., Wang, Y., Pu, Y., Liu, L., Wen, X., Ji, S., Wu, J., Wei, C., Ding, B., et al. (2016). Rice dwarf virus P2 protein hijacks auxin signaling by directly targeting the rice OsIAA10 protein, enhancing viral infection and disease development. PLoS Pathog 12, e1005847.
Jin, Y., Zhao, J.H., and Guo, H.S. (2021). Recent advances in understanding plant antiviral RNAi and viral suppressors of RNAi. Curr Opin Virol 46, 65–72.
Jones, J.D.G., and Dangl, J.L. (2006). The plant immune system. Nature 444, 323–329.
Jones, J.D.G., Staskawicz, B.J., and Dangl, J.L. (2024). The plant immune system: from discovery to deployment. Cell 187, 2095–2116.
Kan, J., Cai, Y., Cheng, C., Chen, S., Jiang, C., He, Z., and Yang, P. (2023). CRISPR/Cas9-guided knockout of *eIF4E* improves *Wheat yellow mosaic virus* resistance without yield penalty. Plant Biotechnol J 21, 893–895.
Kang, B.-., Yeam, I., Frantz, J.D., Murphy, J.F., and Jahn, M.M. (2005). The *pvr1* locus in *Capsicum* encodes a translation initiation factor eIF4E that interacts with *Tobacco etch virus* VPg. Plant J 42, 392–405.
Kapoor, M., Arora, R., Lama, T., Nijhawan, A., Khurana, J.P., Tyagi, A.K., and Kapoor, S. (2008). Genome-wide identification, organization and phylogenetic analysis of Dicer-like, Argonaute and RNA-dependent RNA Polymerase gene families and their expression analysis during reproductive development and stress in rice. BMC Genomics 9, 451.
Kourelis, J., Marchal, C., Posbeyikian, A., Harant, A., and Kamoun, S. (2023). NLR immune receptor–nanobody fusions confer plant disease resistance. Science 379, 934–939.
Lanfermeijer, F.C., Warmink, J., and Hille, J. (2005). The products of the broken Tm-2 and the durable Tm-22 resistance genes from tomato differ in four amino acids. J Exp Bot 56, 2925–2933.
Leon-Reyes, A., Van der Does, D., De Lange, E.S., Delker, C., Wasternack, C., Van Wees, S.C.M., Ritsema, T., and Pieterse, C.M.J. (2010). Salicylate-mediated suppression of jasmonate-responsive gene expression in *Arabidopsis* is targeted downstream of the jasmonate biosynthesis pathway. Planta 232, 1423–1432.
Leyser, O. (2018). Auxin signaling. Plant Physiol 176, 465–479.
Li, F., Pignatta, D., Bendix, C., Brunkard, J.O., Cohn, M.M., Tung, J., Sun, H., Kumar, P., and Baker, B. (2011). MicroRNA regulation of plant innate immune receptors. Proc Natl Acad Sci USA 109, 1790–1795.
Li, F., Qiao, R., Wang, Z., Yang, X., and Zhou, X. (2022a). Occurrence and distribution of geminiviruses in China. Sci China Life Sci 65, 1498–1503.
Li, F., and Wang, A. (2018). RNA decay is an antiviral defense in plants that is counteracted by viral RNA silencing suppressors. PLoS Pathog 14, e1007228.
Li, F., and Wang, A. (2019). RNA-targeted antiviral immunity: more than just RNA silencing. Trends Microbiol 27, 792–805.
Li, F., Zhang, C., Li, Y., Wu, G., Hou, X., Zhou, X., and Wang, A. (2018). Beclin1 restricts RNA virus infection in plants through suppression and degradation of the viral polymerase. Nat Commun 9, 1268.
Li, F., Zhang, C., Tang, Z., Zhang, L., Dai, Z., Lyu, S., Li, Y., Hou, X., Bernards, M., and Wang, A. (2020). A plant RNA virus activates selective autophagy in a UPR-dependent manner to promote virus infection. New Phytol 228, 622–639.
Li, L., Verstraeten, I., Roosjen, M., Takahashi, K., Rodriguez, L., Merrin, J., Chen, J., Shabala, L., Smet, W., Ren, H., et al. (2021a). Cell surface and intracellular auxin signalling for H+ fluxes in root growth. Nature 599, 273–277.
Li, L., Zhang, H., Chen, C., Huang, H., Tan, X., Wei, Z., Li, J., Yan, F., Zhang, C., Chen, J., et al. (2021b). A class of independently evolved transcriptional repressors in plant RNA viruses facilitates viral infection and vector feeding. Proc Natl Acad Sci USA 118, e2016673118.
Li, L., Zhang, H., Yang, Z., Wang, C., Li, S., Cao, C., Yao, T., Wei, Z., Li, Y., Chen, J., et al. (2022b). Independently evolved viral effectors convergently suppress DELLA protein SLR1-mediated broad-spectrum antiviral immunity in rice. Nat Commun 13, 6920.
Li, X., Liu, C.X., Xue, W., Zhang, Y., Jiang, S., Yin, Q.F., Wei, J., Yao, R.W., Yang, L.,

and Chen, L.L. (2017). Coordinated circRNA biogenesis and function with NF90/NF110 in viral infection. Mol Cell 67, 214–227.e7.
Li, Z., Yang, X., Li, W., Wen, Z., Duan, J., Jiang, Z., Zhang, D., Xie, X., Wang, X., Li, F., et al. (2022c). SAMDC3 enhances resistance to *Barley stripe mosaic virus* by promoting the ubiquitination and proteasomal degradation of viral γb protein. New Phytol 234, 618–633.
Liu, C.X., Li, X., Nan, F., Jiang, S., Gao, X., Guo, S.K., Xue, W., Cui, Y., Dong, K., Ding, H., et al. (2019). Structure and degradation of circular RNAs regulate PKR activation in innate immunity. Cell 177, 865–880.e21.
Liu, L., and Chen, X. (2016). RNA quality control as a key to suppressing RNA silencing of endogenous genes in plants. Mol Plant 9, 826–836.
Liu, L., Jin, L., Huang, X., Geng, Y., Li, F., Qin, Q., Wang, R., Ji, S., Zhao, S., Xie, Q., et al. (2014a). OsRFPH2-10, a RING-H2 finger E3 ubiquitin ligase, is involved in rice antiviral defense in the early stages of rice dwarf virus infection. Mol Plant 7, 1057–1060.
Liu, Q., Deng, S., Liu, B., Tao, Y., Ai, H., Liu, J., Zhang, Y., Zhao, Y., and Xu, M. (2020). A helitron-induced RabGDIα variant causes quantitative recessive resistance to maize rough dwarf disease. Nat Commun 11, 495.
Liu, Q., Liu, H., Gong, Y., Tao, Y., Jiang, L., Zuo, W., Yang, Q., Ye, J., Lai, J., Wu, J., et al. (2017). An atypical thioredoxin imparts early resistance to sugarcane mosaic virus in maize. Mol Plant 10, 483–497.
Liu, B., Li, J., and Cairns, M.J. (2014b). Identifying miRNAs, targets and functions. Brief BioInf 15, 1–19.
Lozano-Durán, R., Rosas-Díaz, T., Gusmaroli, G., Luna, A.P., Taconnat, L., Deng, X.W., and Bejarano, E.R. (2011). Geminiviruses subvert ubiquitination by altering CSN-mediated derubylation of SCF E3 ligase complexes and inhibit jasmonate signaling in *Arabidopsis thaliana*. Plant Cell 23, 1014–1032.
Ma, J., Hou, X., Xiao, D., Qi, L., Wang, F., Sun, F., and Wang, Q. (2010). Cloning and characterization of the BcTuR3 gene related to resistance to Turnip mosaic virus (TuMV) from non-heading Chinese cabbage. Plant Mol Biol Rep 28, 588–596.
May, J.P., Johnson, P.Z., Ilyas, M., Gao, F., Simon, A.E., and Palese, P. (2020). The multifunctional long-distance movement protein of *Pea Enation Mosaic Virus 2* protects viral and host transcripts from nonsense-mediated decay. mBio 11, e00204–20.
Meshi, T., Motoyoshi, F., Maeda, T., Yoshiwoka, S., Watanabe, H., and Okada, Y. (1989). Mutations in the tobacco mosaic virus 30-kD protein gene overcome Tm-2 resistance in tomato.. Plant Cell 1, 515–522.
Mhamdi, A., and Van Breusegem, F. (2018). Reactive oxygen species in plant signaling. Annu Rev Plant Biol 69, 209–236.
Miricescu, A., Goslin, K., and Graciet, E. (2018). Ubiquitylation in plants: signaling hub for the integration of environmental signals. J Exp Bot 69, 4511–4527.
Mishina, K., Suzuki, T., Oono, Y., Yamashita, Y., Zhu, H., Ogawa, T., Ohta, M., Doman, K., Xu, W., Takahashi, D., et al. (2023). Wheat *Ym2* originated from *Aegilops sharonensis* and confers resistance to soil-borne *Wheat yellow mosaic virus* infection to the roots. Proc Natl Acad Sci USA 120, e2214968120.
Murphy, A.M., Zhou, T., and Carr, J.P. (2020). An update on salicylic acid biosynthesis, its induction and potential exploitation by plant viruses. Curr Opin Virol 42, 8–17.
Naderpour, M., Lund, O.S., Larsen, R., and Johansen, E. (2010). Potyviral resistance derived from cultivars of *Phaseolus vulgaris* carrying *bc-3* is associated with the homozygotic presence of a mutated *eIF4E* allele. Mol Plant Pathol 11, 255–263.
Nakasugi, K., Crowhurst, R.N., Bally, J., Wood, C.C., Hellens, R.P., Waterhouse, P.M., and Meyer, P. (2013). *De novo* transcriptome sequence assembly and analysis of RNA silencing genes of *Nicotiana benthamiana*. PLoS One 8, e59534.
Nicaise, V.Ã. (2014). Crop immunity against viruses: outcomes and future challenges. Front Plant Sci 5, 1–8.
Nicaise, V., German-Retana, S., Sanjuán, R., Dubrana, M.P., Mazier, M., Maisonneuve, B., Candresse, T., Caranta, C., and LeGall, O. (2003). The eukaryotic translation initiation factor 4E controls lettuce susceptibility to the potyvirus *Lettuce mosaic virus*. Plant Physiol 132, 1272–1282.
Nieto, C., Morales, M., Orjeda, G., Clepet, C., Monfort, A., Sturbois, B., Puigdomènech, P., Pitrat, M., Caboche, M., Dogimont, C., et al. (2006). An *eIF4E* allele confers resistance to an uncapped and non-polyadenylated RNA virus in melon. Plant J 48, 452–462.
Ning, Y., Liu, W., and Wang, G.L. (2017). Balancing immunity and yield in crop plants. Trends Plant Sci 22, 1069–1079.
Pasin, F., Shan, H., García, B., Müller, M., San León, D., Ludman, M., Fresno, D.H., Fátyol, K., Munné-Bosch, S., Rodrigo, G., et al. (2020). Abscisic acid connects phytohormone signaling with RNA metabolic pathways and promotes an antiviral response that is evaded by a self-controlled RNA virus. Plant Commun 1, 100099.
Pidon, H., Ghesquière, A., Chéron, S., Issaka, S., Hébrard, E., Sabot, F., Kolade, O., Silué, D., and Albar, L. (2017). Fine mapping of RYMV3: a new resistance gene to Rice yellow mottle virus from *Oryza glaberrima*. Theor Appl Genet 130, 807–818.
Qi, J., Wang, J., Gong, Z., and Zhou, J.M. (2017). Apoplastic ROS signaling in plant immunity. Curr Opin Plant Biol 38, 92–100.
Qi, X., Bao, F.S., and Xie, Z. (2009). Correction: small RNA deep sequencing reveals role for *Arabidopsis thaliana* RNA-dependent RNA polymerases in viral siRNA biogenesis. PLoS One 4, 1.
Qian, Y., Cheng, Y., Cheng, X., Jiang, H., Zhu, S., and Cheng, B. (2011). Identification and characterization of dicer-like, argonaute and RNA-dependent RNA polymerase gene families in maize. Plant Cell Rep 30, 1347–1363.
Qin, L., Mo, N., Muhammad, T., and Liang, Y. (2018). Genome-wide analysis of DCL, AGO, and RDR gene families in pepper (*Capsicum Annuum* L.). Int J Mol Sci 19, 1038.
Qin, Q., Li, G., Jin, L., Huang, Y., Wang, Y., Wei, C., Xu, Z., Yang, Z., Wang, H., Li, Y., et al. (2020). Auxin response factors (ARFs) differentially regulate rice antiviral immune response against rice dwarf virus. PLoS Pathog 16, e1009118.
Rairdan, G.J., Collier, S.M., Sacco, M.A., Baldwin, T.T., Boettrich, T., and Moffett, P. (2008). The coiled-coil and nucleotide binding domains of the potato Rx disease resistance protein function in pathogen recognition and signaling. Plant Cell 20, 739–751.
Ramirez-Prado, J.S., Abulfaraj, A.A., Rayapuram, N., Benhamed, M., and Hirt, H. (2018). Plant immunity: from signaling to epigenetic control of defense. Trends Plant Sci 23, 833–844.
Ren, B., Guo, Y., Gao, F., Zhou, P., Wu, F., Meng, Z., Wei, C., and Li, Y. (2010). Multiple functions of *Rice Dwarf Phytoreovirus* Pns10 in suppressing systemic RNA silencing. J Virol 84, 12914–12923.
Ren, Y., Tao, X., Li, D., Yang, X., and Zhou, X. (2022). *ty-5* confers broad-spectrum resistance to geminiviruses. Viruses 14, 1804.
Robert-Seilaniantz, A., Grant, M., and Jones, J.D.G. (2011). Hormone crosstalk in plant disease and defense: more than just JASMONATE-SALICYLATE antagonism. Annu Rev Phytopathol 49, 317–343.
de Ronde, D., Butterbach, P., and Kormelink, R. (2014). Dominant resistance against plant viruses. Front Plant Sci 5, 307.
Ruffel, S., Gallois, J.L., Lesage, M.L., and Caranta, C. (2005). The recessive potyvirus resistance gene *pot-1* is the tomato orthologue of the pepper pvr2-eIF4E gene. Mol Genet Genomics 274, 346–353.
Ruffel, S., Gallois, J.L., Moury, B., Robaglia, C., Palloix, A., and Caranta, C. (2006). Simultaneous mutations in translation initiation factors eIF4E and eIF(iso)4E are required to prevent pepper veinal mottle virus infection of pepper. J Gen Virol 87, 2089–2098.
Seo, Y., Jeon, J., Rojas, M.R., and Gilbertson, R.L. (2007). Characterization of a novel Toll/interleukin-1 receptor (TIR)-TIR gene differentially expressed in common bean (*Phaseolus vulgaris* cv. Othello) undergoing a defence response to the geminivirus *Bean dwarf mosaic virus*. Mol Plant Pathol 8, 151–162.
Seo, Y.S., Rojas, M.R., Lee, J.Y., Lee, S.W., Jeon, J.S., Ronald, P., Lucas, W.J., and Gilbertson, R.L. (2006). A viral resistance gene from common bean functions across plant families and is up-regulated in a non-virus-specific manner. Proc Natl Acad Sci USA 103, 11856–11861.
Sett, S., Prasad, A., and Prasad, M. (2022). Resistance genes on the verge of plant–virus interaction. Trends Plant Sci 27, 1242–1252.
Sharma, N., Sahu, P.P., Prasad, A., Muthamilarasan, M., Waseem, M., Khan, Y., Thakur, J.K., Chakraborty, S., and Prasad, M. (2021). The *Sw5a* gene confers resistance to ToLCNDV and triggers an HR response after direct AC4 effector recognition. Proc Natl Acad Sci USA 118, e2101833118.
Shen, Q., Hu, T., Bao, M., Cao, L., Zhang, H., Song, F., Xie, Q., and Zhou, X. (2016). Tobacco RING E3 ligase NtRFP1 mediates ubiquitination and proteasomal degradation of a geminivirus-encoded βC1. Mol Plant 9, 911–925.
Shen, X., Yan, Z., Wang, X., Wang, Y., Arens, M., Du, Y., Visser, R.G.F., Kormelink, R., Bai, Y., and Wolters, A.M.A. (2020). The NLR protein encoded by the resistance gene *ty-2* is triggered by the replication-associated protein Rep/C1 of tomato yellow leaf curl virus. Front Plant Sci 11, 545306.
Spassova, M.I., Prins, T.W., Folkertsma, R.T., Klein-Lankhorst, R.M., Hille, J., Goldbach, R.W., and Prins, M. (2001). The tomato gene *Sw5* is a member of the coiled coil, nucleotide binding, leucine-rich repeat class of plant resistance genes and confers resistance to TSWV in tobacco. Mol Breeding 7, 151–161.
Stein, N., Perovic, D., Kumlehn, J., Pellio, B., Stracke, S., Streng, S., Ordon, F., and Graner, A. (2005). The eukaryotic translation initiation factor 4E confers multiallelic recessive *Bymovirus* resistance in *Hordeum vulgare* (L.). Plant J 42, 912–922.
Stoecklin, G., and Mühlemann, O. (2013). RNA decay mechanisms: specificity through diversity. Biochim Biophys Acta (BBA)-Gene Regulatory Mech 1829, 487–490.
Szádeczky-Kardoss, I., Csorba, T., Auber, A., Schamberger, A., Nyikó, T., Taller, J., Orbán, T.I., Burgyán, J., and Silhavy, D. (2018). The nonstop decay and the RNA silencing systems operate cooperatively in plants. Nucleic Acids Res 46, 4632–4648.
Takahashi, H., Suzuki, M., Natsuaki, K., Shigyo, T., Hino, K., Teraoka, T., Hosokawa,

D., and Ehara, Y. (2001). Mapping the virus and host genes involved in the resistance response in cucumber mosaic virus-infected *Arabidopsis thaliana*. Plant Cell Physiol 42, 340–347.
Tan, X., Zhang, H., Yang, Z., Wei, Z., Li, Y., Chen, J., Sun, Z., and Dinesh-Kumar, S.P. (2022). NF-YA transcription factors suppress jasmonic acid-mediated antiviral defense and facilitate viral infection in rice. PLoS Pathog 18, e1010548.
Tenllado, F., and Canto, T. (2020). Effects of a changing environment on the defenses of plants to viruses. Curr Opin Virol 42, 40–46.
Tian, M., Sasvari, Z., Gonzalez, P.A., Friso, G., Rowland, E., Liu, X.M., van Wijk, K.J., Nagy, P.D., and Klessig, D.F. (2015). Salicylic acid inhibits the replication of *Tomato bushy stunt virus* by directly targeting a host component in the replication complex. Mol Plant Microbe Interact 28, 379–386.
Tomita, R., Sekine, K.T., Mizumoto, H., Sakamoto, M., Murai, J., Kiba, A., Hikichi, Y., Suzuki, K., and Kobayashi, K. (2011). Genetic basis for the hierarchical interaction between *Tobamovirus* spp. and *L* resistance gene alleles from different pepper species. Mol Plant Microbe Interact 24, 108–117.
Tong, X., Liu, S.Y., Zou, J.Z., Zhao, J.J., Zhu, F.F., Chai, L.X., Wang, Y., Han, C., and Wang, X.B. (2021). A small peptide inhibits siRNA amplification in plants by mediating autophagic degradation of SGS3/RDR6 bodies. EMBO J 40, e108050.
Tong, X., Zhao, J.J., Feng, Y.L., Zou, J.Z., Ye, J., Liu, J., Han, C., Li, D., and Wang, X.B. (2023). A selective autophagy receptor VISP1 induces symptom recovery by targeting viral silencing suppressors. Nat Commun 14, 3852.
Törk, L., Moffatt, C.B., Bernhardt, T.G., Garner, E.C., and Kahne, D. (2023). Single-molecule dynamics show a transient lipopolysaccharide transport bridge. Nature 623, 814–819.
Urayama, S., Moriyama, H., Aoki, N., Nakazawa, Y., Okada, R., Kiyota, E., Miki, D., Shimamoto, K., and Fukuhara, T. (2010). Knock-down of OsDCL2 in rice negatively affects maintenance of the endogenous dsRNA virus, *Oryza sativa* endornavirus. Plant Cell Physiol 51, 58–67.
Vallejos, C.E., Astua-Monge, G., Jones, V., Plyler, T.R., Sakiyama, N.S., and Mackenzie, S.A. (2006). Genetic and molecular characterization of the *I* locus of *Phaseolus vulgaris*. Genetics 172, 1229–1242.
Verlaan, M.G., Hutton, S.F., Ibrahem, R.M., Kormelink, R., Visser, R.G.F., Scott, J.W., Edwards, J.D., Bai, Y., and McDowell, J.M. (2013). The tomato yellow leaf curl virus resistance genes *ty-1* and *ty-3* are allelic and code for DFDGD-class RNA–dependent RNA polymerases. PLoS Genet 9, e1003399.
Vidal, S., Cabrera, H., Andersson, R.A., Fredriksson, A., and Valkonen, J.P.T. (2002). Potato gene *Y-1* is an *N* gene homolog that confers cell death upon infection with *potato virus Y*. Mol Plant Microbe Interact 15, 717–727.
Vogel, F., Hofius, D., and Sonnewald, U. (2007). Intracellular trafficking of *Potato Leafroll Virus* movement protein in transgenic *Arabidopsis*. Traffic 8, 1205–1214.
Wang, A. (2015). Dissecting the molecular network of virus-plant interactions: the complex roles of host factors. Annu Rev Phytopathol 53, 45–66.
Wang, H., Chao, S., Yan, Q., Zhang, S., Chen, G., Mao, C., Hu, Y., Yu, F., Wang, S., Lv, L., et al. (2024). Genetic diversity of RNA viruses infecting invertebrate pests of rice. Sci China Life Sci 67, 175–187.
Wang, H., Jiao, X., Kong, X., Hamera, S., Wu, Y., Chen, X., Fang, R., and Yan, Y. (2016). A signaling cascade from miR444 to RDR1 in rice antiviral RNA silencing pathway. Plant Physiol 170, 2365–2377.
Wang, J., Chen, T., Han, M., Qian, L., Li, J., Wu, M., Han, T., Cao, J., Nagalakshmi, U., Rathjen, J.P., et al. (2020). Plant NLR immune receptor Tm-22 activation requires NB-ARC domain-mediated self-association of CC domain. PLoS Pathog 16, e1008475.
Wang, J., Hu, M., Wang, J., Qi, J., Han, Z., Wang, G., Qi, Y., Wang, H.W., Zhou, J.M., and Chai, J. (2019). Reconstitution and structure of a plant NLR resistosome conferring immunity. Science 364, eaav5870.
Wang, Q., Liu, Y., He, J., Zheng, X., Hu, J., Liu, Y., Dai, H., Zhang, Y., Wang, B., Wu, W., et al. (2014). STV11 encodes a sulphotransferase and confers durable resistance to rice stripe virus. Nat Commun 5, 4768.
Wang, W., Ma, S., Hu, P., Ji, Y., and Sun, F. (2021a). Genome editing of rice eIF4G loci confers partial resistance to rice black-streaked dwarf virus. Viruses 13, 2100.
Wang, X.B., Wu, Q., Ito, T., Cillo, F., Li, W.X., Chen, X., Yu, J.L., and Ding, S.W. (2010). RNAi-mediated viral immunity requires amplification of virus-derived siRNAs in *Arabidopsis thaliana*. Proc Natl Acad Sci USA 107, 484–489.
Wang, Y., Gong, Q., Wu, Y., Huang, F., Ismayil, A., Zhang, D., Li, H., Gu, H., Ludman, M., Fátyol, K., et al. (2021b). A calmodulin-binding transcription factor links calcium signaling to antiviral RNAi defense in plants. Cell Host Microbe 29, 1393–1406.e7.
Wasternack, C., and Hause, B. (2013). Jasmonates: biosynthesis, perception, signal transduction and action in plant stress response, growth and development. An update to the 2007 review in Annals of Botany. Ann Bot 111, 1021–1058.
Wei, T., and Li, Y. (2016). Rice reoviruses in insect vectors. Annu Rev Phytopathol 54, 99–120.
Weijers, D., and Wagner, D. (2016). Transcriptional responses to the auxin hormone. Annu Rev Plant Biol 67, 539–574.
Wen, R.H., Khatabi, B., Ashfield, T., Maroof, M.A.S., and Hajimorad, M.R. (2013). The HC-Pro and P3 cistrons of an avirulent *Soybean mosaic virus* are recognized by different resistance genes at the complex *Rsv1* locus. Mol Plant Microbe Interact 26, 203–215.
Wen, X., Chen, Z., Yang, Z., Wang, M., Jin, S., Wang, G., Zhang, L., Wang, L., Li, J., Saeed, S., et al. (2023). A comprehensive overview of cotton genomics, biotechnology and molecular biological studies. Sci China Life Sci 66, 2214–2256.
Whitham, S., Dinesh-Kumar, S.P., Choi, D., Hehl, R., Corr, C., and Baker, B. (1994). The product of the tobacco mosaic virus resistance gene *N*: similarity to toll and the interleukin-1 receptor. Cell 78, 1101–1115.
Wu, D., Qi, T., Li, W.X., Tian, H., Gao, H., Wang, J., Ge, J., Yao, R., Ren, C., Wang, X. B., et al. (2017a). Viral effector protein manipulates host hormone signaling to attract insect vectors. Cell Res 27, 402–415.
Wu, H., Qu, X., Dong, Z., Luo, L., Shao, C., Forner, J., Lohmann, J.U., Su, M., Xu, M., Liu, X., et al. (2020). WUSCHEL triggers innate antiviral immunity in plant stem cells. Science 370, 227–231.
Wu, J., Yang, G., Zhao, S., Zhang, S., Qin, B., Zhu, Y., Xie, H., Chang, Q., Wang, L., Hu, J., et al. (2022a). Current rice production is highly vulnerable to insect-borne viral diseases. Natl Sci Rev 9, nwac131.
Wu, J., Yang, R., Yang, Z., Yao, S., Zhao, S., Wang, Y., Li, P., Song, X., Jin, L., Zhou, T., et al. (2017b). ROS accumulation and antiviral defence control by microRNA528 in rice. Nat Plants 3, 16203.
Wu, J., Yang, Z., Wang, Y., Zheng, L., Ye, R., Ji, Y., Zhao, S., Ji, S., Liu, R., Xu, L., et al. (2015). Viral-inducible Argonaute18 confers broad-spectrum virus resistance in rice by sequestering a host microRNA. eLife 4, e05733.
Wu, J., Zhang, Y., Li, F., Zhang, X., Ye, J., Wei, T., Li, Z., Tao, X., Cui, F., Wang, X., et al. (2024). Plant virology in the 21st century in China: Recent advances and future directions. JIPB 66, 579–622.
Wu, K., Fu, Y., Ren, Y., Liu, L., Zhang, X., and Ruan, M. (2023a). Turnip crinkle virus-encoded suppressor of RNA silencing suppresses mRNA decay by interacting with *Arabidopsis* XRN4. Plant J 116, 744–755.
Wu, Q., Cui, Y., Jin, X., Wang, G., Yan, L., Zhong, C., Yu, M., Li, W., Wang, Y., Wang, L., et al. (2022b). The CC–NB–LRR protein BSR1 from *Brachypodium* confers resistance to *Barley stripe mosaic virus* in gramineous plants by recognising TGB1 movement protein. New Phytol 236, 2233–2248.
Wu, Q., Tong, C., Chen, Z., Huang, S., Zhao, X., Hong, H., Li, J., Feng, M., Wang, H., Xu, M., et al. (2023b). NLRs derepress MED10b- and MED7-mediated repression of jasmonate-dependent transcription to activate immunity. Proc Natl Acad Sci USA 120, e2302226120.
Xia, X.J., Zhou, Y.H., Shi, K., Zhou, J., Foyer, C.H., and Yu, J.Q. (2015). Interplay between reactive oxygen species and hormones in the control of plant development and stress tolerance. J Exp Bot 66, 2839–2856.
Xie, K., Li, L., Zhang, H., Wang, R., Tan, X., He, Y., Hong, G., Li, J., Ming, F., Yao, X., et al. (2018). Abscisic acid negatively modulates plant defence against rice black-streaked dwarf virus infection by suppressing the jasmonate pathway and regulating reactive oxygen species levels in rice. Plant Cell Environ 41, 2504–2514.
Xie, Z., Fan, B., Chen, C., and Chen, Z. (2001). An important role of an inducible RNA-dependent RNA polymerase in plant antiviral defense. Proc Natl Acad Sci USA 98, 6516–6521.
Xu, Z., Zhou, Z., Cheng, Z., Zhou, Y., Wang, F., Li, M., Li, G., Li, W., Du, Q., Wang, K., et al. (2023). A transcription factor ZmGLK36 confers broad resistance to maize rough dwarf disease in cereal crops. Nat Plants 9, 1720–1733.
Yang, D.L., Yang, Y., and He, Z. (2013a). Roles of plant hormones and their interplay in rice immunity. Mol Plant 6, 675–685.
Yang, J., Duan, G., Li, C., Liu, L., Han, G., Zhang, Y., and Wang, C. (2019a). The crosstalks between jasmonic acid and other plant hormone signaling highlight the involvement of jasmonic acid as a core component in plant response to biotic and abiotic stresses. Front Plant Sci 10, 1349.
Yang, J.H., Seo, H.H., Han, S.J., Yoon, E.K., Yang, M.S., and Lee, W.S. (2008). Phytohormone abscisic acid control RNA-dependent RNA polymerase 6 gene expression and post-transcriptional gene silencing in rice cells. Nucleic Acids Res 36, 1220–1226.
Yang, M., Ismayil, A., Gao, T., Ye, Z., Yue, N., Wu, J., Zheng, X., Li, Y., Wang, Y., Hong, Y., et al. (2023). *Cotton leaf curl Multan virus* C4 protein suppresses autophagy to facilitate viral infection. Plant Physiol 193, 708–720.
Yang, M., Ismayil, A., Jiang, Z., Wang, Y., Zheng, X., Yan, L., Hong, Y., Li, D., and Liu, Y. (2022). A viral protein disrupts vacuolar acidification to facilitate virus infection in plants. EMBO J 41, e108713.
Yang, M., Li, Z., Zhang, K., Zhang, X., Zhang, Y., Wang, X., Han, C., Yu, J., Xu, K., and Li, D. (2018a). Barley stripe mosaic virus γb interacts with glycolate oxidase and inhibits peroxisomal ROS production to facilitate virus infection. Mol Plant 11, 338–341.

Yang, M., and Liu, Y. (2022). Autophagy in plant viral infection. FEBS Lett 596, 2152–2162.
Yang, M., Zhang, Y., Xie, X., Yue, N., Li, J., Wang, X.B., Han, C., Yu, J., Liu, Y., and Li, D. (2018b). *Barley stripe mosaic virus* γb protein subverts autophagy to promote viral infection by disrupting the ATG7-ATG8 Interaction. Plant Cell 30, 1582–1595.
Yang, P., Lüpken, T., Habekuss, A., Hensel, G., Steuernagel, B., Kilian, B., Ariyadasa, R., Himmelbach, A., Kumlehn, J., Scholz, U., et al. (2014). *PROTEIN DISULFIDE ISOMERASE LIKE 5-1* is a susceptibility factor to plant viruses. Proc Natl Acad Sci USA 111, 2104–2109.
Yang, P., Perovic, D., Habekuß, A., Zhou, R., Graner, A., Ordon, F., and Stein, N. (2013b). Gene-based high-density mapping of the gene rym7 conferring resistance to barley mild mosaic virus (BaMMV). Mol Breeding 32, 27–37.
Yang, R., Li, P., Mei, H., Wang, D., Sun, J., Yang, C., Hao, L., Cao, S., Chu, C., Hu, S., et al. (2019b). Fine-tuning of MiR528 accumulation modulates flowering time in rice. Mol Plant 12, 1103–1113.
Yang, Z., Huang, Y., Yang, J., Yao, S., Zhao, K., Wang, D., Qin, Q., Bian, Z., Li, Y., Lan, Y., et al. (2020). Jasmonate signaling enhances RNA silencing and antiviral defense in rice. Cell Host Microbe 28, 89–103.e8.
Yang, Z., and Li, Y. (2018). Dissection of RNAi-based antiviral immunity in plants. Curr Opin Virol 32, 88–99.
Yao, S., Yang, Z., Yang, R., Huang, Y., Guo, G., Kong, X., Lan, Y., Zhou, T., Wang, H., Wang, W., et al. (2019). Transcriptional regulation of miR528 by OsSPL9 orchestrates antiviral response in rice. Mol Plant 12, 1114–1122.
Yin, J., Wang, L., Jin, T., Nie, Y., Liu, H., Qiu, Y., Yang, Y., Li, B., Zhang, J., Wang, D., et al. (2021). A cell wall-localized NLR confers resistance to soybean mosaic virus by recognizing viral-encoded cylindrical inclusion protein. Mol Plant 14, 1881–1900.
Ying, X.B., Dong, L., Zhu, H., Duan, C.G., Du, Q.S., Lv, D.Q., Fang, Y.Y., Garcia, J.A., Fang, R.X., and Guo, H.S. (2010). RNA-dependent RNA polymerase 1 from *Nicotiana tabacum* suppresses RNA silencing and enhances viral infection in *Nicotiana benthamiana*. Plant Cell 22, 1358–1372.
Yu, D., Fan, B., MacFarlane, S.A., and Chen, Z. (2003). Analysis of the involvement of an inducible *Arabidopsis* RNA-dependent RNA polymerase in antiviral defense. Mol Plant Microbe Interact 16, 206–216.
Yuan, W., Jiang, T., Du, K., Chen, H., Cao, Y., Xie, J., Li, M., Carr, J.P., Wu, B., Fan, Z., et al. (2019). Maize phenylalanine ammonia-lyases contribute to resistance to *Sugarcane mosaic virus* infection, most likely through positive regulation of salicylic acid accumulation. Mol Plant Pathol 20, 1365–1378.
Zeng, L.R., Vega-Sánchez, M.E., Zhu, T., and Wang, G.L. (2006). Ubiquitination-mediated protein degradation and modification: an emerging theme in plant-microbe interactions. Cell Res 16, 413–426.
Zhang, C., Ding, Z., Wu, K., Yang, L., Li, Y., Yang, Z., Shi, S., Liu, X., Zhao, S., Yang, Z., et al. (2016). Suppression of jasmonic acid-mediated defense by viral-inducible microRNA319 facilitates virus infection in rice. Mol Plant 9, 1302–1314.
Zhang, C., Wei, Y., Xu, L., Wu, K.C., Yang, L., Shi, C.N., Yang, G.Y., Chen, D., Yu, F.F., Xie, Q., et al. (2020a). A bunyavirus-inducible ubiquitin ligase targets RNA polymerase IV for degradation during viral pathogenesis in rice. Mol Plant 13, 836–850.
Zhang, D., Gao, Z., Zhang, H., Yang, Y., Yang, X., Zhao, X., Guo, H., Nagalakshmi, U., Li, D., Dinesh-Kumar, S.P., et al. (2023a). The MAPK-Alfin-like 7 module negatively regulates ROS scavenging genes to promote NLR-mediated immunity. Proc Natl Acad Sci USA 120, e2214750120.
Zhang, H., Li, L., He, Y., Qin, Q., Chen, C., Wei, Z., Tan, X., Xie, K., Zhang, R., Hong, G., et al. (2020b). Distinct modes of manipulation of rice auxin response factor OsARF17 by different plant RNA viruses for infection. Proc Natl Acad Sci USA 117, 9112–9121.
Zhang, H., Tan, X., Li, L., He, Y., Hong, G., Li, J., Lin, L., Cheng, Y., Yan, F., Chen, J., et al. (2019). Suppression of auxin signalling promotes rice susceptibility to *Rice black streaked dwarf virus* infection. Mol Plant Pathol 20, 1093–1104.
Zhang, H., Wang, F., Song, W., Yang, Z., Li, L., Ma, Q., Tan, X., Wei, Z., Li, Y., Li, J., et al. (2023b). Different viral effectors suppress hormone-mediated antiviral immunity of rice coordinated by OsNPR1. Nat Commun 14, 3011.
Zhang, L., Zhang, F., Melotto, M., Yao, J., and He, S.Y. (2017). Jasmonate signaling and manipulation by pathogens and insects. J Exp Bot 68, 1371–1385.
Zhang, X., and Guo, H. (2017). mRNA decay in plants: Both quantity and quality matter. Curr Opin Plant Biol 35, 138–144.
Zhang, X., Wan, Q., Rui, P., Lu, Y., Sun, Z., Chen, J., Wang, Y., and Yan, F. (2023c). Rice stripe virus p2 protein interacts with ATG5 and is targeted for degradation by autophagy. Front Microbiol 14, 1191403.
Zhang, X., Zhu, Y., Liu, X., Hong, X., Xu, Y., Zhu, P., Shen, Y., Wu, H., Ji, Y., Wen, X., et al. (2015). Suppression of endogenous gene silencing by bidirectional cytoplasmic RNA decay in *Arabidopsis*. Science 348, 120–123.
Zhang, Z.J., Gao, Q., Fang, X.D., Ding, Z.H., Gao, D.M., Xu, W.Y., Cao, Q., Qiao, J.H., Yang, Y.Z., Han, C., et al. (2020c). CCR4, a RNA decay factor, is hijacked by a plant cytorhabdovirus phosphoprotein to facilitate virus replication. eLife 9, e53753.
Zhao, S., Hong, W., Wu, J., Wang, Y., Ji, S., Zhu, S., Wei, C., Zhang, J., and Li, Y. (2017). A viral protein promotes host SAMS1 activity and ethylene production for the benefit of virus infection. eLife 6, e27529.
Zhao, S., and Li, Y. (2021). Current understanding of the interplays between host hormones and plant viral infections. PLoS Pathog 17, e1009242.
Zhao, Y., Cao, X., Zhong, W., Zhou, S., Li, Z., An, H., Liu, X., Wu, R., Bohora, S., Wu, Y., et al. (2022). A viral protein orchestrates rice ethylene signaling to coordinate viral infection and insect vector-mediated transmission. Mol Plant 15, 689–705.
Zhou, F., Wu, G., Deng, W., Pu, Y., Wei, C., and Li, Y. (2007). Interaction of rice dwarf virus outer capsid P8 protein with rice glycolate oxidase mediates relocalization of P8. FEBS Lett 581, 34–40.
Zhou, W., Lozano-Torres, J.L., Blilou, I., Zhang, X., Zhai, Q., Smant, G., Li, C., and Scheres, B. (2019). A jasmonate signaling network activates root stem cells and promotes regeneration. Cell 177, 942–956.e14.
Zhu, H., and Guo, H.S. (2012). The role of virus-derived small interfering RNAs in RNA silencing in plants. Sci China Life Sci 55, 119–125.
Zhu, M., Jiang, L., Bai, B., Zhao, W., Chen, X., Li, J., Liu, Y., Chen, Z., Wang, B., Wang, C., et al. (2017). The intracellular immune receptor Sw-5b confers broad-spectrum resistance to tospoviruses through recognition of a conserved 21-amino acid viral effector epitope. Plant Cell 29, 2214–2232.
Zhu, S., Gao, F., Cao, X., Chen, M., Ye, G., Wei, C., and Li, Y. (2005). The rice dwarf virus P2 protein interacts with *ent*-kaurene oxidases *in vivo*, leading to reduced biosynthesis of gibberellins and rice dwarf symptoms. Plant Physiol 139, 1935–1945.