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Viral suppressors from members of the family *Closteroviridae* combating antiviral RNA silencing: a tale of a sophisticated arms race in host-pathogen interactions

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Abstract

RNA silencing is an evolutionarily homology-based gene inactivation mechanism and plays critical roles in plant immune responses to acute or chronic virus infections, which often pose serious threats to agricultural productions. Plant antiviral immunity is triggered by virus-derived small interfering RNAs (vsiRNAs) and functions to suppress virus further replication via a sequence-specific degradation manner. Through plant-virus arms races, many viruses have evolved specific protein(s), known as viral suppressors of RNA silencing (VSRs), to combat plant antiviral responses. Numerous reports have shown that VSRs can efficiently curb plant antiviral defense response via interaction with specific component(s) involved in the plant RNA silencing machinery. Members in the family *Closteroviridae* (closterovirids) are also known to encode VSRs to ensure their infections in plants. In this review, we will focus on the plant antiviral RNA silencing strategies, and the most recent developments on the multifunctional VSRs encoded by closterovirids. Additionally, we will highlight the molecular characters of phylogenetically-associated closterovirids, the interactions of these viruses with their host plants and transmission vectors, and epidemiology.

Keywords: Closterovirids, Antiviral RNA silencing, Viral pathogenicity, Plant immunity, Crop disease

Background

The viruses of the family *Closteroviridae* are characterized by their flexuous, exceptionally long filamentous, and non-enveloped particles with lengths of 950–2200 nm and diameters of 10–13 nm. These closterovirids are composed of positive-sense single-stranded RNA (+ssRNA), causing acute or chronic infections in plants and threatening agricultural production systems globally (Martelli 2019; Jones 2021). Generally, closterovirids have a common pattern of genomic organization that possesses variable numbers of open reading frames (ORFs). However, the presence of cellular heat-shock proteins HSP70 homology (HSP70h) with a duplicated and deviated form of coat protein (designated as minor coat protein or CPm) in genome are hallmarks of viruses in this family, distinguishing them from other plant viruses (Agranovsky 2016; Ruiz et al. 2018). Strikingly, their genomic expression strategy is predicated according to the proteolytic processing, such as a+1 translational/ribosomal frameshifting together with subgenomic mRNAs. Therefore, strong negative selection and recombination are primary factors influencing their genetic diversity (Rubio et al. 2013; Fuchs et al. 2020).

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Presently, according to the 2020 master species list-36 (MSL36) released by the International Committee on Taxonomy of Viruses (ICTV), the *Closteroviridae* family contains 4 genera (*Amplelovirus*, *Crinivirus*, *Closterovirus* and *Velavirus*) and 52 identified species, with 7 unassigned species (https://ictv.global/taxonomy). Most closterovirids have evolved from common ancestors and are transmitted through specific insect vectors (arthropods), such as mealybugs (*Ampelovirus*), aphids (*Closterovirus*), and whiteflies (*Crinivirus*). The vector for members of the genus *Velarivirus* is yet to be identified. However, there is merely an exception for mint vein banding-associated virus (MVBaV). MVBaV is an aphid-borne virus, which is phylogenetically distant from other members of the family (Martelli et al. 2012; Fuchs et al. 2020). The majority of closterovirids cause substantial disease epidemics in combined infections with other plant viruses that may result in synergistic effects. Moreover, the host range and environmental factors determining vector population dynamics have important epidemiological consequences (Tzanetakis et al. 2007, 2013; Quito-Avila et al. 2014).

To improve crop quality and quantity, a study of the progress of viral infection and defense strategies remains most significant. It is obvious from molecular characterization of plant viruses that virus infection process is coordinated with their restricted viral factors, through which viruses interact with host proteins and recruit biological processes crucial for their multiplication and translocation (Tatineni et al. 2012; Castillo-Gonzalez et al. 2015; Liu et al. 2021). On the other hand, plants develop a set of complex antiviral defense system to counter viral infection, including antiviral RNA silencing, systemic acquired resistance (SAR), hypersensitive responses (HR), DNA methylation, ubiquitin–proteasome system (UPS), and hormone signaling pathways such as salicylic acid (SA), jasmonic acid (JA), and ethylene (ET) (Huh et al. 2012; Mandadi and Scholthof 2013; Balint-Kurti 2019; Li and Wang 2019; Tirnaz and Batley 2019; Kamle et al. 2020; Dubiella and Serrano 2021; Zhao and Li 2021).

Recently, significant research has been done to understand the mechanisms and functions of antiviral RNA silencing as well as viral strategies to counter this defense (Jones and Dangl 2006; Shi et al. 2008; Sansregret et al. 2013; Csorba et al. 2015; Jin et al. 2021; Teixeira et al. 2021). Antiviral RNA silencing is a highly conserved regulatory mechanism of gene expression, which plays the most important role in different biological processes associated with host protection against viral infection. Plants recruit this mechanism as a shield against viruses by encoding abundant crucial components, such as RNA-dependent RNA polymerases (RdRps), Dicer ribonucleases (DCLs), Argonaute endonucleases (AGO), double-stranded RNA (dsRNA), and helicases (Lee and Carroll 2018; Muhammad et al. 2019; Lax et al. 2020). In response to this defense strategy, viruses have also evolved to produce various multifunctional proteins that act as VSRs to avoid being silenced and thus ensure successful infections (Daròs 2017; Gaffar and Koch 2019).

Our current review highlights recent progress and breakthroughs in plant-targeting VSRs together with particular strategies and mechanisms aimed at evading antiviral RNA silencing. It also demonstrates mechanistic insights into various VSR strategies by which closterovirids evade antiviral responses of host plants in order to execute successful viral infections. Closterovirids are presented here according to their evolutionary relatedness (Fig. 1) as members of each genus tend to have similar genomic organization, transmission vectors, and host ranges, but diverse modes of infection (Table 1).

Antiviral RNA silencing and plant immunity

Plants and microbial pathogens are fascinatingly engaged in a continuous battle for survival. Plant immunity to non-viral pathogens has revealed well-organized, multilayered, and sophisticated signaling pathways, which are triggered by the perception of diverse pathogen-associated molecular patterns (PAMPs) to initiate the first layer of defense. This fundamental defense mechanism (PAMP-triggered immunity, PTI) elucidates the entire co-evolutionary arms race between pathogen and host plant. For invading pathogens, they encode virulence factors, named as effectors, to suppress PTI (Shan et al. 2008; Calil and Fontes 2017; Gouveia et al. 2017; Teixeira et al. 2021). Therefore, plants respond to PTI suppression through resistance (R) proteins using a highly precise and effective type of immunity, known as effector-triggered immunity (ETI) (Cui et al. 2015; Hatsugai et al. 2017; Peng et al. 2018). Since this phenomenon represents protein-based rather than RNA-involved defense mechanisms, it was considered that PTI and ETI are remarkably independent of antiviral RNA silencing. The mechanism of antiviral RNA silencing in plants reveals a related process, termed as post-transcriptional gene silencing (PTGS). Recently, biological evidences have indicated that PTGS and transcriptional gene silencing (TGS), induced by endogenic small RNAs (sRNAs), are evolving as significantly crucial drivers of ETI and PTI signaling pathways, as well as R gene expression (Zhai et al. 2011; Baltusnikas et al. 2018; Tan et al. 2020; Sanan-Mishra et al. 2021). Furthermore, several classes of microorganisms, such as plant viruses, bacteria, and oomycetes, have been discovered to produce suppressor proteins as part of their virulence effectors, to suppress RNA silencing in host plants and cause disease (Navarro et al. 2008; He et al. 2019).

The mechanism and localization of antiviral RNA silencing

In plants, an antiviral silencing pathway is triggered by two discrete classes of sRNAs, including microRNAs (miRNAs) and small interfering RNAs (siRNAs), exerting diverse actions such as mRNA degradation, DNA methylation, and translational inhibition (Li and Wang 2019; Tan et al. 2020). Mechanistically, the antiviral RNA silencing model is divided into initiation, effector, and amplification phases (Zhang et al. 2015; Csorba and Burgyán 2016). Plant viruses, containing RNA genomes with defective regulatory stem-loop, are transcribed into complementary dsRNA replication intermediates through viral encoded RdRps. This dsRNA is designed as a virus-associated molecular pattern (VAMP), which is a form of PAMP. During the initiation phase, VAMPs are recognized and cleaved by dsRNA-specific RNases named as DCL enzymes (primarily by DCL4 and secondarily by DCL2), producing 21 to 24-nt double-stranded vsiRNA duplexes (Pumplin and Voinnet 2013). On the other hand, microRNA (*MIR*) genes encode pre-miRNAs, which are transcribed into primary miRNAs (pri-miRNAs) through RNA polymerase II (Pol II). Subsequently, stem-loop containing pri-miRNAs are processed into mature miRNA duplexes through the RNA III family enzyme DCL1. These miRNA and vsiRNA duplexes are stabilized at their 3′ end, mediated by the HUA Enhancer 1 (HEN1)-dependent methylation process (Burgyan and Havelda 2011; Rogers and Chen 2013; Zhang et al. 2015). Furthermore, stabilized vsiRNA and miRNA duplexes were unzipped and separated into two strands, termed as guide and passenger strand, respectively, by helicase. The guide strand is incorporated into AGO proteins, facilitating the formation of a complex (RNA-induced silencing complex, RISC), while the passenger strand is shattered (Vaucheret 2008; Zhang et al. 2015; Waheed et al. 2021). To complete the effector phase of antiviral RNA silencing, RISCs target vsiRNA or miRNA complementary mRNAs and trigger their PTGS via endonucleolytic cleavage or translational inhibition (Waterhouse and Helliwell 2003; Brodersen et al. 2008; Carbonell and Carrington 2015; Mengistu and Tenkegna 2021). For the amplification of antiviral RNA silencing, AGO-sliced products serve as templates for RdRp (RDR for cellular RdRps) complexes using RNA-helicases (SGS3 and SDE5), the cofactors of RDR-mediated pathways (Vaucheret 2006; Jauvion et al. 2010; Csorba et al. 2015; Tong et al. 2021).

In plant, antiviral RNA silencing spreads between adjacent cells and over long distances via plasmodesmata and phloem, respectively. In local spread of RNA silencing, most probably, silencing molecules (siRNAs, miRNAs) move beyond the production sites to 10–15 cells. However, the activation of local silencing signaling is independent of homologous transcripts. The activity of RDR6 and SDE3 is not essential for the synthesis or detection of silencing signals, but at least three *SILENCING MOVEMENT DEFICIENT* genes (SMD1-3) are mandatory for cell-to-cell movements (Voinnet 2005; Mermigka et al. 2016). Meanwhile, in the particular scenario of systemic spread of silencing, the amplification process requires RDR6, SDE3 or SDE5 to convert homologous transcripts into new dsRNAs in

(See figure on next page.)

Fig. 1 A phylogenetic tree demonstrating the linkages between species and genera within the family *Closteroviridae* on the basis of the amino acids sequence of RdRp. A multiple sequence alignment of RdRp amino acids sequence was performed using CLUSTALW. A phylogenetic tree was constructed with MEGA7 software using the neighbor-joining method and bootstrapped 1000 times. The numbers on nodes particularly signify bootstrapped confidence percentages. The abbreviations and accession numbers of closterovirids involved in this tree: Actinidia virus 1 (AcV-1; YP_009407919.1); Air potato virus 1 (AiPoV-1; AZB50207.1); Areca palm velarivirus 1 (APV-1; YP_009140432.1); Bean yellow disorder virus (BnYDV; YP_001816770.1); Beet pseudo yellows virus (BPYV; NP_940796.1); Blueberry virus A (BVA; YP_006638806.1); Blackberry vein banding-associated virus (BVBaV; YP_008411010.1); Beet yellow stunt virus (BYSV; AAC55659.2); Beet yellows virus (BYV; AAF14300.1); Blackberry yellow vein-associated virus (BYVaV; YP_227378.1); Cucurbit chlorotic yellows virus (CCYV; AFN61343.1); Cordyline virus 1 (CoV-1; YP_009506344.1); Cordyline virus 2 (CoV-2; AFJ05046.1); Cordyline virus 3 (CoV-3; AFJ05056.2); Cordyline virus 4 (CoV-4; AFJ05062.2); Carnation necrotic fleck virus (CNFV; YP_009506332.1); Citrus tristeza virus (CTV; ANA04448.1); Carrot yellow leaf virus (CYLV; YP_003075965.1); Cucurbit yellow stunting disorder virus (CYSDV; AAM73639.2); Diodia vein chlorosis virus (DVCV; YP_009507950.1); Grapevine leafroll-associated virus 1 (GLRaV-1; YP_004940642.1); Grapevine leafroll-associated virus 2 (GLRaV-2; AFV34734.1); Grapevine leafroll-associated virus 3 (GLRaV-3; AXI81954.1); Grapevine leafroll-associated virus 4 (GLRaV-4; AKB90851.1); Grapevine leafroll-associated virus 7 (GLRaV-7; YP_004935919.1); Grapevine leafroll-associated virus 13 (GLRaV-13; BAU68561.1); Little cherry virus 1 (LChV-1; AXN70106.1); Little cherry virus 2 (LChV-2; AAM96221.1); Lettuce chlorosis virus (LCV; AST35785.1); Lettuce infectious yellows virus (LIYV; AAA61798.1); Mint virus 1, (MV-1; YP_224091.1); Olive leaf yellowing-associated virus (OLYaV; CAD29306.1); Plum bark necrosis stem pitting-associated virus (PBNSPaV; AGL80631.1); Persimmon virus B (PeVB; YP_009112883.1); Pineapple mealybug wilt-associated virus 1 (PMWaV-1; ABR68934.1); Pineapple mealybug wilt-associated virus 2 (PMWaV-2; AAG13939.1); Pineapple mealybug wilt-associated virus 3 (PMWaV-3; ABD62348.1); Potato yellow vein virus (PYVV; ASD49931.1); Raspberry leaf mottle virus (RLMoV; YP_874185.1); Rose leaf rosette-associated virus (RLRaV; YP_009058929.1); Strawberry chlorotic fleck-associated virus (SCFaV; YP_762622.1); Strawberry pallidosis-associated virus (SPaV; YP_003289291.1); Sweet potato chlorotic stunt virus (SPCSV; AEA92656.1); Tomato infectious chlorosis virus (TICV; YP_003204952.1); Tomato chlorosis virus (ToCV; YP_293695.1); Tobacco virus 1 (TV-1; YP_009162622.1); Tetterwort vein chlorosis virus (TVCV; YP_009507961.1)

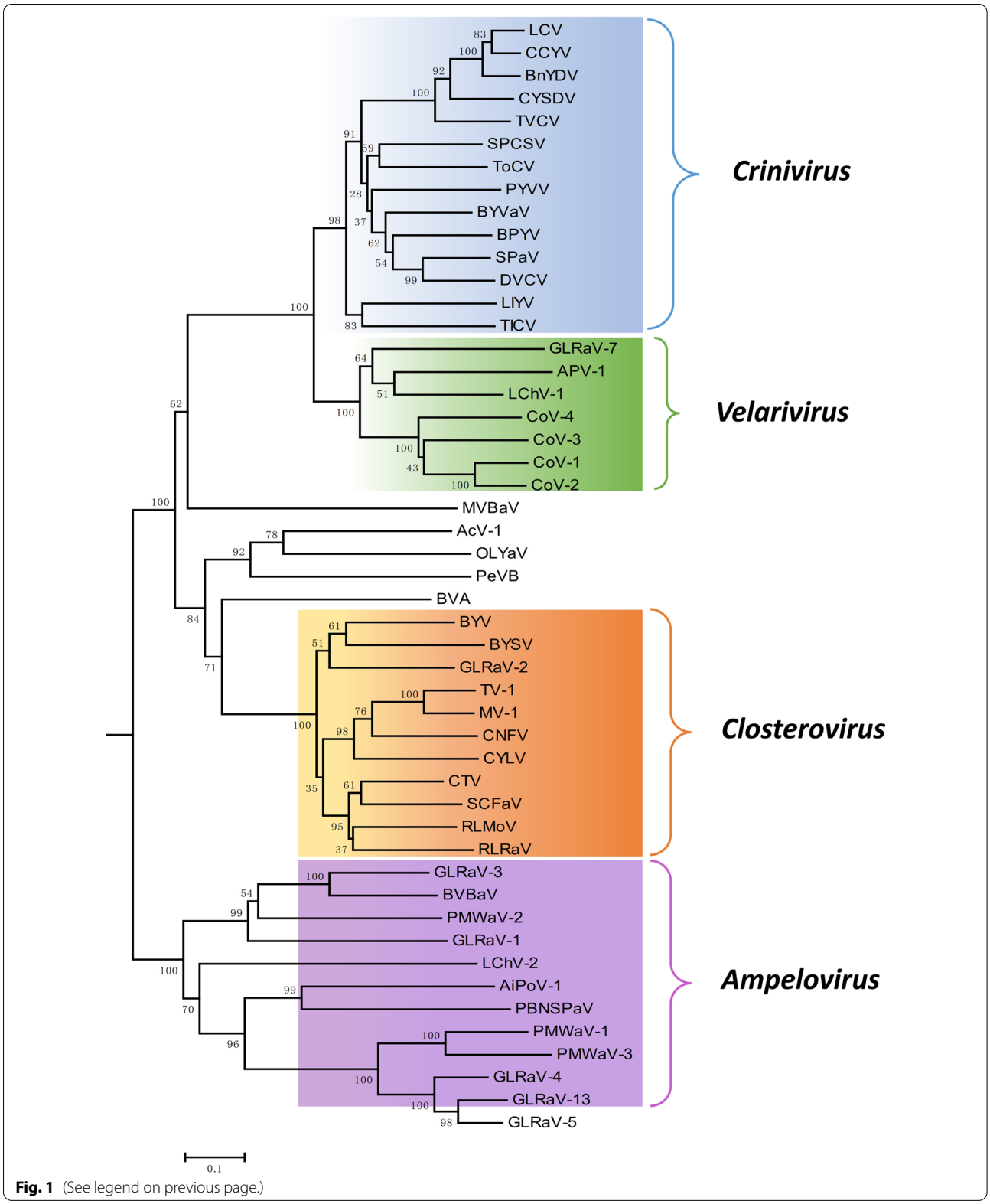

Fig. 1 (See legend on previous page.)

Table 1 Closterovirids with their origin, natural hosts, transmission vectors, diverse modes of infection, and disease epidemiology

Genus	Species	Origin	Natural hosts	Vector	Mixed infection	Disease epidemiology	References
Ampelovirus	AiPoV1	Florida, USA	*Dioscorea bulbifera*	*Pseudococcidae*	DVM	It causes mild disease symptoms in isolated conditions, while co-infection may also result in synergistic effects, creating additional symptomology. Environmental factors promising to vector population dynamics result in disease epidemics	Dey et al. (2019)
	BVBaV	Mississippi, USA	Blackberry, *Beta vulgaris*	*Pseudococcus maritimus*	BYVaV, BVY, BPYV, BCRV, INSV, TRSV	The impact of BVBaV longevity and quality attributes has been documented in several blackberry cultivars. Rapid disease spread was observed in the affected areas, causing significant losses	Thekke-Veetil et al. (2013)
	GLRaV-1	Australia	*Vitis vinifera*, *V. rotundifolia*	*Pseudococcidae*, *Coccidae*	GLRaV-1, GLRaV-3, GLRaV-4, GLRaV-5, GLRaV-9, GLRaV-13, GLRaV-Cn	Grapevine leafroll disease (GLRD) spreads through grafting and vectors, causing about 60% of the losses in grapes production globally. Resistant cultivars and insect vector management reduce the disease impact significantly	Martelli et al. (2012) and Naidu et al. (2014)
	GLRaV-3	Australia	*V. vinifera* (Pinot noir, Cabernet Sauvignon, Barbera)	*Pseudococcidae*, *Coccoidea*	GRSPaV, GVA, GVB	It is considered as a main etiological agent contributing to GLRD, consistently affecting vine health and crop production economically over the lifespan of a vineyard when no intervention strategies are implemented	Maree et al. (2013)

Table 1 (continued)

Genus	Species	Origin	Natural hosts	Vector	Mixed infection	Disease epidemiology	References
	GLRaV-4	Australia	*V. vinifera*	*Planococcus ficus*, *Ceroplastes rusci*	GLRaV-1, GLRaV-3, GLRaV-4, GLRaV-Cn, GLRaV-13	In ampelovirus disease epidemics, the diversity, morphology, fecundity, and transmission efficiency of a vector species in a specific grapevine-growing area have significant epidemiological consequences	Naidu et al. (2014)
	GLRaV-13	Japan	*V. vinifera*	*Pseudococcidae*, *Coccoidea*	GLRaV-1, GLRaV-3	Its infection causes severe mortality due to graft incompatibility in nurseries and vineyards, and delayed or irregular ripening which can affect harvest timing and crop production up to 50%	Ito and Nakaune (2016)
	LChV2	Australia	*Prunus Avium*, *P. cerasus*	*Phenacoccus aceris*, *Pseudococcus maritimus*	LChV1, LChV-2/USA6a, LChV-2/USA6b	It is a primary causal agent of little cherry disease (LCD). In British Columbia and Canada, a region that borders Washington State, LChV2 was reported at an epidemic level, resulting in 90% losses of marketable sweet cherry production between 1947 and 1979. Vector magnitude, grafting, and sample transportation are the main epidemiological consequences	Rott and Jelkmann (2005) and Mekuria et al. (2014)

Table 1 (continued)

Genus	Species	Origin	Natural hosts	Vector	Mixed infection	Disease epidemiology	References
	PMWaV-1, PMWaV-2, PMWaV-3	Hawaii, USA	*Ananas comosus*	*Dysmicoccus brevipes*, *D. neobrevipes*	PMWaV-2, PMWaV-3, PMWaV-1	PMWaV-1 is a causal agent of Mealybug wilt of pineapple disease (MWPD). Plants affected by MWPD were infected by both PMWaV-3 and PMWaV-2, indicating that a complex of ampeloviruses may be widespread in Cuban pineapple fields. Favourable environmental conditions for vector population dynamics result in disease epidemics. Implement certification procedures for pineapple propagation materials and various cultivars to reduce the economic impact of MWPD on pineapple crops in Cuba and all over the world	Sether and Hu (2002), Hernández-Rodríguez et al. (2017) and Dey et al. (2018)
	PBNSPaV	USA	*Prunus salicina*, *P. persica*, *P. avium*, *P. dulcis*, *P. armeniaca*	Not reported	CVA, CNRMV, CGRMV, LChV-1	PBNSPaV has high epidemiological consequences because of the large number of hosts. In mixed infection conditions, it causes severe economic losses globally. Certification of propagation materials can reduce the economic impact of disease	Sabanadzovic et al. (2005) and Salleh et al. (2011)

Table 1 (continued)

Genus	Species	Origin	Natural hosts	Vector	Mixed infection	Disease epidemiology	References
Closterovirus	BYSV	California, USA	*Beta vulgaris, Chenopodium capitatum, Chenopodiaceae, Compositae, Geraniaceae, Portulacaceae, Solanaceae*	*Nasonovia lactucae, Myzus persicae, Macrosiphum euphorbiae*	BYV, BtMV, BWYV	BYSV incidence is very high during prolonged dry conditions if plants are infected with other viruses. Epidemics of disease have been reported in rows adjacent to the sowthistle-infested area, but progressively decrease with increasing distance from the virus source	Karasev et al. (1998)
	BYV			*M. persicae, Aphis fabae, Rhopalostphum padi, Macrostphum rosae*	BWYV, BMV, BCTV, BChV, BMYV, BtMV	BYV infection decreases 20–35% of crop production and has epidemic consequences for yield loss under co-infection conditions with BWYV and BMV. It also increases beet plant susceptibility to infection by several pathogenic fungi and BCTV	Kirk et al. (1991) and Wintermantel (2005)
	CNFV	Japan	*Dianthus caryophyllus, D. barbatus, Chrysanthemum morifolium* Ramat cv., White Snowdon	*M. persicae, A. craccivora, A. gossypii*	CarMV, TAV, TSWV, CVB	It has caused severe disease in carnation crops, particularly in mixed infections with other viruses. The virus was reported to have about 14% disease incidence in California, USA, in 1983. In general, high levels of crop hygiene, including vector control and resistant cultivars, have reduced the virus incidence	Raikhy et al. (2003) and Mitiouchkina et al. (2018)

Table 1 (continued)

Genus	Species	Origin	Natural hosts	Vector	Mixed infection	Disease epidemiology	References
	CYLV		*Daucus carota* L., *Heracleum sphondylium* L., Beet roots	*Cavariella aegopodii, C. archangelicae, C. theobaldi, C. pastinacae*	CtRLV, CMoV, CtRLVa-RNA, PYFV	CYLV is a causal pathogen of carrot internal necrosis, causing the incidence of necrosis by 96%. Favorable climatic conditions for vector populations and viral sources are the main consequences of CYLV disease epidemiology	Adams et al. (2014)
	CTV	Florida, USA	*Citrus sinensis*, *C. reticulata* Blanco, *C. paradisi* Macf., *C. aurantifolia* (Christm.), *C. limon* (L.) Burn. f	*Toxoptera citricida*, *A. gossypii*	CDVd, CTV isolates (CTV9R-MCA13NR, T30-1)	The most devastating tristeza epidemics occurred in Argentina (1930), Brazil (1937), California (1939), Florida (1951), Spain (1957), Israel (1970), and Venezuela (1980), but significant outbreaks have also been reported from Cyrus (1989), Cuba (1992), Mexico (1995), Dominican Republic (1996), and Italy (2002). The certification of budstock and the planting of resistant rootstock are major counter-measures in combating the disease	Moreno et al. (2008)
	GLRaV -2	North America	*V. vinifera* L., *V. rotundifolia*, *V. aestivalis*	Not reported	GLRaV-3, GLRaV-1, GLRaV-4, GLRaV-13, GLRaV-Cn	It plays a significant role in GLRD epidemics through grafting and causes severe losses in grape production globally. Rootstocks and bud treatment as well as resistant cultivars, reduced the disease impact significantly	Maree et al. (2013) and Naidu et al. (2014)

Table 1 (continued)

Genus	Species	Origin	Natural hosts	Vector	Mixed infection	Disease epidemiology	References
	MV-1	USA	Golden ginger, Ginger, Mint	*Ovatus crataegarius*	SLRSV, TRSV, MV-2	In a solitary condition, MV-1 has less epidemiological impact than other viruses. Although under co-infections and suitable environmental conditions, this virus displays striking yellow vein banding symptoms on Variegata plants	Tzanetakis et al. (2005a)
	RLMV	UK	Raspberry, Blackberry, Red raspberry	*Amphorophora agathonica* Hottes	RMoV, RLSV, RpLV, RYNV, BRNV	Its synergistic interactions with RMoV, RpLV, RYNV, and BRNV resulted in black raspberry decline (BRD) and raspberry mosaic disease (RMD) epidemics	Tzanetakis et al. (2007) and Quito-Avila et al. (2014)
	RLRaV	Canada	*Rosa multiflora* Thunb, *Rosa rugosa* Thunb	Not reported	ASGV, BCRV, PNRSV	The most destructive disease of commercial roses is a wild rose leaf rosette disease (WRLRD), primarily caused by RLRaV, resulting in severe epidemics with ASGV, BCRV, and PNRSV	He et al. (2015)
	SCFaV	Western coast of North, America	*Fragaria* × *ananassa* Duch., *F. vesca, F. virginiana*	*A. gossypii*	SPaV, BPYV	Strawberry decline (SD) is characterized by plant collapse and death associated with SCFaV in mixed infection of SPaV and BPYV. Favorable meteorological factors and vector populations resulted in 100% crop losses	Tzanetakis and Martin (2008)
	TV1	Hertfordshire, Lanarkshire	*Nicotiana tabacum, Solanum lycopersicum*	Not reported	TMV, TVBMV, TSV	TV1 causes enation mosaic in tomato and tobacco plants with TMV, TSV, and TVBMV. This virus has high biological consequences	Wang et al. (2016a)

Table 1 (continued)

Genus	Species	Origin	Natural hosts	Vector	Mixed infection	Disease epidemiology	References
Crinivirus	AYV	USA (Illinois)	*Abutilon theophrasti* Medic., *Anoda abutiloides* A. Gray, *Malvaceae*	*T. abutilonea* Haldeman	Not reported	It is a whitefly-transmitted virus. Suitable meteorological factors for the vector population lead to a wide spread of disease	Tzanetakis et al. (2013)
	BnYDV	Spain	*Phaseolus vulgaris* L., *Pisum sativum* L., *Lens culinaris* Medik., *Vicia faba* L.	*Bemisia tabaci* (Q-biotype)	BYMV, BLRV	BnYDV is a serious disease of beans. Its incidence increased from 34 to 50% in bean-growing greenhouses from 2004 to 2005 in Spain	Martín et al. (2011)
	BPYV	California, USA	*Cucumis sativus*, *C. melo*, *Amaranthus retroflexus*, *Selosia cristata*, *Sonchus oleraceus*	*T. vaporariorum*	CYSDV, CCYV, CABYV, SPaV	BPYV, together with CYSDV and CABYV, causes yellowing disease in the *Cucurbitaceae* family. Epidemics of disease are associated with its large host range, high light intensity, and vector population	Boubourakas et al. (2006)
	BYVaV	North and South Carolina, USA	Blackberry, Raspberry	*T. abutilonea*, *T. vaporariorum*	BVY, INSV, BVE, BCRV	Blackberry yellow vein disease (BYVD) is a destructive disease of blackberries in the USA, caused by BYVaV in combination with several viruses. This virus appears to be the most prevalent and needs more attentions to its epidemiology	Poudel et al. (2013)
	CYSDV	United Arab Emirates	*Cucurbitaceae*	*B. tabaci* (biotypes A, B), *B. argentifolii*	BPYV, CABYV	It causes yellowing disease in the *Cucurbitaceae* family in association with BPYV and CABYV. That has recently become a devastating production threat in cucurbit-growing regions of Mexico, southern USA, and Central America	Boubourakas et al. (2006)

Table 1 (continued)

Genus	Species	Origin	Natural hosts	Vector	Mixed infection	Disease epidemiology	References
	DVCV	Virginia, USA	*Diodia virginiana* L., *Rubiaceae*	*T. abutilonea*, *T. vaporariorum*	Not Reported	Disease epidemics in the vicinity of infected crops are characterized by vector population dynamics. DVCV has fewer biological consequences than other viruses	Tzanetakis et al. (2011)
	LChV	California, USA	*Lactuca sativa*, *Phaseolus vulgaris*, *Spinacia oleracea*, *Phaseolus vulgaris*	*B. tabaci* (A, B biotypes)	LIYV	In co-infection with LIYV, it causes yellowing disease on lettuce and sugarbeet. A disease epidemic was recorded in the southwest desert region of the USA because of its wide host range and vector population	Kubota and Ng (2016)
	LIYV	USA	*Lactuca sativa, Beta vulgaris, Cucumis melo, Daucus carota*, *Citrullus lanatus*	*B. tabaci* (biotype A)	LChV	LIYV infected various autumn-planted vegetable crops (lettuce, sugarbeets, crucifers, and cucurbits). In the early 1980s, 100% of susceptible plants were affected, resulting in $20 million crop losses in a single growing season	Medina et al. (2005)
	PYVV	Venezuela, Columbia, Ecuador, Peru	Genera; *Solanum*, *Polygonum*, *Rumex*, *Tagetes*, *Catharanthus*, *Malva*	*T. vaporariorum*	PVY, ToCV, TICV	In various regions of Colombia, epidemiological surveys for potato yellow vein disease (PYVD) indicated *Polygonum* spp., *Polygonum mepalense*, *Rumex obtusifolium*, *Tagetes* spp., and *Catharanthus roseus* as potential viral reservoirs	Muñoz Baena et al. (2017)

Table 1 (continued)

Genus	Species	Origin	Natural hosts	Vector	Mixed infection	Disease epidemiology	References
	SPaV	USA	*Fragaria* × *ananassa* Duch.	*T. vaporariorum*	BPYV	Strawberry pallidosis disease (PD), exhibiting decline symptoms in strawberries, is characterized by a mixed infection of SPaV and BPYV. The disease epidemic was reported during 2002–2003 in California, causing about 50-million-dollar losses in two seasons	Tzanetakis et al. (2006)
	SPCSV	Sub-Saharan, Africa	*Ipomoea batatas* L., *I. setosa, I. acuminata, I. hederacea, I. hederifolia*	*B. tabaci* (biotype *B*), *T. vaporariorum, B. afer* sensu lato	SPFMV, SPMSV, SPMMV	It is an extremely destructive virus that causes yield loss, resistance breaking in sweet potato to SPFMV, and the combined infection causes a devastating severe sweet potato virus disease (SPVD)	Kreuze et al. (2002)
	TVCV	South Korea	*Chelidonium majus*	Not reported	Not reported	A valuable herbaceous plant, *Chelidonium majus*, has undergone a serious viral threat because of TVCV. Epidemiological and biological consequences of this virus still need to be reported	Zhao et al. (2015)

Table 1 (continued)

Genus	Species	Origin	Natural hosts	Vector	Mixed infection	Disease epidemiology	References
	ToCV	Florida, USA	*Lycopersicon esculentum* Mill., *Solanum tuberosum, Capsicum annuum, Physalis philadelphica*	*B. tabaci* (biotype A, B, Q), *T. abutilonea, T. vaporariorum*	TICV, ToSRV, TYLCV	ToCV and TICV are quarantine pathogens that cause yellow leaf disorder disease with TYLCV to agricultural crops in Florida (USA) and around the world. Disease epidemic has been reported in South Africa in whitefly-infested crops. Meteorological factors favoring vector and mixed infection of these viruses have significant epidemiological consequences	Wintermantel et al. (2009) and Tzanetakis et al. (2013)
	TICV	California, USA		*T. vaporariorum*	ToCV, ToSRV, TYLCV		
Velarivirus	APV1	Hainan, China	*Areca catechu* L.	Not reported	Not reported	It causes a serious yellow leaf disease (YLD) of Areca palm, characterized by yellowing of leaves in the inner whorl and progressively extending to the outer whorl of the crown	Yu et al. (2015)
	CoV-1, CoV-2, CoV-3, CoV-4	Hawaii, USA	*Cordyline fruticosa* L.	–	CoV-1, CoV-2, CoV-3, CoV-4, LChV-1, GLRaV-7	These viruses cause ti ringspot disease (TRD) in mixed infection conditions. TRD was reported in commercial and residential ti plants harboring multiple velariviruses on the islands of Oahu, Maui, and Hawaii	Melzer et al. (2013a; b)
	GLRaV-7	Albania	*V. vinifera*	–	GLRaV-1, GLRaV-2, GLRaV-3	GLRaV-7 plays a vital role in GLRD expression in association with other GLRaVs, contributing 40% economic loss to grape production	Jelkmann et al. (2012) and Martelli et al. (2012)

Table 1 (continued)

Genus	Species	Origin	Natural hosts	Vector	Mixed infection	Disease epidemiology	References
	LChV1	Japan	*Prunus avium, P. cerasus, P. mahaleb*	–	LChV1, LChV2	Little cherry disease (LCD) is a serious concern for sweet cherry producers globally. Disease epidemics occurred in Canada and Washington State in 1938 and 1940s, respectively, resulting in significant acreage eradication	Wang et al. (2016b)
Unassigned	AcV-1	Italy	*Actinidia chinensis, A. deliciosa*	–	PZSV, AcVA, AcVB	This virus is a serious threat to the kiwifruit in China, Italy, New Zealand, and Chile. It is characterized by chlorotic and necrotic rings on leaves followed by general decline and death of the scion but not of the rootstock	Blouin et al. (2018)
	BVA	Michigan, USA	*Vaccinium corymbosum, V. ashei*	*Aphidoidea*	BlSSV, BLMoV	It causes a devastating threat to blueberry production in association with other viruses. Infected planting material is a major source of disease epidemics. Blueberry certification programs have minimized the impact of disease	Isogai et al. (2013)
	MVBaV	Oregon, USA	*Mentha × gracilis*	*Ovatus crataegarius*	MVX	Mint has great importance for its unique fragrance, food, medical industry, and ornamentals. MVBaV adversely affects this crop and deteriorates its commercial characteristics	Tzanetakis et al. (2005b)

Table 1 (continued)

Genus	Species	Origin	Natural hosts	Vector	Mixed infection	Disease epidemiology	References
	OLYaV	Mediterranean	*Olea Europaea* L.	–	–	It has been found with the highest incidence of 93.8% in California. In Southern Italian regions, OLYaV-infected olive trees have also been detected (60% in Sicily and 86% in Calabria) in a large number of cultivars (positive/tested 35/50 and 18/25, respectively)	Fontana et al. (2019)
	PeVB	Japan	*Diospyros virginiana* L., *D. kaki* Thunb.	–	–	PeVB is a serious disease of American and Japanese persimmon, influencing the vigor, production, and quality of fruits	Ito et al. (2015)

AcV-1 Actinidia virus 1, *AcVA* Actinidia virus A, *AcVB* Actinidia virus B, *AiPoV1* Air potato ampelovirus 1, *APV1* Areca palm velarivirus 1, *ASGV* Apple stem grooving virus, *AWSV* Alligatorweed stunting virus, *AYV* Abutilon yellows virus, *BChV* Beet chlorosis virus, *BCRV* Blackberry chlorotic ringspot virus, *BCTV* Beet curly top virus, *BLRV* Bean leafroll virus, *BMV* Brome mosaic virus, *BMYV* Beet mild yellowing virus, *BnYDV* Bean yellow disorder virus, *BPYV* Beet pseudo-yellows virus, *BRNV* Black raspberry necrosis virus, *BtMV* Beet mosaic virus, *BWYV* Beet western yellows virus, *BVA* Blueberry virus A, *BVBaV* Blackberry vein banding-associated virus, *BVE* Blackberry virus E, *BVY* Blackberry virus Y, *BYMV* Bean yellow mosaic virus, *BYSV* Beet yellow stunt virus, *BYV* Beet yellows virus, *BYVaV* Blackberry yellow vein-associated virus, *CABYV* Cucurbit aphid-borne yellows virus, *CarMV* Carnation mottle carmovirus, *CCYV* Cucurbit chlorotic yellows virus, *CDVd* Citrus dwarfing viroid, *CGRMV* Cherry green ring mottle virus, *CNRMV* Cherry necrotic rusty mottle virus, *CoV-1* Cordyline virus 1, *CoV-2* Cordyline virus 2, *CoV-3* Cordyline virus 3, *CoV-4* Cordyline virus 4, *CMoV* Carrot mottle virus, *CNFV* Carnation necrotic fleck virus, *CtRLV* Carrot red leaf virus, *CtRLVaRNA* Carrot red leaf associated viral RNA, *CTV* Citrus tristeza virus, *CVA* Cherry virus A, *CVB* Chrysanthemum virus B, *CYLV* Carrot yellow leaf virus, *CYSDV* Cucurbit yellow stunting disorder virus, *DMV* Dioscorea mosaic virus, *DVCV* Diodia vein chlorosis virus, *GLRaV-1* Grapevine leafroll-associated virus 1, *GLRaV-2* Grapevine leafroll-associated virus 2, *GLRaV-4* Grapevine leafroll-associated virus 4, *GLRaV-5* Grapevine leafroll-associated virus 5, *GLRaV-7* Grapevine leafroll-associated virus 7, *GLRaV-9* Grapevine leafroll-associated virus 9, *GLRaV-13* Grapevine leafroll-associated virus 13, *GLRaV-Cn* Grapevine leafroll-associated Carnelian virus, *GRSPaV* Grapevine Rupestris stem pitting-associated virus, *GVA* Grapevine virus A, *GVB* Grapevine virus B, *INSV* Impatiens necrotic spot tospovirus, *LChV* Lettuce cholorosis virus, *LChV1* Little cherry virus 1, *LChV2* Little cherry virus 2, *LIYV* Lettuce infectious yellows virus, *MV-1* Mint virus 1, *MV-2* Mint virus-2, *MVBaV* Mint vein banding-associated virus, *MVX* Mint virus X, *OLYaV* Olive leaf yellowing associated virus, *PBNSPaV* Plum bark necrosis stem pitting-associated virus, *PeVB* Persimmon virus B, *PMWaV-1* Pineapple mealybug wilt-associated virus 1, *PMWaV-2* Pineapple mealybug wilt-associated virus 2, *PMWaV-3* Pineapple mealybug wilt-associated virus 3, *PNRSV* Prunus necrotic ringspot virus, *PVY* Potato virus Y, *PYFV* Parsnip yellow fleck virus, *PYVV* Potato yellow vein virus, *PZSV* Pelargonium zonate spot virus, *RLMV* Raspberry leaf mottle virus, *RLRaV* Rose leaf rosette-associated virus, *RLSV* Raspberry leaf spot virus, *RMoV* Raspberry mottle virus, *RpLV* Raspberry latent virus, *RYNV* Rubus yellow net virus, *SCFaV* Strawberry chlorotic fleck-associated virus, *SLRSV* Strawberry latent ringspot virus, *SPaV* Strawberry pallidosis associated virus, *SPCSV* Sweet potato chlorotic stunt virus, *SPFMV* Sweet potato feathery mottle virus, *SPMSV* Sweet potato mild speckling potyvirus, *SPMMV* Sweet potato mild mottle ipomovirus, *TAV* Tomato aspermy virus, *TICV* Tomato infectious chlorosis virus, *ToCV* Tomato chlorosis virus, *ToSRV* Tomato severe rugose virus, *TMV* Tobacco mosaic viruses, *TRSV* Tobacco ringspot virus, *TSV* Tobacco streak virus, *TSWV* Tomato spotted wilt virus, *TV1* Tobacco virus 1, *TVBMV* Tobacco vein banding mosaic virus, *TVCV* Tetterwort vein chlorosis virus, *TYLCV* Tomato yellow leaf curl virus

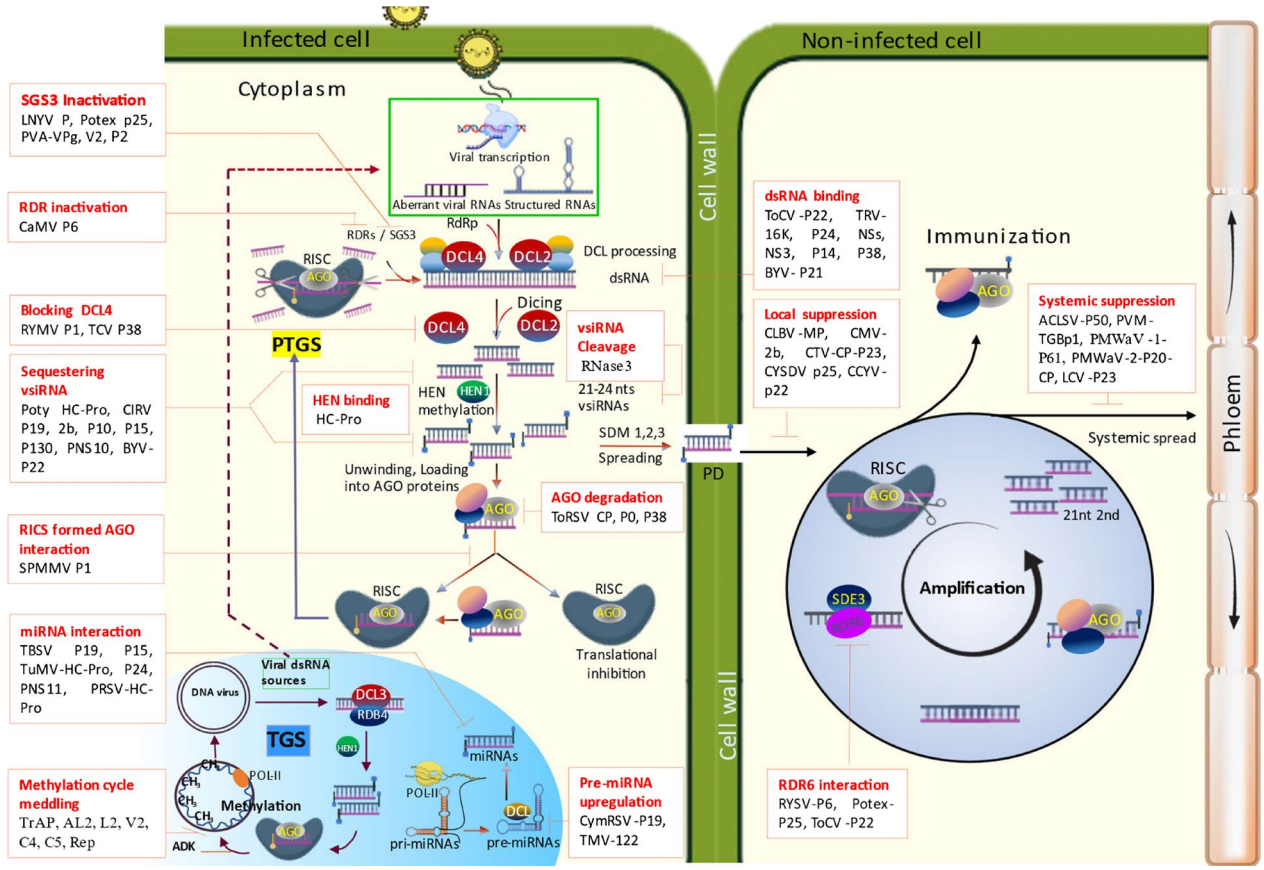

Fig. 2 The antiviral RNA silencing model and VSRs with diverse sets of strategies for battling against RNA silencing [adapted from Burgyán and Havelda (2011), Csorba et al. (2015), Gaffar and Koch (2019)]. An antiviral response is initiated with the biogenesis of 21–24 nt vsiRNAs and miRNAs, the silencing molecules that are produced through DCL processing (vsiRNAs are produced primarily by DCL4 and secondarily by DCL2 in the cytoplasm, whereas miRNAs are synthesized in the nucleus by DCL1). These silencing molecules are stabilized by the HEN1 methylation process and subsequently loaded into AGOs to form an RNA-induced silencing complex (RISC), followed by the effector phase of post-transcriptional gene silencing (PTGS) completed through viral RNA cleavage or translational inhibition. To amplify the antiviral RNA silencing response, AGO-sliced products and aberrant viral transcripts or RNAs are used as templates for RDR complexes employing RNA-helicases (SGS3 and SDE5), the cofactors of RDRs triggered pathways. Transcriptional gene silencing (TGS) is triggered by the production of 24-nt vsiRNAs via DCL3 processing (nucleus) in the case of DNA virus infection. HEN1-methylated 24-nt vsiRNAs are loaded into AGO4 to complete TGS after methylation of the viral genome through a DNA methylation cycle mediated by Pol II, ADK, SAHH, and SAMS. VSRs that interfere with PTGS or TGS are presented in boxes along the sides of the figure. The lines indicate the target site at which VSRs interact with the antiviral pathway

cells where local silencing signals have been received. These new dsRNAs are processed into secondary siRNAs through DCL4 and movement proceeds across additional 10–15 cells (Fig. 2). Therefore, the local and systemic silencing signaling events are mediated by primary and secondary vsiRNAs synthesis, respectively (Voinnet 2005; Melnyk et al. 2011; Mermigka et al. 2016).

Viral strategies to evade antiviral RNA silencing

PTGS is an important defense mechanism of plants against viral infection. To recruit a successful infection, viruses have evolved diversified strategies, of which the production of VSRs can negatively control PTGS by constraining miRNA and siRNA regulation in plants (Hu et al. 2020). Extensive studies on a large number of plant viruses have demonstrated the multi-functionality of VSRs. In addition to suppress RNA silencing, they also play significant roles as transcriptional activation factors (helicase, protease, and replicase), symptom determinants, as well as helper components in the viral infection process. Therefore, VSRs have evolved independently with no structural similarities, repressing antiviral RNA silencing in hosts by affecting the involved core components (Li and Wang 2019) (Table 2, Fig. 2).

Table 2 Viral suppressors of RNA silencing (VSRs) encoded by plant-infecting viruses and the corresponding strategies to suppress antiviral RNA silencing

Family	Genus	Species	Genome	VSR	Viral strategies to suppress antiviral RNA silencing	References
Caulimoviridae	*Caulimovirus*	CaMV	dsDNA	P6 (TAV)	Suppresses RNA silencing by DRB4 inactivation and interference with RDR6-dependent trans-acting and secondary vsiRNA pathways	Haas et al. (2008) and Shivaprasad et al. (2008)
Geminiviridae	*Begomovirus*	TYLCV	ssDNA	V2	Suppresses antiviral RNA silencing locally by interacting with SGS3	Zrachya et al. (2007) and Glick et al. (2008)
		TYLCCNV	ssDNA	βC1	Interacts with an endogenous suppressor of RNA silencing (rgsCAM) to inhibit RDR6 expression and secondary siRNA production	Li et al. (2014)
	Curtovirus	BCTV	ssDNA	L2/AL2	Inactivates ADK, suppresses SAMS and SAHH to interfere with the methylation process	Buchmann et al. (2009) and Csorba et al. (2015)
	Mastrevirus	WDV	ssDNA	Rep	Impedes both local and systemic silencing via 21-nt and 24-nt siRNA binding	Wang et al. (2014)
Tospoviridae	*Orthotospovirus*	GBNV	(−)ssRNA	NSs	Interferes with miRNA biogenesis and suppresses antiviral RNA silencing responses locally	Schnettler et al. (2010) and Goswami et al. (2012)
		GRSV, INSV, TSWV, TYRV	(−)ssRNA	NSs	Binds to dsRNA duplexes, including siRNA and miRNA, and suppresses local and systemic silencing	Schnettler et al. (2010) and Hedil et al. (2015)
Phenuiviridae	*Tenuivirus*	RHBV, RSV	(−)ssRNA	NS3	Suppresses RNA silencing in plants and insects. Binds to RNA/RNA or RNA/DNA duplexes with a length greater than 9 nt and long ssRNA	Hemmes et al. (2007) and Shen et al. (2010)
		RSV	(−)ssRNA	P2	Interacts with SGS3 and enhances PVX pathogenicity	Du et al. (2011)
Rhabdoviridae	*Cytorhabdovirus*	LNYV	(−)ssRNA	Phosphoprotein (P)	Interacts with protein AGO (1, 2, 4), RDR6, and SGS3 to suppress RISC-mediated cleavage and RNA silencing amplification	Mann et al. (2016)
	Nucleorhabdovirus	RYSV	(−)ssRNA	P6	Suppresses systemic RNA silencing only via inhibiting RDR6-mediated secondary siRNA biosynthesis and enhancing PVX virulence	Guo et al. (2013)
Bromoviridae	*Cucumovirus*	CMV	(+)ssRNA	2b	Interacts with RDR proteins, suppresses AGO1 via upregulating miR168, and binds to siRNA as well	Goto et al. (2007) and Varallyay and Havelda (2013)
		TAV	(+)ssRNA	2b	Suppresses RNA silencing through the size-selective binding of siRNA	Chen et al. (2008)
	Ilarvirus	AV-2	(+)ssRNA	2b	Viral suppressor activity is retained in systemic silencing but not in local silencing	Shimura et al. (2013)
Comovirinae	*Comovirus*	CPMV	(+)ssRNA	CP (S)	C-terminal 16 amino acids of the CP (S) are predominantly essential for VSR activity	Canizares et al. (2004)
Secoviridae	*Nepovirus*	ToRSV	(+)ssRNA	CP	Suppresses RNA silencing through AGO1 binding and CP-mediated AGO1 degradation. VSR activity is conserved in the WG motif of CP	Karran and Sanfacon (2014)

Table 2 (continued)

Family	Genus	Species	Genome	VSR	Viral strategies to suppress antiviral RNA silencing	References
Reoviridae	*Phytoreovirus*	RDV	dsRNA	PNS10	Suppresses both local and systemic silencing, enhancing viral replication and stability. Down-regulation of RDR6 through binding with siRNA	Ren et al. (2010)
		RGDV	dsRNA	PNS11, PNS12	PNS11 suppresses RNA silencing through miRNA pathway interference. PNS12 shows nucleus localization and suppresses RNA silencing locally	Wu et al. (2011) and Shen et al. (2012)
	Oryzavirus	RRSV	dsRNA	PNS6	It is a local VSR, and ablation of the RNA binding region eliminates suppressor activity. Overexpression enhances PVX pathogenicity in *N. benthamiana*	Wu et al. (2010)
Luteoviridae	*Enamovirus*	PEMV-1	(+)ssRNA	P0	Suppresses local and systemic silencing via destabilization of AGO1	Fusaro et al. (2012)
	Polerovirus	BWYV, CYDV, CABYV	(+)ssRNA	P0	Targets AGO proteins through destabilization and the autophagy pathway and suppresses only local silencing	Pazhouhandeh et al. (2006), Baumberger et al. (2007), Bortolamiol et al. (2007) and Derrien et al. (2012)
		PLRV	(+)ssRNA	P0	Targets AGO1 and suppresses both local and systemic silencing	Fusaro et al. (2012)
		CLRDV, SCYLV	(+)ssRNA	P0	Suppresses local RNA silencing but not systemic silencing	Mangwende et al. (2009) and Delfosse et al. (2014)
Potyviridae	*Ipomovirus*	SPMMV	(+)ssRNA	P1	Interacts with AGO1 through WG/GW motifs and inhibits si/miRNA-programmed RISC activity	Giner et al. (2010)
		CVYV	(+)ssRNA	P1	It enhances the VSR activity HC-Pro in members of *Potyvirus*. Its duplicated form, P1b, down-regulates dsRNA formation and suppresses local RNA silencing	Valli et al. (2006)
	Tritimovirus	WSMV	(+)ssRNA	P1	Suppresses antiviral RNA silencing and downstream GFP siRNA accumulation significantly	Young et al. (2012)
	Poacevirus	TriMV, SCSMV	(+)ssRNA	P1	Suppresses dsRNA-triggered systemic silencing more efficiently than HC-Pro of TuMV and enhances the pathogenicity of heterologous virus	Tatineni et al. (2012)

Table 2 (continued)

Family	Genus	Species	Genome	VSR	Viral strategies to suppress antiviral RNA silencing	References
	Potyvirus	TEV	(+)ssRNA	HC-Pro	Binds to siRNA and inhibits its 3′ methylation, and suppresses AGO1 through miR168 up-regulation	Lakatos et al. (2006) and Varallyay and Havelda (2013)
		TuMV	(+)ssRNA	HC-Pro	Interacts with RAV2 to restrict primary siRNA biogenesis, and interferes with the miRNA pathway as well	Kasschau et al. (2003) and Endres et al. (2010)
		ZYMV	(+)ssRNA	HC-Pro	Binds to siRNA duplexes, interacts and inhibits HEN1 methylation activity	Fuellgrabe et al. (2011) and Jamous et al. (2011)
		PVY	(+)ssRNA	HC-Pro	Suppresses antiviral RNA silencing through binding with 21–22 nt sRNA of viral sequences	Del Toro et al. (2017)
		SCMV	(+)ssRNA	HC-Pro	Down-regulates the accumulation of secondary siRNAs and suppresses both sense RNA and dsRNA-induced silencing	Zhang et al. (2008)
		PVA	(+)ssRNA	HC-Pro, VPg	HC-Pro interferes with host gene expression (SAHH) and interrupts the methionine cycle to suppress RNA silencing. VPg demonstrates SGS3 interaction	Rajamaki and Valkonen (2009) and Ivanov et al. (2016)
		PRSV	(+)ssRNA	HC-Pro	Interferes with the miRNA pathway and down-regulates AGO1 via miR168 upregulation	Azad et al. (2014)
		PPV	(+)ssRNA	HcPro-P1	Enhances silencing suppression in a synergistic form (HcPro-P1)	Valli et al. (2006)
	Rymovirus	AgMV, HoMV	(+)ssRNA	HC-Pro	Suppresses RNA silencing with tritimoviruses P1 while its VSR function is still unclear	Young et al. (2012)

Table 2 (continued)

Family	Genus	Species	Genome	VSR	Viral strategies to suppress antiviral RNA silencing	References
Tombusviridae	*Aureusvirus*	PoLV	(+)ssRNA	P14	Binds to dsRNAs without size selection and suppresses silencing through sequestering dsRNAs and ds siRNAs	Merai et al. (2005)
	Tombusvirus	CIRV	(+)ssRNA	P19	Suppresses RNA silencing via binding with siRNA and hampering the 3′ methylation of siRNAs	Lozsa et al. (2008) and Rawlings et al. (2011)
		CymRSV	(+)ssRNA	P19	Silencing suppression through AGO1 repression via miR168 upregulation and binding silencing-generated 21–25 nt dsRNA size dependently	Silhavy et al. (2002) and Varallyay et al. (2010)
		TBSV	(+)ssRNA	P19	Suppresses antiviral RNA silencing via interacting miRNA pathways at the intermediate level, including miRNA methylation	Chapman et al. (2004) and Yu et al. (2006)
		CNV	(+)ssRNA	P20	Its VSR activity is quite similar to TBSV p19 but 50-fold lower than p19	Hao et al. (2011)
	Carmovirus	TCV	(+)ssRNA	P38	Targets AGO1 through GW-motifs and modifies DCL usage by increasing DCL1 levels, resulting in considerable DCL3 and DCL4 downregulation. Binds to dsRNA size independently and interferes with primary siRNA biogenesis (effect dependent on interaction with RAV2)	Merai et al. (2006), Azevedo et al. (2010) and Endres et al. (2010)
		PFBV, HCRSV, PLPV	(+)ssRNA	CP/ P37	Binds to siRNAs and suppresses RNA silencing with the enhancement of PVX pathogenicity. Mutations in the GW motif affects siRNA binding capability	Meng et al. (2006), Martinez-Turino and Hernandez (2009) and Perez-Canamas and Hernandez (2015)
		MNSV	(+)ssRNA	P7B, P42	P7B delays RNA silencing. P42 enhances local spread similar to that of potyviral HC-Pro, probably because of its VSR activity	Genoves et al. (2006)
	Dianthovirus	RCNMV	(+)ssRNA	MP	It is an unknown DCL1 dependent MP and interferes with miRNA biogenesis	Takeda et al. (2005)

Table 2 (continued)

Family	Genus	Species	Genome	VSR	Viral strategies to suppress antiviral RNA silencing	References
Closteroviridae	*Ampelovirus*	GLRaV-3	(+)ssRNA	P19.7	Impedes RNA silencing through siRNA and miRNA pathway interference	Gouveia and Nolasco (2012) and Gouveia et al. (2012)
		PMWaV-1,-2	(+)ssRNA	P61, CP, P20	P61 suppresses RNA silencing systemically, CP subdues both local and systemic silencing, and P20 affects dsRNA-induced local silencing	Dey et al. (2015)
	Closterovirus	BYV	(+)ssRNA	P21	Interacts with 21 nt or longer ssRNAs and dsRNAs and suppresses dsRNA-induced silencing of GFP mRNA and blocks HEN1 methyltransferase	Reed et al. (2003), Lu et al. (2004) and Ye and Patel (2005)
		CTV	(+)ssRNA	CP, P20, P23	CP and p20 suppress antiviral RNA silencing pathways at the intercellular level, while P23 does so at the intracellular level	Lu et al. (2004), Fagoaga et al. (2011) and Benitez-Galeano et al. (2015)
		RLRaV	(+)ssRNA	P17	Suppresses antiviral RNA silencing in wild roses	He et al. (2015)
	Crinivirus	CYSDV	(+)ssRNA	P25	Inhibits siRNA accumulation and suppresses dsRNA- or ssRNA-induced silencing of GFP mRNA	Kataya et al. (2009)
		CCYV	(+)ssRNA	P22	Binds with CsSKP1LB and suppresses antiviral RNA silencing	Chen et al. (2019)
		SPCSV	(+)ssRNA	RNase3, P22	RNase3 displays endonuclease activity and cleaves siRNAs, but the P22 silencing strategy is still elusive	Kreuze et al. (2005) and Cuellar et al. (2008)
		LCV	(+)ssRNA	P23	Enhances degradation of siRNAs and suppresses silencing	Kubota and Ng (2016)
		ToCV	(+)ssRNA	P22, CP, CPm	P22 counteracts RDR6-mediated antiviral response, CP induces SAHH-interaction, and CPm silencing mechanism is unidentified	Canizares et al. (2008), Landeo-Rios et al. (2016a) and Liu et al. (2021)
		TICV	(+)ssRNA	P27	Suppresses S-PTGS and IR-PTGS	Mashiko et al. (2019)

Table 2 (continued)

Family	Genus	Species	Genome	VSR	Viral strategies to suppress antiviral RNA silencing	References
Virgaviridae	*Hordeivirus*	BSMV, PSLV	(+)ssRNA	γB	Suppresses RNA silencing by preferentially binding to siRNA size selectively	Yelina et al. (2002) and Merai et al. (2006)
	Pecluvirus	PCV	(+)ssRNA	P15	Binds to siRNA size selectively and interferes with the miRNA pathway to suppress antiviral silencing	Dunoyer et al. (2004) and Merai et al. (2006)
	Tobamovirus	ORMV	(+)ssRNA	P126	Interferes with the methylation pathway mediated by HEN1 and the accumulation of novel miRNA-like sRNAs	Vogler et al. (2007)
		TMV	(+)ssRNA	P126, P122	P126 mechanism of suppression is unclear, while P122 suppresses RNA silencing through siRNA and miRNA binding and downregulates AGO1 via upregulation of miR168	Csorba et al. (2007) and Wang et al. (2012)
		ToMV	(+)ssRNA	P130	Suppresses silencing signals through siRNA binding	Kubota et al. (2003)
	Tobravirus	TRV	(+)ssRNA	16 K	Blocks local silencing via down streaming of dsRNA biogenesis	Martinez-Priego et al. (2008)
	Furovirus	CWMV, SBWMV	(+)ssRNA	19 K (CRP)	Suppresses silencing signal, thereby enhancing PVX pathogenicity, and interacts with N-ext/CP	Te et al. (2005) and Sun et al. (2013)
Benyviridae	*Benyvirus*	BNYVV, BSBMV	(+)ssRNA	P14	Its VSR activity requires both ZF and NoLS basic residues	Chiba et al. (2013)
		BNYVV	(+)ssRNA	P31	Plays a role in root-specific silencing suppression and interacts with PR-10	Rahim et al. (2007) and Wu et al. (2014)
Tymoviridae	*Tymovirus*	TYMV	(+)ssRNA	P69	Inhibits DNA methylation, boosting miRNA and DCL1 upregulation	Chen et al. (2004)
		CVB	(+)ssRNA	P12	Its VSR activity is ZF domain-dependent but independent of NLS. Promotes PVX infection	Lukhovitskaya et al. (2014)
Alphaflexiviridae	*Potexvirus*	PVX, PlAMV, PepMV	(+)ssRNA	P25 (TGBp1)	Suppresses RNA silencing via degrading RNA silencing effector nuclease AGO1, co-aggregation with SGS3/RDR6, CAT1 interaction, and X-body organization	Chiu et al. (2010), Tilsner et al. (2012), Mathioudakis et al. (2013) and Okano et al. (2014)
		PepMV	(+)ssRNA	CP	Sequesters silencing signals by inhibiting systemic signaling	Mathioudakis et al. (2014)

Table 2 (continued)

Family	Genus	Species	Genome	VSR	Viral strategies to suppress antiviral RNA silencing	References
Betaflexiviridae	*Carlavirus*	PVM	(+)ssRNA	CRP, TGBp1	TGBp1 suppresses merely systemic silencing, whereas CRP inhibits both local and systemic silencing	Senshu et al. (2011)
		SPCFV	(+)ssRNA	NaBp	Suppresses local and systemic silencing, mediated by either sense or dsRNA molecules	Deng et al. (2015)
	Trichovirus	ACLSV	(+)ssRNA	P50	Suppresses silencing systemically, probably by preventing the movement of silencing signals	Yaegashi et al. (2007)
	Vitivirus	GVA	(+)ssRNA	P10	Suppresses local and systemic silencing by siRNA binding	Zhou et al. (2006)
	Citrivirus	CLBV	(+)ssRNA	MP	Interferes with silencing pathways via downregulating the biogenesis of dsRNA and siRNA	Renovell et al. (2012)
Solemoviridae	*Sobemovirus*	RYMV	(+)ssRNA	P1	Suppresses RNA silencing through reduction of 21–24 nt siRNAs and deregulation of DCL4-dependent endogenous siRNA pathways	Lacombe et al. (2010)
		CfMV	(+)ssRNA	P1, CP	P1 shows weak systematic suppression without siRNA binding, while CP suppresses antiviral RNA silencing, but the mechanism of suppression is not clear	Sarmiento et al. (2007) and Olspert et al. (2014)

ACLSV Apple chlorotic leaf spot virus, *AgMV* Agropyron mosaic virus, *AV-2* Asparagus virus 2, *BCTV* Beet curly top virus, *BNYVV* Beet necrotic yellow vein virus, *BSBMV* Beet soil-borne mosaic virus, *BSMV* Barley stripe mosaic virus, *BWYV* Beet western yellows virus, *BYV* Beet yellow virus, *CABYV* Cucurbit aphid borne yellows virus, *CaMV* Cauliflower mosaic virus, *CCYV* Cucurbit chlorotic yellow virus, *CfMV* Cocksfoot mottle virus, *CIRV* Carnation italian ringspot virus, *CLBV* Citrus leaf blotch virus, *CLRDV* Cotton leafroll dwarf virus, *CMV* Cucumber mosaic virus, *CNV* Cucumber necrosis virus, *CPMV* Cowpea mosaic virus, *CTV* Citrus tristeza virus, *CVYV* Cucumber vein yellowing virus, *CWMV* Chinese wheat mosaic virus, *CVB* Chrysanthemum virus B, *CYDV* Cereal yellow dwarf virus, *CYSDV* Cucurbit yellow stunting disorder virus, *CymRSV* Cymbidium ringspot virus, *GBNV* Groundnut bud necrosis virus, *GLRaV-3* Grapevine leafroll-associated virus 3, *GRSV* Groundnut ringspot virus, *GVA* Grapevine virus A, *HCRSV* Hibiscus chlorotic ringspot virus, *HoMV* Hordeum mosaic virus, *LCV* Lettuce chlorosis virus, *LNYV* Lettuce necrotic yellows virus, *MNSV* Melon necrotic spot virus, *ORMV* Oilseed rape mosaic virus, *PCV* Peanut clump virus, *PEMV-1* Pea enation mosaic virus-1, *PepMV* Pepino mosaic virus, *PFBV* Pelargonium flower break virus, *PlAMV* Plantago asiatica mosaic virus, *PLPV* Pelargonium line pattern virus, *PLRV* Potato leafroll virus, *PMWaV-1,-2* Pineapple mealybug wilt-associated virus 1,-2, *PoLV* Pothos latent virus, *PPV* Plum pox potyvirus, *PRSV* Papaya ringspot virus, *PSLV* Poa semilatent virus, *PVA* Potato virus A, *PVM* Potato virus M, *PVX* Potato virus X, *PVY* Potato Virus Y, *RCNMV* Red clover necrotic mosaic virus, *RDV* Rice dwarf virus, *RGDV* Rice gall dwarf virus, *RHBV* Rice hoja blanca virus, *RLRaV* Rose leaf rossette-associated virus, *RRSV* Rice ragged stunt virus, *RSV* Rice stripe virus, *RYMV* Rice yellow mottle virus, *RYSV* Rice yellow stunt rhabdovirus, *SBWMV* Soil-borne wheat mosaic virus, *SCFaV* Strawberry chlorotic fleck-associated virus, *SCMV* Sugarcane mosaic virus, *SCSMV* Sugarcane streak mosaic poacevirus, *SCYLV* Sugarcane yellow leaf virus, *SPCSV* Sweet potato chlorotic stunt virus, *SPMMV* Sweet potato mild mottle virus, *TAV* Tomato aspermy virus, *TBSV* Tomato bushy stunt virus, *TCV* Turnip crinkle virus, *TEV* Tobacco etch virus, *TICV* Tomato infectious chlorosis virus, *TMV* Tobacco mosaic virus, *ToCV* Tomato chlorosis virus, *ToMV* Tomato mosaic virus, *ToRSV* Tomato ringspot virus, *TriMV* Triticum mosaic virus, *TRV* Tobacco rattle virus, *TSWV* Tomato spotted wilt virus, *TuMV* Turnip mosaic virus, *TYLCCNV* Tomato yellow leaf curl China virus, *TYLCV* Tomato yellow leaf curl virus, *TYRV* Tomato yellow ring virus, *TYMV* Turnip yellow mosaic virus, *WDV* Wheat dwarf virus, *WSMV* Wheat streak mosaic virus, *ZYMV* Zucchini yellow mosaic virus

Meddling with methylation cycle and suppressing TGS

DNA viruses such as geminiviruses and their associated betasatellites encode VSRs to suppress TGS through inhibiting or degrading key regulators of the methylation cycle. For example, transcriptional activator protein (TrAP) of beet severe curly top virus (BSCTV), tomato golden mosaic virus (TGMV), and cabbage leaf curl virus (CaLCuV), the pathogenicity factor βC1 of tomato yellow leaf curl China virus (TYLCCNV) and associated betasatellite, C4 protein of cotton leaf curl Multan virus (CLCuMuV), and L2 protein of beet curly top virus (BCTV), as well as AL2 protein of CaLCuV, can compromise the methylation process by impeding its major regulatory enzymes. These enzymes include *S*-adenosyl methionine synthetase (SAMS), adenosine kinase (ADK), and *S*-adenosyl homocysteine hydrolase (SAHH), which are also essential for TGS (Fig. 2) (Yang et al. 2011; Csorba et al. 2015; Jackel et al. 2015; Ismayil et al. 2018). Most geminiviruses, such as TGMV and CaLCuV, encode TrAP protein to activate the viral gene transcription by inhibiting the histone methyltransferase KYP/SUVH4 activity (Castillo-Gonzalez et al. 2015; Guerrero et al. 2020). Other geminiviral factors, including Rep, V2, C4, and C5, can also suppress TGS by interfering with methylation factors (Rodríguez-Negrete et al. 2013; Wang et al. 2018).

Binding with dsRNA and cleaving vsiRNA

The binding of VSRs to dsRNA is a general approach for VSRs to suppress RNA silencing. For example, various VSRs such as P38, P14, NS3, and NSs from turnip crinkle virus (TCV), pothos latent virus (PoLV), rice stripe virus (RSV)/rice hoja blanca virus (RHBV), and tomato spotted wilt virus (TSWV)/groundnut bud necrosis virus (GBNV), respectively, bind to dsRNA and inhibit the vsiRNA biogenesis (Fig. 2) (Yang and Li 2018). Strikingly, the sweet potato chlorotic stunt virus (SPCSV)-encoded VSR, RNase3, disables RNA silencing via an endonuclease activity-dependent pathway by cleaving dsRNA and vsiRNA duplexes (Cuellar et al. 2009).

Interfering with RDR and DCL4

The P6 suppressor protein of cauliflower mosaic virus (CaMV) binds with a double-stranded RNA-binding protein 4 (DRB4) to compromise the function of DCL4 in RNA-mediated PTGS. Moreover, P6 can also interfere with RDR6-dependent trans-acting and secondary vsiRNA pathways through interacting with DCL4 and impairing its functions (Haas et al. 2008; Shivaprasad et al. 2008). The TCV-associated P38 protein interacts with AGO1 and alters DCL usage by boosting DCL1 levels, resulting in significant downregulation of DCL3 and DCL4 (Azevedo et al. 2010; Endres et al. 2010).

Binding with HEN and sequestrating vsiRNA

As briefly described above, vsiRNA duplexes undergo methylation mediated by HEN1 before they are loaded onto AGOs. This methylation protects sRNAs from exonuclease degradation, facilitating systemic spread of antiviral RNA silencing signals. To counteract host antiviral RNA silencing, HC-Pro protein of zucchini yellow mosaic virus (ZYMV) binds to HEN1 and inhibits its methyltransferase activity (Jamous et al. 2011). Several VSRs, including 2b protein of cucumber mosaic virus (CMV), p19 protein of tomato bushy stunt virus (TBSV), HC-Pro protein of turnip mosaic virus (TuMV), and p21 protein of beet yellow virus (BYV), can bind to miRNA and vsiRNA duplexes and prevent HEN1-mediated methylation (Burgyan and Havelda 2011; Duan et al. 2012). The sequestration of vsiRNA by VSRs has been demonstrated to inhibit the plant-mediated antiviral RNA silencing pathways.

Degrading AGOs and interfering with RISC assembly

The AGO family is characterized as a critical component of RISC assembly, and plays key regulatory functions during the process of antiviral RNA silencing. Several VSRs interact with AGO1 and suppress RNA silencing response (Müller et al. 2020), such as the *Polerovirus* F-box P0, the tomato ringspot virus (ToRSV) CP, and the TCV P38; they all target AGO1 and trigger its instability and degradation (Azevedo et al. 2010; Karran and Sanfacon 2014; Derrien et al. 2018). Interestingly, the 16K protein of tobacco rattle virus (TRV) interacts with AGO4 and interferes with the assembly of RISC (Fernandez-Calvino et al. 2016). Similarly, the P1 protein of sweet potato mild mottle virus (SPMMV) suppresses RNA silencing by restricting RNA binding to AGO1, thus negatively regulates RISC assembly (Kenesi et al. 2017).

Interacting with miRNA and upregulating pre-miRNA

The miRNA is a discrete class of sRNA that triggers an antiviral defense against viral infection. However, several VSRs can interact with miRNA pathways to disrupt this antiviral function through upregulation of pre-miRNA. For instance, the rice gall dwarf virus (RGDV) PNS11 protein and the TBSV P19 protein suppress antiviral RNA silencing through interacting with miRNA pathways, including miRNA methylation (Yu et al. 2006; Burgyan and Havelda 2011; Shen et al. 2012). Similarly, HC-Pro from papaya ringspot virus (PRSV) and tobacco etch virus (TEV), and P19 from cymbidium ringspot virus (CymRSV) repress AGO1 accumulation via upregulation of pre-miR168, leading to the suppression of antiviral RNA silencing (Varallyay et al. 2010; Varallyay and Havelda 2013; Azad et al. 2014).

Meddling with the amplification of antiviral RNA silencing

The amplification of RNA silencing is most important for the inhibition of viral infection. Therefore, plant recruits several RDR proteins and RNA-helicases (SGS3 and SDE5), the extremely crucial components in antiviral RNA silencing pathways, to perform this amplification. Plant viruses of different groups encode VSRs to interact with RDR6 and SGS3, resulting in the suppression of RDR-mediated pathways. Some VSRs, such as VPg and HC-Pro from potyviruses, βC1, V2, and AC2 from geminiviruses, P2 from RSV, P6 from rice yellow stunt rhabdovirus (RYSV), and TGBp1 from plantago asiatica mosaic potexvirus (PlAMV), can bind to SGS3 and RDR6, and trigger the suppression of antiviral RNA silencing (Du et al. 2011; Guo et al. 2013; Cheng and Wang 2017) (Table 2, Fig. 2). Among them, V2 of CLCuMuV may also target calmodulin (CaM) to suppress anti-RNAi defense (Wang et al. 2021). In addition, V2 of TYLCV binds to SGS3 and suppresses RNA silencing locally (Glick et al. 2008). Whereas, βC1 protein of TYLCCNV satellite suppresses RDR6 expression and secondary vsiRNA production by upregulating an endogenous suppressor of RNA silencing (rgsCAM) (Li et al. 2014). Moreover, both 2b of CMV and p22 of tomato chlorosis virus (ToCV) interact with RDR6 to inhibit secondary vsiRNA biosynthesis (Landeo-Rios et al. 2016b).

Inhibiting the spread of antiviral RNA silencing

In virus-infected plants, local and systemic spread of silencing signal molecules constitutes the basis of antiviral RNA silencing pathways. To establish successful infection, viruses encode VSRs accordingly to target key regulators of these antiviral pathways. The Rep protein of wheat dwarf virus (WDV) suppresses RNA silencing locally and systemically through binding with vsiRNA duplexes (Wang et al. 2014). The P6 protein of RYSV suppresses systemic RNA silencing by constraining RDR6-mediated biosynthesis of secondary vsiRNAs. Similarly, the CMV-encoded 2b protein block the translocation of RNA silencing signals and inhibit their spread (Guo and Ding 2002). Moreover, the potato virus X (PVX) suppressor protein (p25) inhibits the production of RNA silencing signals and interferes with their propagation (Voinnet et al. 2016).

Viral proteins of closterovirids to counteract antiviral RNA silencing

GLRaV-2 p24 protein as a VSR

The p24 protein of GLRaV-2 is a strong VSR encoded by a cryptic ORF in the viral genome. It can effectively block siRNA accumulation and suppress antiviral RNA silencing (Wang et al. 2019). The N-terminus (amino acids 1–188) of p24 is a main functional region, comprising all predicted α-helicases and β-strands that are most essential for its VSR activity. Moreover, self-interaction, pathogenicity, and silencing suppression of p24 are associated with its hydrophobic residues (135/F38/V85/V89/W149, V162/L169/L170). Specifically, p24 suppresses RNA silencing by adopting an RNA-binding strategy. Substituting two basic amino acid residues at positions 2 and 86, which are involved in RNA-binding, totally negates the VSR activity of p24 (Liu et al. 2016). W54 in the WG/GW-like motif (W54/G55) is also primarily significant for retaining p24 suppressor activity (Li et al. 2018). Furthermore, p24 has no physical interaction with AGO1 of *N. benthamiana*, and it up-regulates mRNA expression of AGO1 without boosting AGO1 degradation. This impact is specifically correlated with the VSR activity of p24, demonstrating that it may hamper miRNA-directed processes (Li et al. 2018).

GLRaV-3 p19.7 protein as a VSR

The 3′ end monopartite RNA genome of GLRaV-3 encodes a VSR (p19.7) with a molecular weight of around 20 kDa. The p19.7 protein demonstrates VSR activity in various silencing induction systems. This VSR shares several characteristics with p21, a BYV suppressor protein capable of overcoming powerful silencing inducers (Gouveia et al. 2012). In co-infiltration assays, the VSR activity of p19.7 varies among GLRaV-3 variants, resulting in variable levels of accumulation of green fluorescent protein (GFP) mRNA and specific siRNA in the transgenic *N. benthamiana* line 16c. A comparison analysis of protein sequences demonstrated that the substitutions of a few amino acids in p19.7 may be linked to the variation in its suppression activity (Gouveia and Nolasco 2012).

PMWaV-1 p61 protein as a VSR

The p61 protein is reported to have a systemic silencing suppressor activity up to 18 days post-infiltration (dpi) among the 3′-proximal ORFs of PMWaV-1 that encode p61, p24, CP, and Hsp70, while no protein with local suppressor activity was identified (Dey et al. 2015).

PMWaV-2 p20 and CP proteins as VSRs

It has been stated that the two 3′-proximal ORFs of PMWaV-2 encode CP and p20 proteins, which adversely affect the induction of secondary siRNAs and prevent the systemic spread of antiviral silencing signals. Therefore, CP and p20 proteins affect RNA silencing (both local and systemic) in *N. benthamiana*, while CPd and p22 proteins solely mitigate systemic silencing. In addition, p20 protein suppresses dsRNA-induced local silencing,

specifically, when there are lower levels of dsRNA. Furthermore, it has also been indicated that p20 can boost the infectivity of PVX in *N. benthamiana* (Dey et al. 2015).

BYV p21 protein as a VSR

The 3′-proximal genomic region of BYV encodes a 21 kDa protein (p21). The p21 protein belongs to an important gene family of alike proteins that are possessed by other viruses in the *Closterovirus* genus, exhibiting a silencing suppressor role. The p21 is capable of interfering and suppressing dsRNA-induced silencing of GFP mRNA, however, BYSV- and CTV-encoded VSRs, p22 and p20, only show weak suppressor activity as compared to BYV p21. BYV p21 exhibits its VSR functionality in two model systems. In the first system, dsRNA induces a robust silencing of reporter mRNA, while in the second system, weak silencing is initiated via ectopic expression of mRNA from a stronger promoter (Reed et al. 2003). Furthermore, the crystalline structure of p21 shapes an RNA binding octameric ring architecture with a large central cavity of 90 Å diameter and the inner surface of the ring is positively charged. Tombusviruses VSRs, p19 and p21, both inhibit silencing through binding to siRNA directly. Contrastingly, besides interacting with peculiar dimeric-structured p19-siRNA duplex, BYV p21 is also a nucleic acid-binding protein that interacts in vitro with 21 nt or longer ssRNAs and dsRNAs. This p21-adopted precise RNA binding structure highlights various open challenges to study the structure-based interaction mechanism of other VSRs (Ye and Patel 2005).

CTV CP, p20, and p23 proteins as VSRs

Three distinct VSRs such as CP, p20, and p23 are encoded by the 3′-proximal region of large RNA genome of CTV, protecting virus from the antiviral silencing machinery of its perennial woody citrus host (Hajeri et al. 2014). Unlike p20 and other VSRs known to interfere with intercellular silencing, such as CMV 2b and PVX p25, CP is a unique VSR that suppresses antiviral RNA silencing pathways at an intercellular level, and this is not associated with intracellular silencing suppression. CMV 2b and p20 share features in silencing suppression, and are potent suppressors of intercellular silencing but incomplete in suppressing intracellular silencing, however, unlike CMV 2b, the intercellular silencing suppression of p20 is not linked to substantially declined DNA methylation of the target GUS transgene (Lu et al. 2004; Benitez-Galeano et al. 2015). In terms of local silencing suppressor activity, p23 is the most effective VSR among the three aforementioned CTV VSRs, and its localization is restricted to both the plasmodesmata and nucleus. Although p23 is a strong intracellular silencing suppressor like HC-Pro, it does not restrict intercellular silencing and DNA methylation of the target transgene. There are many conserved amino acids in p23 and mutations of E95A/V96A and M99A/L100AA may compromise its VSR activity and stability, furthermore, deletions of Q93A and R143A/E144A completely abolish its VSR activity (Li et al. 2019). Moreover, ectopic expression of p23 enhances CTV accumulation and dispersion in woody hosts and, in addition, promotes systemic infection of the resistant sour orange host (Fagoaga et al. 2011).

RLRaV p17 protein as a VSR

RLRaV encodes a 17 kDa protein (p17). Its BLASTp search results indicate conserved motif characteristics of the viral suppressor p20 superfamily, such as p23 or p20 of RLMV, CTV, and SCFaV. RLRaV uses p17 as a VSR to combat antiviral RNA silencing response in wild roses (*Rosa multiflora* Thumb) upon the occurrence of wild rose leaf rosette disease (He et al. 2015).

CYSDV p25 protein as a VSR

The papain-like protease proteins, including p25, p5.2, and p22, were hypothesized to have VSR activity. However, only p25 has been identified as a PTGS suppressor of CYSDV. Its VSR strategy is to suppress dsRNA- or ssRNA-induced silencing of GFP mRNA. In plant tissues where silencing is already established, it is unable to prevent silencing signals from spreading locally and restoring GFP expression. Therefore, p25 has no ostensible effects on the accumulation of siRNAs (Kataya et al. 2009).

CCYV p22 protein as a VSR

To combat antiviral RNA silencing, a putative suppressor protein p22 binds with CsSKP1LB, a *Cucumis sativus* ortholog of S-phase kinase-associated protein 1 (SKP1). It is reported that the F-box-like motif in this protein is most crucial for p22-mediated viral pathogenicity and VSR activity (Chen et al. 2019). CCYV p22 suppresses antiviral RNA silencing at a local level and does not affect local or systemic movement of RNA silencing signals. Moreover, it is comparatively weaker in suppressing local RNA silencing than ToCV p22 and CYSDV p25, the two well-known VSRs of criniviruses (Orfanidou et al. 2019).

LCV p23 protein as a VSR

LCV employs p23 as a sophisticated tool for evasion of host antiviral defense and modulation of disease symptoms. It has been demonstrated that p23 suppresses initiation of local silencing and enhances degradation as well as inhibits accumulation of siRNAs in infiltrated leaves. This protein may also inhibit cell-to-cell and long-distance movement of RNA silencing signals in

GFP-transgenic *N. benthamiana* (line 16c). At an elevated incubation temperature, p23-agroinfiltrated *N. benthamiana* leaves exhibit localized necrosis and an increased disease severity (within 5 dpi). This is a novelty about the direct temperature effect on VSR, which has been given to p23 of LCV and has never before been reported in other viruses (Kubota and Ng 2016).

SPCSV RNase3 and p22 as VSRs

SPCSV genomic RNA demonstrates variability in gene contents at 3′ proximal ORFs. Therefore, molecular analysis shows heterogeneity at the p22- and RNase3-encoding regions of various SPCSV isolates. The Ugandan SPCSV isolate encodes p22 and RNase3 proteins, which are involved in RNA silencing suppression. RNase3 displays endonuclease activity and cleaves siRNA into ~ 14 nt products, enhancing VSR activity of p22. Furthermore, RNase3 expression alone in sweet potato plants is enough for breaking plant resistance to sweet potato feathery mottle virus (SPFMV) and developing this viral disease (Cuellar et al. 2008).

ToCV p22, CP, and CPm as VSRs

The bipartite genomic RNAs of ToCV encode multiple VSRs with diverse functions to compromise host antiviral defense. Upon heterologous expression, CP and CPm induce obvious systemic symptoms in *N. benthamiana*, including leaf curling, necrotic mottling, and leaf deformation within 5 dpi (Canizares et al. 2008). Meanwhile, p22 is a strong VSR that preferentially binds to long dsRNAs via a putative zinc finger motif, preventing them from being diced into siRNAs (Landeo-Rios et al. 2016b). By using a ToCV infectious clone based on ToCV-BJ isolate, a consistent expression of p22 was detected in *N. benthamiana* leaves (Zhao et al. 2013, 2016). Furthermore, p22 interferes with the auxin signaling pathway through binding to SKP1.1 via its C-terminus and disrupting SCF complex formation by competing with transport inhibitor response 1 (TIR1) to promote viral infection (Liu et al. 2021). RdRps play a significant role in antiviral defense, and the RDR6 of *N. benthamiana* is also involved in defense via combatting ToCV. In *rdr6* mutant, p22 is dispensable for ToCV replication, while during systemic infection, p22 counteracts RDR6-mediated antiviral response (Landeo-Rios et al. 2016a). However, the heterologous expression of p22 causes an exacerbation of disease symptoms and ultimately the death of whole plants, although it could not complement suppressor-defective mutant viruses (Landeo-Rios et al. 2017).

TICV-p27 as a VSR

The ORF2 of TICV-RNA1 encodes p27, with a genomic location similar to other criniviruses. It has been reported that p27 can suppress a sense transgene-induced PTGS (S-PTGS) and an inverted repeat-induced PTGS (IR-PTGS) without inhibiting local and systemic movement of RNA silencing signals. Furthermore, upon heterologous expression, p27 induces more severe mosaic and necrosis symptoms with the accumulation of a heterologous virus (Mashiko et al. 2019).

Plant strategies to counter viral suppression of RNA silencing

Plants have evolved counter-suppression pathways to combat viral infection in response to VSR-mediated RNA silencing suppression. Primarily, upon recognizing invading viruses, the plant immune system upregulates the expression of resistance R genes (NBS-LRR), triggering host defensive components (i.e., R proteins, monitor or guard) to guard antiviral RNA silencing response (Shao et al. 2019). In addition to regulating immune system, plants may target VSRs directly to counter the RNA silencing suppression mechanism. For instance, the interaction of the TuMV suppressor protein VPg with SGS3 and RDR6 activates a versatile cellular mechanism in host plants, facilitating the degradation of the RDR6-SGS3-VPg complex through autophagy pathways and ubiquitination to boost host antiviral RNA silencing (Cheng and Wang 2017). Similarly, the calmodulin-like protein in tobacco (rgs-CaM) binds to VSRs, including 2b of CMV and tomato aspermy virus (TAV) and HC-Pro of TuMV, to inhibit RNA silencing suppression through autophagy-mediated degradation of these VSRs (Nakahara et al. 2012). Plants may also trigger antiviral immune responses via regulating the components of RNA silencing pathway, such as dsRNAs. DsRNAs are conserved molecular patterns produced during virus replication and can induce PTI signaling pathway in plants. This dsRNA-mediated antiviral PTI, which requires pattern-recognition co-receptor SERK1 rather than DCLs, elicits antiviral protection independent of RNA silencing. However, the underlying mechanism of the corresponding signaling pathway is still a matter of consideration (Niehl et al. 2016; Niehl and Heinlein 2019). Hence, plant antiviral RNA silencing, viral suppression of RNA silencing, and plant counter-suppression may result in an endless battle of survival between viruses and plants.

Conclusions and future perspectives

It is now evident that antiviral RNA silencing, together with its rudimentary role in antiviral defense, establishes a fundamental regulatory hub in plant immunity to counter large numbers of viral pathogens. However, antiviral RNA silencing pathways should evolve continuously and rapidly due to diverse silencing suppression strategies of VSR. Mostly, viral suppressors hinder RNA silencing

pathways by targeting essential elements of siRNAs and miRNAs or proteins like DCLs and AGOs. The interconnections between VSRs and host factors have demonstrated that a single viral suppressor can target several elements in a silencing pathway. For example, potyvirus HC-Pro may interfere with the miRNA pathway via sequestering siRNA biogenesis, downregulating AGO1 expression, and preventing 3′ end methylation of siRNA (Azad et al. 2014; Pollari et al. 2020). Similarly, closterovirid p22 may also compromise the silencing pathway in several ways, including dsRNA binding, counteracting RDR6-mediated antiviral response, accelerating disease symptoms, and ultimately causing plant death upon heterologous expression (Landeo-Rios et al. 2016b) (Fig. 2). Even though investigations into the mechanism of VSR have been on the frontline for more than a decade, many aspects are still indefinable. Interestingly, several VSRs, such as CP, movement protein, protease, and replicase, have analogous functions. Therefore, silencing activities and non-silencing functions should be coordinated to accomplish various tasks and attain optimal infection.

The structure-based interaction mechanism of VSRs is still elusive, although the precise adopted RNA binding octameric ring structure of closterovirid p21 and specific dimeric structured p19-siRNA duplex interaction have revealed various open challenges to investigate structure-based interaction mechanisms of other VSRs (Ye and Patel 2005). In addition, VSRs are supposed to be major contributors to viral disease induction and symptom development due to their negative effects on endogenous small RNA accumulations (Diaz-Pendon et al. 2007). Regardless of the paramount importance of VSRs in counteracting PTGS, their symptom induction is a less-studied aspect of the virus infection process. Furthermore, the direct temperature impact on VSR in modulation of disease symptoms needs more investigations. So far, only p23 of LCV was reported to evade host antiviral defense at elevated temperature (Kubota and Ng 2016).

The molecular characteristics of VSRs are much more complex than we thought. Indeed, the p38 protein of TCV, in addition to dsRNA binding, specifically interferes with the biogenesis of primary vsiRNA through downregulation of DCL4 (Azevedo et al. 2010). Similarly, the p19 protein of CymRSV confiscates vsiRNA through binding with 21–25 nt dsRNA and upregulating miR168 expression, resulting in arrests of antiviral AGO1 translation (Silhavy et al. 2002; Varallyay et al. 2010). Some other VSRs may interact with RNA silencing pathways in multiple ways, which need further investigations.

There are still a lot of lapses in knowledge about plant effectors regarding their silencing mechanisms and mi/siRNA RISC assemblies or some components of plant RISCs, which are potential targets of VSRs. Therefore, VSRs like p38, P1, and P0 may be used as a powerful tool to explore the RISC complexes in future research.

Plant resistant (R) proteins have evolved to recognize strategies of VSRs against RNA silencing. Therefore, the identity of such dedicated proteins, specifically in guarding the RNA silencing components among the plethora of plant R proteins, is an important future question. Presumably, hosts can also neutralize VSRs through appropriate defensive activities that degrade or displace them into inappropriate subcellular compartments. The former probably has explained that the alleles of CMV-encoded VSR 2b protein fail to accumulate in Arabidopsis because of proteolysis, while nuclear re-localization of tombusviral p19 is caused by its interaction with plant ALY protein (Canto et al. 2006; Zhang et al. 2006). These observations of host-directed suppression mechanisms of VSRs and their polymorphic alleles may propose a future direction to study the variations in viral vulnerability between species or subspecies.

Abbreviations

ADK: Adenosine kinase; AGO: Argonaute; BCTV: Beet curly top virus; BMV: Brome mosaic virus; BSCTV: Beet severe curly top virus; BYSV: Beet yellow stunt virus; BYV: Beet yellow virus; CaLCuV: Cabbage leaf curl virus; CaM: Calmodulin; CaMV: Cauliflower mosaic virus; CCYV: Cucurbit chlorotic yellow virus; CLCuMuV: Cotton leaf curl multan virus; CMV: Cucumber mosaic virus; CTV: Citrus tristeza virus; CymRSV: Cymbidium ringspot virus; CYSDV: Cucurbit yellow stunting disorder virus; DCL: Dicer like; DRB4: Double-RNA binding protein 4; ER: Endoplasmic reticulum; ETI: Effector-triggered immunity; GBNV: Groundnut bud necrosis virus; GLRaV-3: Grapevine leafroll-associated virus 3; HC-Pro: Helper component-proteinase; HEN1: HUA enhancer 1; HSP70h: Heat-shock proteins HSP70 homology; HR: Hypersensitive responses; ICTV: International Committee on Taxonomy of Viruses; IR-PTGS: Inverted repeat-induced PTGS; LCV: Lettuce chlorosis virus; miRNAs: MicroRNAs; MVBaV: Mint vein banding-associated virus; PAMPs: Pathogen-associated molecular patterns; PlAMV: Plantago asiatica mosaic potexvirus; PMWaV-1: Pineapple mealybug wilt-associated virus 1; PMWaV-2: Pineapple mealybug wilt-associated virus-2; Pol II: Polymerase II; PoLV: Pothos latent virus; pri-miRNAs: Primary miRNAs; PRSV: Papaya ringspot virus; PTGS: Post-transcriptional gene silencing; PTI: PAMP-triggered immunity; PVX: Potato virus X; RdRp: RNA-dependent RNA polymerase; RDR: Cellular RdRps; RGDV: Rice gall dwarf virus; RHBV: Rice hoja blanca virus; RISC: RNA-induced silencing complex; RLMV: Raspberry leaf mottle virus; RLRaV: Rose leaf rossette-associated virus; RSV: Rice strip virus; RYSV: Rice yellow stunt rhabdovirus; SAHH: *S*-adenosyl homocysteine hydrolase; SAMS: *S*-adenosyl methionine synthetase; SAR: Systemic acquired resistance; siRNAs: Small interfering RNAs; SKP1: S-phase kinase associated protein 1; SMD: Silencing movement deficient; SPCSV: Sweet potato chlorotic stunt virus; SPFMV: Sweet potato feathery mottle virus; SPMMV: Sweet potato mild mottle virus; S-PTGS: Sense-transgene-induced PTGS; TBSV: Tomato bushy stunt virus; TCV: Turnip crinkle virus; TEV: Tobacco etch virus; TGMV: Tomato golden mosaic virus; TGS: Transcriptional gene silencing; TICV: Tomato infectious chlorosis virus; TIR1: Transport inhibitor response 1; ToCV: Tomato chlorosis virus; ToRSV: Tomato ringspot virus; TrAP: Transcriptional activator protein; TRV: Tobacco rattle virus; TSWV: Tomato spotted wilt virus; TuMV: Turnip mosaic virus; TYLCCNV: Tomato yellow leaf curl China virus; UPS: Ubiquitin proteasome system; VAMP: Virus-associated molecular pattern; vsiRNAs: Virus-derived small interfering RNAs; VSR: Viral suppressor of RNA silencing; WDV: Wheat dwarf virus; ZYMV: Zucchini yellow mosaic virus.

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MDH and TZ conceived the idea and wrote the manuscript. MDH, TF and XC designed and made the Figures and Tables. MT, TJ, TF, SL, and TZ edited and revised the manuscript. All authors read and approved the final manuscript.

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