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Advances in understanding the soil-borne viruses of wheat: from the laboratory bench to strategies for disease control in the field

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Abstract

In China, soil-borne viruses transmitted by the root parasite *Polymyxa graminis* have caused significant yield loss in winter wheat for many years. At present, it is believed that two main soil-borne RNA viruses, namely wheat yellow mosaic virus (WYMV) and Chinese wheat mosaic virus (CWMV) are responsible for such losses. The molecular characteristics and infection processes of these two viruses have been intensively investigated and described substantially in detail, following the complete sequencing of their respective genomes. In this review, we highlight our recent findings on the distribution of WYMV and CWMV in China, the associated crop damage, the biological functions of WYMV and CWMV proteins as well as the viral temperature sensitivities. We also describe the characteristics of the resistance genes and discuss the novel virus–plant arms race strategies in hope of enlarging our understanding on the theme of virus-plant interactions. Finally, we compare current disease-management options and suggest the application of biotechnology-based genetic resistance to develop more cost-effective countermeasures for controlling soil-borne virus diseases in the future.

Keywords: Soil-borne virus disease, Yield loss, Disease control

Background

Wheat (*Triticum aestivum*) is one of the most important cereal crops worldwide. However, global wheat production is threatened by a wide range of abiotic stresses including cold, drought, and salt, as well as biotic stresses mainly represented by insect pests and diseases. Viral diseases alone can cause yield reduction of up to 70% in China (Chen 2005). Most viruses that naturally infect wheat are spread between plants by insect vectors (Zhang et al. 2017). However, soil-borne viruses transmitted by the root parasite *Polymyxa graminis* have also caused severe disease in winter wheat grown in China (Diao et al. 1999). In this review, we present an overview of the key soil-borne viruses infecting wheat grown in China and highlight recent progress made towards understanding these viruses with the primary focus on plant–virus interactions. Finally, we discuss new strategies for disease control and suggest future research directions.

Soil-borne viruses and the diseases they cause in wheat

The wheat viruses that have caused severe damage to wheat crops in many countries include wheat streak mosaic virus (WSMV) transmitted by the eriophyid wheat curl mite (Singh et al. 2018), wheat dwarf virus (WDV) transmitted by the leafhopper *Pasmmotettix alienus* (Abt et al. 2019), barley yellow dwarf viruses (BYDV) transmitted by aphids (Aradottir and Crespo-Herrera 2021) and soil-borne viruses transmitted by

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the zoospores of the soil-inhabiting plasmodiophoraceous microorganism *Polymyxa graminis* (Estes and Brakke 1966). Several soil-borne viruses are agriculturally important on autumn-sown wheat and are of two taxonomically distinct types. Wheat yellow mosaic virus (WYMV) in Asia (Sawada 1972) and wheat spindle streak mosaic virus (WSSMV) in Europe and North America (Sohn et al. 1994) are both classified in the genus *Bymovirus* (family *Potyviridae*). Wheat-infecting members of the genus *Furovirus* are Chinese wheat mosaic virus (CWMV) in Asia (Diao et al. 1999), soil-borne wheat mosaic virus (SBWMV) in the United States (McKinney 1923; Shirako et al. 2000) and soil-borne cereal mosaic virus (SBCMV) in Europe (Kanyuka et al. 2003). Here, we mainly focus on WYMV and CWMV which are widely distributed in China. Wheat yellow mosaic disease was first found in Sichuan in the 1960s and was first thought to be caused by WSSMV (Tao et al. 1980; Zhou et al. 1990). WYMV and WSSMV are easily confused because they have similar host range, serological characteristics, particle morphology, and vector (Li et al. 1999) but in 1999 sequence analysis showed that the virus in China was actually WYMV (Chen 1999). WYMV has spread gradually into the middle and lower valleys of the Yangtze and Huai rivers in China (Sun et al. 2013a). Typical symptoms of WYMV are mosaic or yellow-striped leaves on stunted plants, beginning as irregular patches of pale green or yellow on leaves at the early stage of infection and leading to stunted growth with a few abnormal tillers in the later stages of infection (Chen 1993). When wheat fields are severely affected by WYMV, all plants turn yellow (Fig. 1a). Wheat yellow mosaic disease typically causes yield losses of 10–30%, but can be as much as 70% in an epidemic year (Sun et al. 2013a). Results of nearly 20 years of field monitoring by our group, show that WYMV is present in Anhui, Henan, Hubei, Jiangsu, Sichuan, and Shaanxi provinces along the Yangtze River, whereas WSSMV has never been found in these samples (Fig. 2). WYMV also occurs in Japan where the virus populations have been classified into three pathotypes (I–III) based on their pathogenicity to wheat cultivars: pathotype I causes systemic infection in plants of the cultivars Nambukomugi and Fukuhokomugi, but not in plants of the cultivar Hokkai 240; Pathotype II causes a systemic infection only on Nambukomugi; Pathotype III causes infection on all three wheat cultivars (Ohki et al. 2014; Ohki et al. 2019). In 1999, CWMV was first isolated from winter wheat in Yantai, Shandong Province, China (Diao et al. 1999). It is mainly restricted to Shandong (cities: Yantai and Rongcheng), Henan (city: Haozhou), Hebei (city: Langfang) and Jiangsu (city: Dafeng) provinces (Sun et al. 2013a) (Fig. 2). Symptoms caused by CWMV are light chlorotic streaking on young leaves but bright yellow chlorotic streaking or even purple chlorotic stripes on older leaves. At the later stages of infection, the intermittent chlorosis on leaves can gradually develop into chlorotic stripes and the plants become stunted, wilt and later die (Guo et al. 2019) (Fig. 1b).

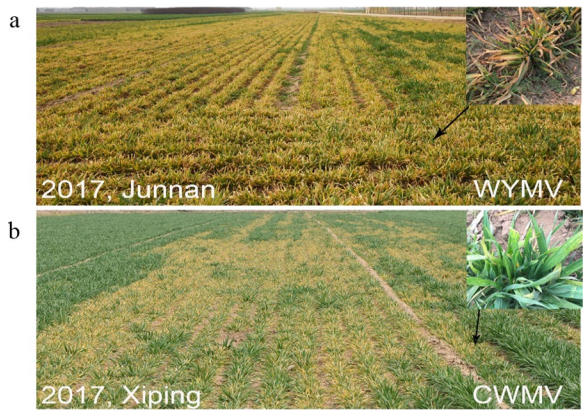

Fig. 1 Wheat plants showing lethal necrosis and mosaic. **a** The overall incidence of WYMV in a field in Junnan, Shandong Province. The small figure on the upper right shows an individual wheat plant infected with WYMV. **b** The overall incidence of CWMV in a field in Xiping, Henan Province. The small figure on the upper right shows an individual wheat plant infected with CWMV

Genetics of resistance against bymovirus-induced diseases

WYMV can be retained in soil within the dormant spores of the parasite *P. graminis* for many years (Chen 2005) and it is therefore difficult to control the viral disease once a field becomes infested with this viruliferous microorganism. The most environmentally friendly and effective measure for controlling the disease is to breed disease-resistant wheat varieties (Chen 1993; Ohto et al. 2006; Chen et al. 2014). However, climatic and environmental conditions greatly influence the severity of the disease and this makes it difficult to screen seedlings for resistance to WYMV within a plant breeding program. Nevertheless, great efforts have been devoted to identifying and mapping resistance genes, and several wheat varieties with high resistance to WYMV have been developed (Jiang et al. 2020), although the genetic and molecular mechanisms underlying this resistance are still poorly understood. Inherited resistance to WYMV/WSSMV appears to be complex and is influenced by many factors. Thus, the development and utilization of molecular markers to accurately select WYMV-resistant breeding materials is very important. Molecular-marker analysis in wheat is primarily based on microsatellite or simple-sequence repeat (SSR) markers (Conti et al. 2011). During the past decade, with the availability of efficient

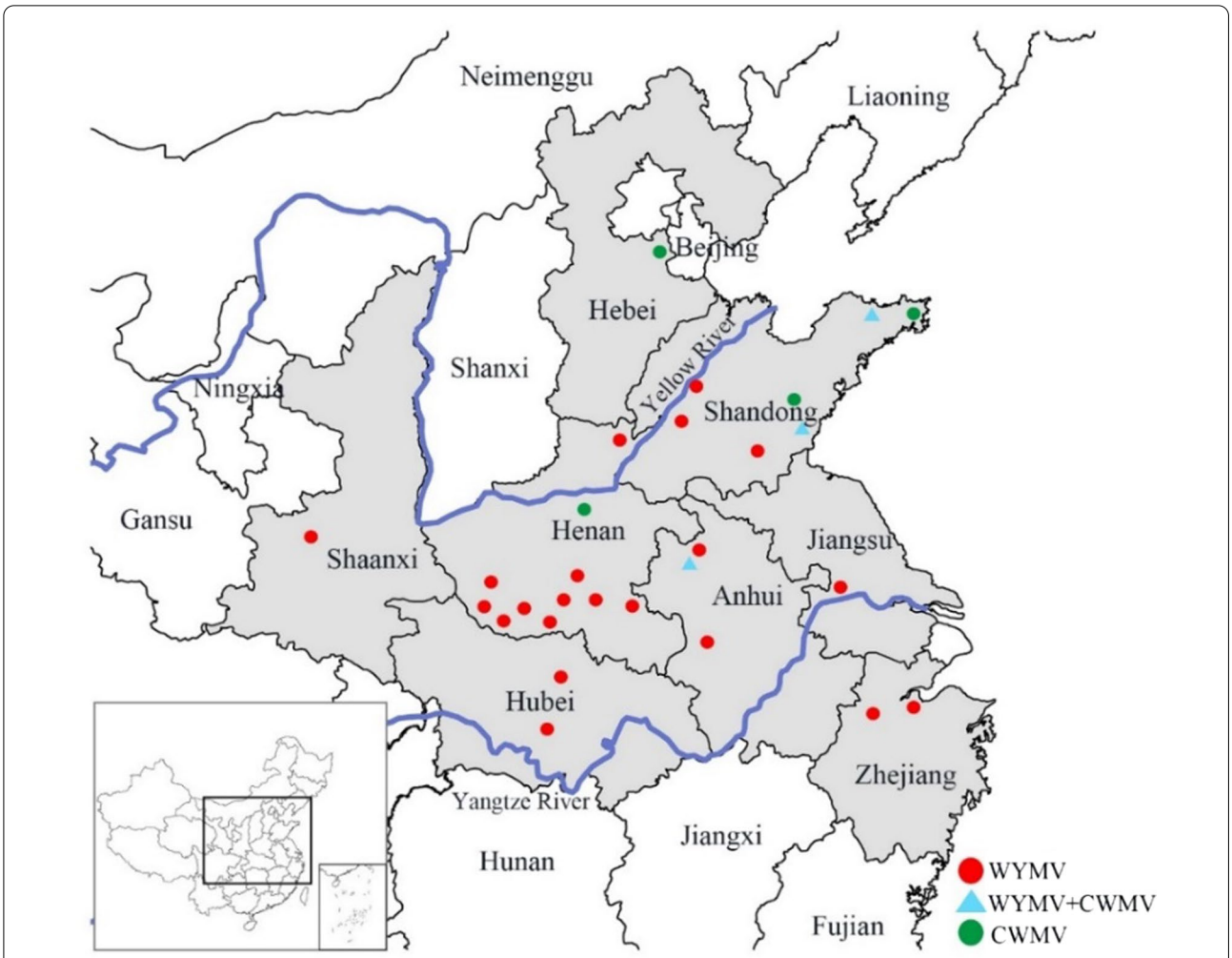

Fig. 2 Distribution of wheat yellow mosaic virus (WYMV) and Chinese wheat mosaic virus (CWMV) in China. Shading shows the extent of WYMV and CWMV occurrence in winter wheat. Samples with WYMV are shown as red circles, CWMV as green circles, and sites with both viruses as blue triangles

tools such as molecular-marker techniques and sophisticated software packages, the number of identified quantitative trait loci (QTLs) associated with disease resistance has considerably increased. According to earlier studies, WYMV resistance is quantitative and controlled by 1 to 3 major genes (Liu et al. 2004). Sustained efforts have been made to mine genetic resources, including WYMV-resistant germplasms, and to identify associated molecular markers. Thirteen genes or QTL, which are resistant against wheat-infecting bymoviruses, have been identified on chromosomes 2A, 2DL, 3BS, 5AL, 6DS, 7A and 7BS (Table 1). Furthermore, an exogenetic WYMV- or WSSMV-resistant gene *Wss1* was found on chromosome 4VS of *Haynaldia villosa*, a wild relative of wheat (Zhang et al. 2005). Wheat varieties from different countries carry genetic resistance loci to WYMV on chromosome 2DL (Table 1), indicating that this chromosomal region plays an important role in WYMV resistance. These QTLs may be allelic, and therefore pyramiding them is not a viable option.

Genome organization and functions of viral proteins

The genome organization of the furovirus CWMV and the function of its encoded viral proteins have been described in detail in a previous review (Guo et al. 2019) and will not therefore be repeated. Here, we mainly focus on the genome structure and function of WYMV, which has two genomic RNAs. WYMV RNA1 (~7.6 Kb) encodes a large polyprotein (~270 kDa) that is processed by a protease to generate eight mature proteins, namely the third protein (P3), 7-kDa peptide (7 K), cylindrical

Table 1 Summary of bymovirus resistance genes and QTLs in wheat

No	Resistance gene	Chromosomal location	Resistance donor (origin)	Resistance against virus	References
1	*Xbcd1095*	2DL	Geneva (US)	WSSMV	Khan et al. (2000)
2	*Xcdo373*	2DL	Geneva (US)	WSSMV	Khan et al. (2000)
3	*YmYF*	2DL	Yangfu9311 (China)	WYMV	Liu et al. (2005b)
4	*Wss1*	T4DL·4VS	*Haynaldia villosa* (wild relative)	WSSMV/WYMV	Zhang et al. (2005), Zhao et al. (2013), Dai et al. (2020)
5	*YmNM*	2A	Ningmai9 (China)	WYMV	Liu et al. (2004), Liu et al. (2005a)
6	*Ym1b*	2DL	Ibis (Netherland)	WYMV	Nishio et al. (2010)
7	*YmMD*	2DL	Madsen (US)	WYMV	Takeuchi et al. (2010)
8	*QYm.njau-5A.1*	5AL	Xifeng wheat (Japan)	WYMV	Zhu et al. (2012)
9	*QYm.njau-3B.1*	3BS	Xifeng wheat (Japan)	WYMV	Zhu et al. (2012)
10	*QYm.njau-7B.1*	7BS	Xifeng wheat (Japan)	WYMV	Zhu et al. (2012)
11	*Qym1*	2DL	Madsen (US) Hokkaido (Japan)	WYMV	Suzuki et al. (2015)
12	*Qym2*	3BS	Madsen (US) Hokkaido (Japan)	WYMV	Suzuki et al. (2015), Liu et al. (2016)
13	*Q.Ymym*	2DL	Yumechikara (Japan)	WYMV	Kojima et al. (2015)
14	*Qym4*	6DS	OW104 (Japan)	WYMV	Yamashita et al. (2020)

inclusion protein (CI), 14-kDa peptide (14 K), viral protein genome-linked (VPg), nuclear inclusion-a protease (NIa-Pro), nuclear inclusion "b" protein (NIb), and coat protein (CP). Researchers have speculated that a small open reading frame (ORF), termed *PIPO,* is also present overlapping with the P3-coding region (Chung et al. 2008; Sun et al. 2013d). WYMV RNA2 (~3.5 Kb) encodes a polyprotein (~100 kDa) that is cleaved to give rise to two mature proteins, named P1 and P2 (Fig. 3) (Namba et al. 1998; Sun et al. 2013d). Although very few detailed functional studies have been performed on the putative WYMV-encoded proteins, their functions can be inferred from well-characterized homologs in other members of the family. Among the proteins encoded by RNA1, the primary function of WYMV CP is to encapsidate the genomic RNA to form linear virus particles. CP regulates the assembly or disassembly of viral particles by interacting with viral RNA and then participates in viral replication and translation (Fig. 3 and Table 2) (Yang et al. 2021). The CPs of two potyviruses, tobacco etch potyvirus (TEV) and turnip mosaic virus (TuMV), are also indispensable for viral cell-to-cell transport but not for viral genome replication (Dolja et al. 1995; Li and Shirako 2015; Dai et al. 2020). It is suggested that WYMV CP may have similar function. The WYMV CI protein comprises two domains that are predicted to possess helicase activity (Deng et al. 2015). Moreover, this versatile protein forms a laminate or pinwheel-shaped inclusion in the cytoplasm of infected cells, which is typical of infection by potyvirids (Sorel et al. 2014). Indeed, substantial genetic and cell biology data have revealed that CI is present in the viral replication complex (VRC), where it can contribute to viral genome replication by unfolding the viral RNA duplexes (Rodríguez-Cerezo et al. 1997; Rojas

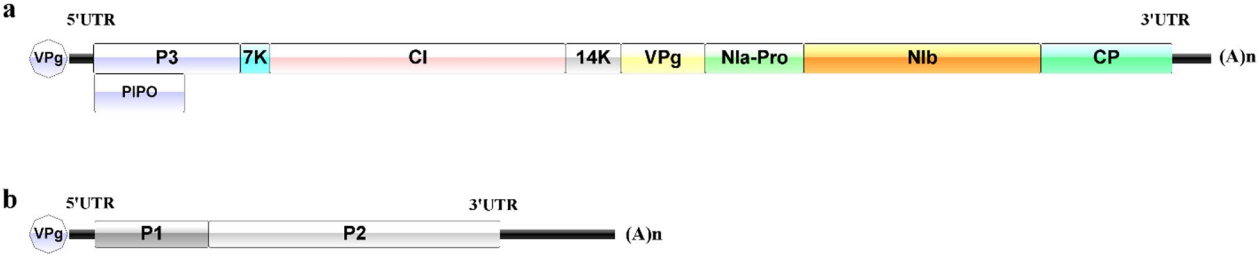

Fig. 3 Schematic diagram of the gene expression strategy and genomic organization of wheat yellow mosaic virus (WYMV). **a** RNA 1 of WYMV. A single large polyprotein is cleaved into the eight mature proteins shown within the smaller boxes that make up the ORF. P3N-PIPO derives from frameshifting of the P3 protein, and (A)n represents the poly(A) tail. 7 K, 7-kDa peptide; CI, cylindrical inclusion protein; 14 K, 14-kDa peptide; VPg, viral protein genome-linked; NIa-Pro, nuclear inclusion a-protease; NIb, nuclear inclusion b; CP, coat protein. **b** RNA 2 of WYMV. A single large polyprotein is cleaved into the two mature proteins, P1 protein and P2 protein, shown within the smaller boxes that make up the ORF

Table 2 The potential function of the proteins encoded by CWMV and WYMV

No	Viral protein name	Source of viral protein	Viral protein function	References
1	Third protein (P3)	WYMV RNA1	Viral intercellular movement	Vijayapalani et al. (2012), Chai et al. (2020), Yu et al. (2021)
2	7-kDa peptide (7 K)	WYMV RNA1	Viral-replication vesicles formation	Cui and Wang (2016), González et al. (2019), Yang et al. (2021)
3	Cylindrical inclusion protein (CI)	WYMV RNA1	Viral replication and viral intercellular movement	Wei and Wang (2008)
4	14-kDa peptide (14 K)	WYMV RNA1	Endoplasmic reticulum (ER) export and intercellular viral movement	Cui and Wang (2016), González et al. (2019), Yang et al. (2021)
5	VPg protein	WYMV RNA1	Viral RNA translation and replication	Jiang and Laliberté (2011)
6	Nuclear inclusion-a protease (NIa-Pro)	WYMV RNA1	Ploypotein cleaving and processing	Riechmann et al. (1992)
7	Nuclear inclusion "b" protein (NIb)	WYMV RNA1	Viral-replication	Zhang et al. (2019a), Shen et al. (2020)
8	Coat protein (CP)	WYMV RNA1	Viral particle assembly, viral replication and translation	Dolja et al. (1995), Li and Shirako (2015), Dai et al. (2020), Yang et al. (2021)
9	PIPO	WYMV RNA1	Viral intercellular movement	Vijayapalani et al. (2012), Chai et al. (2020), Yu et al. (2021)
10	First protein (P1)	WYMV RNA2	RNA-silencing suppressor	Tatineni et al. (2012), Valli et al. (2018)
11	Second protein (P2)	WYMV RNA2	Viral replication	Sun et al. (2014), Li et al. (2018)
12	153-kDa protein	CWMV RNA1	Viral replication and movement	Yang et al. (2017)
13	RNA-dependent RNA polymerase (RdRp)	CWMV RNA1	Viral replication	Yang et al. (2017)
14	Movement protein (MP)	CWMV RNA1	Viral intercellular movement	Andika et al. (2013b), Guo et al. (2019)
15	Coat protein (CP)	CWMV RNA2	Assembly of virus particles	Haeberlé et al. (1994), Cowan et al. (1997), Torrance et al. (2009)
16	Read-through fusion protein (CP-RT)	CWMV RNA2	Virion assembly and viral transmission	Yamamiya and Shirako (2000), Crutzen et al. (2009), Yang et al. (2016)
17	N-terminal extension coat protein (N-CP)	CWMV RNA2	Virus replication, virion assembly and systemic movement	Diao et al. (1999), Sun et al. (2013c), Yang et al. (2016), Guo et al. (2019)
18	Cysteine-rich protein (CRP)	CWMV RNA2	RNA-silencing suppressor and pathogenicity determinant	Xu et al. (2002), Te et al. (2005)

et al. 1997). Furthermore, CI can form conical structures at plasmodesmata (PD) during viral intercellular movement (Wei and Wang 2008). In a recent study, three amino acids located in the N-terminal domain of CI were found to be significantly associated with WYMV pathogenicity (Ohki et al. 2019). As in other potyviruses such as plum pox virus (PPV), both the 7 K and 14 K proteins of WYMV contain a central hydrophobic transmembrane domain. This indicated that the 7 K and 14 K proteins may also be involved in the formation of viral-replication vesicles, endoplasmic reticulum (ER) export and intercellular viral movement (Cui and Wang 2016; González et al. 2019; Yang et al. 2021). Like the NIb of other potyviruses, WYMV NIb also belongs to the viral RNA-dependent RNA polymerase (RdRp) superfamily II and contains a conserved GDD motif, which is required for RdRp activity (Zhang et al. 2019a; Shen et al. 2020; Yang et al. 2021). NIa contains two domains, an N-terminal VPg domain and a C-terminal protease. NIa has serine protease-hydrolysis activity, which primarily enables it to cleave the virual polyproteins. VPg and NIa-Pro are produced through the partial processing of NIa (Riechmann et al. 1992). Numerous studies have indicated that VPg is covalently linked to the 5′-end of the genomic RNA to mediate viral RNA translation and replication (Jiang and Laliberté 2011). As an important regulator of gene expression, VPg not only promotes viral RNA translation and accumulation but can also inhibit host RNA translation (Eskelin et al. 2011). Though WYMV VPg contains nuclear localization and exports signal domains, CP facilitates the nuclear export of VPg during WYMV infection (Sun et al. 2013d; Yang et al. 2021). P3 targets the ER membrane to form inclusions and is transported along actin filaments to participate in the formation of replication vesicles (Cui et al. 2017; Yang et al. 2021). WYMV PIPO is usually expressed with a part of the P3 ORF as P3N-PIPO, which promotes the movement of viruses from cell to cell (Vijayapalani et al. 2012; Chai et al. 2020; Yu et al. 2021).

In the proteins encoded by RNA2, the N-terminal region of WYMV P1 is highly conserved and similar to the active region of potyvirus HC-Pro. Although this has not been experimentally confirmed, it seems likely that WYMV P1 may resemble HC-Pro by functioning as an

RNA-silencing suppressor (Fig. 3 and Table 2) (Tatineni et al. 2012; Valli et al. 2018). WYMV P2 is a protein unique to bymoviruses that can recruit P1, P3, VPg, NIb, and other viral proteins through protein interactions to form the VRC (Sun et al. 2014). Moreover, WYMV P2 has been reported to be involved in the formation of WYMV genome replication-related membrane compartments (Sun et al. 2014; Li et al. 2018). It has recently been shown that P1 and P2 are also involved in the production of membranous bodies (MBs) in the viral factory of WYMV-infected host cells and constitute the main components of MBs within the ER (Xie et al. 2019).

It is known that the poly(A) and 5′-cap regions of WYMV can interact with host factors to increase the translation efficiency. Variable polyadenylation, including the absence of a poly(A) at the 3′-end of WYMV RNAs, was found in different WYMV isolates. The diversity of polyadenylation leads to significant differences in the translation level and minus-strand synthesis of WYMV (Gallie et al. 1995; Khan and Goss 2012; Geng et al. 2019). Variable polyadenylation may play a vital role in template selection for WYMV translation and replication or the molecular transition between WYMV translation and replication (Geng et al. 2019). In addition, a new internal ribosome entry site (IRES) was first discovered in the 5′-untranslated region (UTR) of WYMV RNA1, and the dynamic equilibrium state of the tertiary RNA structure was essential for promoting IRES activity in the 5′-UTR of WYMV RNA1. These findings indicated that robustness is a potential target for selection and evolutionary optimization during evolution of WYMV RNA1 (Levis and Astier-Manifacier 1993; Zhang et al. 2015b; Geng et al. 2020).

Temperature sensitivity

Temperature is an important environmental factor that affects virus infection because intracellular viral replication requires a specific temperature range and also because virus movement, virus transmission and plant defense systems are also affected by temperature. In infected wheat plants, bright yellow mosaic symptoms typically occur in early spring but disappear in new leaves during early summer. These findings suggest that low temperatures may be favorable for virus infection. For SBWMV, the optimal infection temperature was 17 °C (Ohsato et al. 2003). Substantial work has been conducted to determine the optimal infection temperatures of CWMV and WYMV. Following mechanical inoculation of the sap of virus-infected leaves onto wheat and *Nicotiana benthamiana* (*N. benthamiana*), CWMV completes its infection at 16 °C but not at 24 °C (Andika et al. 2013b). Inoculation studies of wheat and *N. benthamiana*, using a full-length complementary infectious DNA clone of CWMV that was constructed using reverse genetics showed that the optimum temperature for replication was 15–17 °C. The optimum temperature for WYMV multiplication and systemic infection was 8 °C, but the optimal temperature for viral movement and silencing is not known (Yang et al. 2016; Zhang et al. 2021a). The underlying molecular mechanisms whereby low temperatures contribute to viral infections are not well understood. A previous study revealed that low temperatures can inhibit host antiviral defenses mediated by RNA silencing, including virus-derived small interfering RNAs (vsiRNAs) (Szittya et al. 2003). Recently, numerous vsiRNAs derived from CWMV-infected wheat were identified by next-generation or deep sequencing, and these vsiRNAs exhibited a strong bias in their 5′-terminal nucleotides (Yang et al. 2014). The significance of this bias needs to be investigated and it is unclear whether these vsiRNAs are temperature-dependent, but there is no denying that this phenomenon may be the result of the evolution of the virus during the long-term competition with the host.

Impacts of virus–plant interactions

Because RNA viruses only encode a few proteins, successful viral infection depends on the complicated interaction network between viruses and hosts. Therefore, virus–host interactions serve two purposes for the virus. First, viruses need to recruit a series of host proteins to complete all steps in the complex infection cycle, which include viral particle disassembly, viral translation, formation of the VRC, virion assembly, cell-to-cell movement, and long-distance transport. To date, many host proteins have been shown to be involved in these processes (Nagy and Pogany 2011; Schoelz et al. 2011; Tilsner and Oparka 2012; Wang 2015; Yang et al. 2021). Secondly, viruses have to incite and utilize some specific cellular factors to assist them in suppressing and evading the multi-layered antiviral immune responses of plants, including hypersensitive and necrotic resistance as well as systemic acquired resistance from R gene-mediated responses. Indeed, emerging evidence has revealed that viruses have evolved various effective strategies for overcoming these antiviral pathways (Alcaide-Loridan and Jupin 2012; Mandadi and Scholthof 2013; Csorba et al. 2015; Li and Wang 2019; Yang et al. 2020b). Once a virus enters a plant cell, its needs first to shed the capsid composed of CP subunits, after which the viral genome can be exposed to the cellular translation machinery. The results of studies on tobacco mosaic virus suggest that initial virion disassembly starts when CP subunits begin to disassemble from the 5′-end of viral genomic RNA (Culver 2002). Subsequently, ribosomes bind the exposed 5′-end of the genomic RNA to translate the replicase

proteins (Wu and Shaw 1997). Some eukaryotic initiation factors (eIFs) are employed to complete this step. Recent data revealed that TaeEF1A could be upregulated upon CWMV infection and that TaeEF1A can specifically bind to stem-loop structures in CWMV RNA2 to promote CWMV replication and translation (Chen et al. 2021). Interestingly, WYMV and barley stripe mosaic virus infection can also induce TaeEF1A expression, suggesting that eEF1A may be a general host factor required for different viruses (Chen et al. 2021). In addition, the WYMV VPg protein was shown to associate with the eIF4E protein during viral replication, a phenomenon that has also been reported for many other potyviruses, such as TuMV and TEV (Kang et al. 2005; Jenner et al. 2010; Li and Shirako 2015). After virion disassembly, a conducive location in plant cells is urgently required for virus multiplication. Then, viral proteins can remodel cellular membranes together with the recruited host factors to form the VRC to promote safe replication of the viral genome. A previous transmission electron microscopy study showed that WYMV infection induced ER remodeling, and MBs with at least two different morphologies, including lamellar and tubular MBs, were observed (Xie et al. 2019). The roles of heat shock proteins (HSPs) in viral replication have been comprehensively described. For CWMV, the replicase protein was confirmed to recruit HSP70 from the cytoplasm or nucleus to the intracellular membrane system for regulating conformation or replication of VRC, and HSP70 overexpression promoted viral genomic RNA accumulation (Yang et al. 2017). To spread in plants, viruses must undergo cell-to-cell movement. Viruses typically encode movement proteins (MPs) to facilitate cell-to-cell spread from the initially infected cell(s) through PD (Maule 2008; Schoelz et al. 2011). The 37 K MP of CWMV (with two TMDs) is responsible for intracellular CWMV transport and cell-to-cell movement. The interaction of the MP with pectin methylesterase, a cell wall-associated protein, may be important for CWMV movement (Andika et al. 2013b). However, due to the difficulties in inoculating monocotyledons (particularly wheat) and the complexities of wheat genomes, research on the infection cycle of WYMV and CWMV has been restricted.

In response to viral infection, plants depend on elaborate protein interaction networks to activate antiviral defenses. However, viruses have evolved various strategies to counter these apparently ubiquitous antiviral defenses. RNA silencing is an ancient and conserved mechanism that depends on the siRNAs binding to host Argonaute (AGO) proteins and directing the RNA-induced silencing complexes (RISC) to the target transcripts to modulate gene expression (Pumplin and Voinnet 2013; Rosa et al. 2018). Viral infection leads to accumulation of vsiRNAs, which play significant roles in antiviral RNA-silencing defenses by targeting viral RNA for degradation in a sequence-specific manner (Ding and Voinnet 2007; Llave 2010; Zhang et al. 2015a). Two distinct classes of vsiRNAs have been discovered in plants, one class results from DCL-mediated dsRNA cleavage and the second class requires a RdRp (Burgyán and Havelda 2011). Research has revealed that RDR6 accumulates at high temperatures in CWMV-infected wheat root tissues, which helps to inhibit CWMV infection (Andika et al. 2013a). Previously, we characterized WYMV and CWMV vsiRNA profiles in infected wheat cells and found that the WYMV and CWMV vsiRNAs were both predominantly 21–22 nucleotides long (Yang et al. 2014; Li et al. 2018). The vsiRNAs from both viruses share features with host siRNAs, and their 5′-terminal base is biased toward A/U (Yang et al. 2014; Li et al. 2018); therefore, some of these siRNAs may be loaded into diverse AGO-containing RISCs to degrade homologous cellular transcripts involved in many biological processes (Ding and Voinnet 2007; Ruiz-Ferrer and Voinnet 2009). In CWMV-infected plants, it has also been shown that CWMV-derived vsiRNA-20 can target transcripts encoding vacuolar (H+)-PPases (*TaVP*) to inhibit cell death, thereby promoting viral infection (Yang et al. 2020a). To counter this antiviral RNA-silencing response, viruses have evolved efficient defensive proteins, known as viral suppressors of RNA silencing (VSRs) (Voinnet et al. 1999; Burgyán and Havelda 2011). The CWMV Cys-rich protein (CRP) has been identified as a VSR protein that inhibits the spread of silencing signals (Sun et al. 2013b). According to recent studies, CWMV CRP protein can be phosphorylated by SAPK7 in CWMV infection. The phosphorylated CRP interacts with RNA-binding protein UBP1-associated protein 2C (TaUBA2C), which inhibits CWMV infection through binding to the pre-mRNA of TaNPR1, TaPR1, and TaRBOHD to induce cell death and H_2O_2 production, thereby changing TaUBA2C chromatin-bound status and attenuating the RNA- or DNA- binding activities (Li et al. 2022). However, no VSR protein has been reported for WYMV. Interestingly, *DCL4* transcripts were downregulated in WYMV-infected root tissues (Yang et al. 2014), which suggested that WYMV may regulate *DCL4* expression to affect vsiRNA biogenesis, thereby promoting viral infection. We recently found that CWMV infection upregulated the expression of the long-noncoding RNA (lncRNA) XLOC_006393, a precursor of miR168c, thereby promoting viral infection (Zheng et al. 2021). miR168 is one of the most abundant and highly conserved miRNAs in plants, and it can directly regulate AGO1 expression (Gursinsky et al. 2015). These findings suggest that CWMV may regulate the expression of host

lncRNAs to inhibit antiviral RNA silencing. In addition, N^6-methyladenosine (m^6A)-nucleotide modification, another regulatory mechanism occurring at the mRNA level, has been found to be extensively involved in virus–host interactions (Li et al. 2018; Zhang et al. 2021c). Increasing evidence has revealed that viral infection can affect the overall level of m^6A modification in host cells, leading to a range of changes in transcripts related to various biological processes (Lichinchi et al. 2016; Zhang et al. 2021c). Research from our laboratory indicated that WYMV infection in susceptible and resistant wheat varieties resulted in different transcriptome-wide m^6A profiles. Several transcripts enriched in plant–pathogen interaction pathways were modified by m^6A and exhibited differential expression patterns between virus-infected plants of the two varieties (Zhang et al. 2021d). These results suggest that WYMV can disrupt the expression of immunity-related genes through m^6A methylation to inhibit host antiviral responses. In addition, another epigenetic modification (acetylation) was associated with CWMV infection: the acetylation levels of chloroplast proteins, histone 3, and some metabolic pathway-related proteins were significantly higher in CWMV-infected plants than in uninfected plants (Gao et al. 2021; Yuan et al. 2021). Several other biological processes are also involved in WYMV or CWMV infection, including ubiquitination and the regulation pathways by plant hormones. Several ubiquitin-specific protease (UBP) family genes were differentially expressed after WYMV or CWMV infection, and silencing TaUBP1A.1 in wheat plants promoted CWMV infection (Xu et al. 2021). These results suggest that ubiquitination may act as a host antiviral defense that inhibits CWMV infection. In addition, CWMV infection was found to suppress the abscisic acid (ABA) pathway in *N. benthamiana*, and ABA production was downregulated in CWMV-infected plants when compared to that in mock-infected plants (He et al. 2021). Another report revealed that WYMV NIb interacts with the wheat light-induced protein TaLIP to facilitate viral infection by interfering with the ABA signaling pathway (Zhang et al. 2019a). The results of both these studies indicated that both WYMV and CWMV can disturb plant hormone pathways to promote viral infection. However, a knowledge gap remains regarding the host factors involved in WYMV and CWMV infection. This topic requires urgent attention from researchers.

Microbial mechanisms for controlling the onset of wheat soil-borne virus diseases

A better establishment of microbes and their interactions in the plant–soil system to prevent soil-borne diseases and enhance plant disease resistance can be achieved through multiple mechanisms, such as stimulating the production of plant growth hormones (Liu et al. 2021), competition with pathogens for nutrients (Gu et al. 2020; Tao et al. 2020), production of certain compounds (e.g., antibiotics) that are inhibitory against pathogens (Syed-Ab-Rahman et al. 2019), and induction of systemic resistance in plants (Pieterse et al. 2014). Our recent studies have demonstrated that when wheat plants had increasing levels of WYMV infection, alternations were found in the microbial communities across the soil–plant continuum (bulk soil, rhizosphere soil, roots, and leaves), showing a significant enrichment in the plant-beneficial bacteria of specific genera, like *Streptomyces, Stenotrophomonas, Bradyrhizobium, Sphingomonas*and and *Bacillus* (which are notable antibiotic producers) as well as growth-promoting microbes. The scale, connectivity, and complexity of the rhizosphere and root endosphere co-occurrence networks was also increased under these disease conditions (Wu et al. 2021a). Moreover, relatively high microbial diversity and stable community structures were detected in the soil of WYMV-tolerant cultivars compared to WYMV-sensitive ones. WYMV-tolerant cultivars greatly recruited many known beneficial bacterial and fungal taxa into their rhizosphere soil, including *Xanthomonadales*, *Actinomycetales*, *Sphingomonas*, *Rhizobium*, *Bacillaceae*, *Bacillus*, *Streptomycetaceae*, *Streptomyces*, *Nocardioides*, *Pseudonocardia*, *Bradyrhizobium*, *Pseudonocardiaceae*, and *Solibacteraceae*. In contrast, there were more potential pathogens (*Fusarium*) associated with WYMV-sensitive cultivars than with WYMV-tolerant ones. WYMV-tolerant cultivars had much more complex belowground microbial networks, with larger numbers of mutually beneficial and keystone bacterial taxa, and such microbial association networks may have been responsible for maintaining the stability and ecological balance of the microbial communities (Wu et al. 2021b). Data indicates that wheat plants, particularly disease-tolerant cultivars, may be capable of recruiting beneficial microbial microorganism and preventing the collapse of belowground microbial networks after infection with a disease. To evaluate whether the structure of microbial communities associated with wheat soil-borne virus diseases can be directly shaped, the underlying mechanisms controlling the assembly of microbial communities as well as the biotic and abiotic drivers under different WYMV levels have been explored. With increasing levels of WYMV infection, the deterministic processes were greatly enhanced during the assembly of bacterial communities, the contribution of deterministic processes to the assembly of bacterial communities increased, and the habitat niche breadth of the bacterial communities decreased. Intensified competition between bacteria and fungi and increased soil total-nitrogen and soil-organic carbon contents under the

diseased conditions were primarily responsible for this phenomenon (Zhang et al. 2021b). These findings provide an important insight into the associations between the onset of wheat soil-borne virus diseases and alternations in the microbial communities and warrant future research on the application of specific microbial taxa to inhibit wheat soil-borne virus diseases. Although progress has been made in understanding the relationships between microbial communities and wheat soil-borne virus diseases, utilizing microbiomes to suppress diseases is still in the initial stages. Further studies guided by community-based "omics" approaches will require the translation of functional potential to functional activity. The integration of sequencing technology, microfluidics, synthetic community analysis and modeling, robotics and machine learning, and functional joint analysis should provide novel ways to capitalize on microbiomes and increase disease resistance in soil and plants (Toju et al. 2018; Du et al. 2021). The potentially beneficial microbes recruited by wheat plants can be isolated and cultured to assess the functions that influence biocontrol efficacy, including rhizosphere competence, niche and/or substrate competition, and induced systemic resistance (Dignam et al. 2016). Synthetic microbial communities (SynComs) containing these potential beneficial microbes can be designed, and reductionist experiments can be performed to investigate whether the SynCom added to sterile plants has the expected biological functions (Bai et al. 2022). Simultaneously, we can deliberately shape a microbiome community through nutritional restriction or genetic modification and then apply it to control viral diseases in the field. Therefore, more knowledge about the nutritional needs of various microbiomes, including pathogens and their correlation networks is essential to design and produce such plant-beneficial microbiomes or adopt new tailored disease-prevention strategies (Du et al. 2021).

Disease management

As plant viruses are obligate intracellular parasites, prophylactic measures play a crucial role in restricting virus dispersion to mitigate economic losses caused by viruses (Nicaise 2014). Integrated pest management (IPM) is a general eco-friendly management strategy for disease control. The specific measures of IPM mainly include the use of resistant varieties, crop rotation, control of vectors, elimination of reservoirs etc. (Jones and Naidu 2019). Prevention of viruses spread is also an important step for virus disease management. During the plant growing season, the vector of soil borne viruses should be closely monitored and managed. However, it will be difficult to eliminate the vector of wheat soil-borne viruses from the soil by chemical control or conventional crop management. Since the dormant spores of the vector can be retained in soil for many years, it is unlikely that crop rotation and fallowing will control the viral disease. Care needs to be taken not to transport soil containing WYMV and CWMV between fields, regions or countries. Accordingly, the most effective approach to control wheat soil-borne viruses is the use of resistant cultivars. Indeed, a large number of resistant varieties have been developed through traditional breeding methods. To obtain more resistant cultivars, modern biotechnology methods must be used to deploy the increasing numbers of resistance genes and/or QTLs that are being identified on wheat chromosomes (as described above). For example, two major antiviral strategies, RNA silencing and genome editing, have been successfully used in antiviral breeding (Zhao et al. 2020). Previous studies showed that transgenic wheat plants expressing an antisense *NIb8* gene (the *NIb* encoding the replicase of WYMV) had high and broad-spectrum resistance to WYMV isolates from different sites in China. The grain yield of transgenic wheat was approximately 10% greater than that of the susceptible wild-type control in field nurseries (Chen et al. 2014). In addition, transgenic expression of constructs capable of driving dsRNA expression resulted in a higher yield of high-level virus-resistant plants than did expressing constructs that produced either sense or antisense RNA alone (Chen et al. 2014). In a recent study, vsiRNA1 derived from the WYMV *NIb* gene was shown to target the wheat gene *TaAAED1* to degrade its transcript leading to inhibited ROS scavenging. Transgenic wheat lines expressing vsiRNA1 had broad-spectrum disease resistance to viruses and non-viral pathogens with significantly improved agronomic traits in the endemic presence of pathogens (Liu et al. 2021). These results indicated that "novel RNA-based agricultural technologies" can potentially protect crops against viral diseases (Leonetti and Pantaleo 2021).

The CRISPR/Cas (clustered regularly interspaced short palindromic repeats/ CRISPR-associated) system, a genome editing technology, has been applied for the genetic improvement of plant viruses resistance and has accelerated resistance breeding (Zhang et al. 2019b). In cucumbers, knocking out the *eIF4E* gene using the CRISPR/Cas9 system resulted in broad-spectrum resistance to members of the *Potyviridae* family, including cucumber vein yellowing virus, papaya ringspot virus, and zucchini yellow mosaic virus (Chandrasekaran et al. 2016). In wheat, *TaeIF(iso)4e*-mutant lines were obtained using CRISPR-Cas9 genome-editing technology, which revealed that the corresponding mutations can potentially confer WSSMV and WYMV resistance (Hahn et al. 2021). Recently, the wheat orthologs of the barley susceptibility factor disulfide

isomerase like 5–1 (HvPDIL5-1) were edited in the wheat genome, conferring WYMV resistance in wheat (Kan et al. 2022). Several CRISPR/Cas systems from some bacterial strains (e.g., *Leptotrichia wadei* and *Francisella novicida*) have been reported to target the genome of RNA viruses, which suggests new strategies against RNA viruses (Sampson et al. 2013; Abudayyeh et al. 2017). For example, the Cas13a system from *Leptotrichia shahii* (LshCas13a) was engineered to degrade the genomic RNA of southern rice black-streaked dwarf virus and rice stripe mosaic virus in rice plants (Zhang et al. 2019b). However, there are no reports of wheat varieties generated using CRISPR/Cas9 systems to target the genomes of WYMV or CWMV. Owing to their simplicity, high efficiency, and affordability, CRISPR/Cas systems offer great prospects for restricting damage to wheat production caused by soil-borne viruses.

Conclusions and future prospects

In recent decades, significant progress has been made to reveal the mechanisms underlying the interaction mechanisms between wheat and soil-borne viruses in an effort to develop countermeasures against WYMV and CWMV. We now know that the proteins encoded by CWMV and WYMV recruit a large number of host factors to establish infection and that wheat plants have a multilayered antiviral surveillance and defense system for limiting viral infection. In turn, CWMV and WYMV have multiple counteracting strategies to suppress the antiviral response. It is critical to explore the potential functions of the genes involved in wheat-virus interactions to achieve durable and high levels of resistance. With the improvement of gene cloning and genetic techniques, some novel functional genes/QTLs have been identified, and these genes/QTLs can be incorporated into new wheat cultivars. These advances will provide precious information for forecasting and controlling WYMV and CWMV effectively in the future as well as offer guidance for preventing and controlling other viral diseases in wheat. Based on the information reviewed above, we propose tentatively the following topics for further research: i) molecular identification of host factors that are critical for viral infection but dispensable for plant growth and development, especially the recessive resistance mediated factors for candidates of CRISPR-Cas gene editing; ii) mechanisms by which the virus–host arms race has evolved and iii) molecular mechanisms underlying the host or non-host resistance against wheat soil-borne viruses; with the aim to provide a theoretical basis for antiviral molecular breeding.

Abbreviations

ABA: Abscisic Acid; BYDV: Barley yellow dwarf viruses; CI: Cylindrical inclusion protein; CP: Coat protein; CRISPR/Cas: Clustered regularly interspaced short palindromic repeats/ CRISPR-associated; CRP: Cys-rich protein; CWMV: Chinese wheat mosaic virus; EIFs: Eukaryotic initiation factors; ER: Endoplasmic reticulum; HSPs: Heat shock proteins; IPM: Integrated pest management; IRES: Internal ribosome entry site; LncRNA: Long-non coding RNA; m^6A: N^6-methyladenosine; MP: Movement protein; MBs: Membranous bodies; *N. benthamiana*: *Nicotiana benthamiana*; NIa-Pro: Nuclear inclusion-a protease; Nib: Nuclear inclusion "b" protein; ORF: Open reading frame; P3: Third protein; PD: Plasmodesmata; *P. graminis*: *Polymyxa graminis*; PMTV: Potato mop-top virus; PPV: Plum pox virus; QTL: Quantitative trait loci; RdRp: RNA-dependent RNA polymerase; RISC: RNA-induced silencing complexes; SBCMV: Soil-borne cereal mosaic virus; SBWMV: Soil-borne wheat mosaic virus; SSR: Simple-sequence repeat; SynComs: Synthetic microbial communities; TEV: Tobacco etch potyvirus; TMDs: Transmembrane domains; TuMV: Turnip mosaic virus; UBP: Ubiquitin-specific protease; UTR: Untranslated region; VPg: Viral protein genome-linked; VRC: Viral replication complex; VsiRNAs: Virus-derived small interfering RNAs; VSRs: Viral suppressors of RNA silencing; WDV: Wheat dwarf virus; WYMV: Wheat yellow mosaic virus; WSMV: Wheat streak mosaic virus; WSSMV: Wheat spindle streak mosaic virus; 7 K: 7-kDa peptide; 14 K: 14-kDa peptide.

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