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Review

Wheat streak mosaic virus: a century old virus with rising importance worldwide

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SUMMARY

Wheat streak mosaic virus (WSMV) causes wheat streak mosaic, a disease of cereals and grasses that threatens wheat production worldwide. It is a monopartite, positive-sense, single-stranded RNA virus and the type member of the genus *Tritimovirus* in the family *Potyviridae*. The only known vector is the wheat curl mite (WCM, *Aceria tosichella*), recently identified as a species complex of biotypes differing in virus transmission. Low rates of seed transmission have been reported. Infected plants are stunted and have a yellow mosaic of parallel discontinuous streaks on the leaves. In the autumn, WCMs move from WSMV-infected volunteer wheat and other grass hosts to newly emerged wheat and transmit the virus which survives the winter within the plant, and the mites survive as eggs, larvae, nymphs or adults in the crown and leaf sheaths. In the spring/summer, the mites move from the maturing wheat crop to volunteer wheat and other grass hosts and transmit WSMV, and onto newly emerged wheat in the fall to which they transmit the virus, completing the disease cycle. WSMV detection is by enzyme-linked immunosorbent assay (ELISA), reverse transcription-polymerase chain reaction (RT-PCR) or quantitative RT-PCR (RT-qPCR). Three types of WSMV are recognized: A (Mexico), B (Europe, Russia, Asia) and D (USA, Argentina, Brazil, Australia, Turkey, Canada). Resistance genes *Wsm1, Wsm2* and *Wsm3* have been identified. The most effective, *Wsm2*, has been introduced into several wheat cultivars. Mitigation of losses caused by WSMV will require enhanced knowledge of the biology of WCM biotypes and WSMV, new or improved virus detection techniques, the development of resistance through traditional and molecular breeding, and the adaptation of cultural management tactics to account for climate change.

Keywords: *Aceria tosichella*, cereal crops, *Tritimovirus*, wheat curl mite, WSMV.

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INTRODUCTION

Wheat streak mosaic virus (WSMV) was first observed by Peltier in Nebraska in the Central Great Plains of the USA in 1922 and described as '*yellow mosaic*' (McKinney, 1937; Staples and Allington, 1956). WSMV was formerly placed in the genus *Rymovirus* together with mite-transmitted viruses of the family *Potyviridae*. Later, the complete genome sequence and evolutionary analysis established WSMV with the whitefly-transmitted *Sweet potato mild mottle virus* and not with *Ryegrass mosaic virus*, the type member of the genus *Rymovirus* (Stenger *et al.*, 1998). The finding thus proposed a new genus known as '*Tritimovirus*' within the family *Potyviridae*, for which WSMV is the type member (Rabenstein *et al.*, 2002). It is transmitted by the wheat curl mite (WCM, *Aceria tosichella* Keifer). The virus is widely distributed in most wheat-growing regions of the world, including the USA, Canada, Mexico, Brazil, Argentina, Europe, Turkey, Iran, Australia and New Zealand (Hadi *et al.*, 2011; Navia *et al.*, 2013).

WSMV is hosted by many plant species of the family *Poaceae*, including wheat (*Triticum aestivum* L.), oat (*Avena sativa* L.), barley (*Hordeum vulgare* L.), maize (*Zea mays* L.), millet (*Panicum*), *Setaria* and *Echinochloa* spp., and several other grasses (Chalupníková *et al.*, 2017; Dráb *et al.*, 2014; French and Stenger, 2002). Given the potentially devastating impact of WSMV on affected cereal crops, the occurrence of this disease in wheat has been a cause for concern because losses can range from minimal to complete crop failure (French and Stenger, 2003). Improvement in WSMV resistance is an important aspect of wheat production, and the development of resistant cultivars has helped to increase production (Price *et al.*, 2010a).

Because of the devastating economic impact caused by WSMV in wheat-growing countries around the globe, and its significance in the plant pathology community, a comprehensive report updating the knowledge on WSMV is warranted. Therefore, this review examines current knowledge on WSMV including virus biology, genome architecture, mechanism of transmission, host range, disease symptoms and cycle, diagnostic tools, genetic diversity, host resistance, and management strategies and tactics.

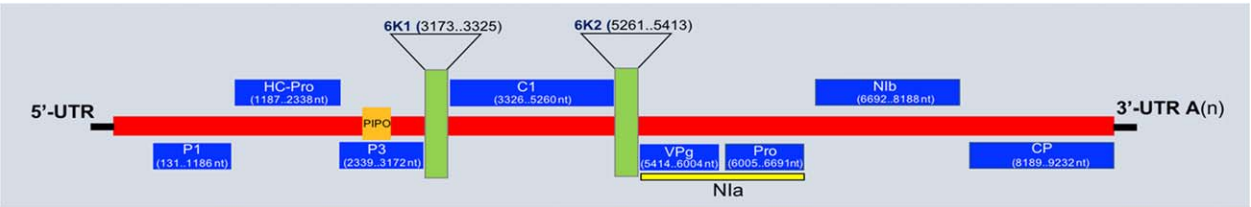

Fig. 1 Genome architecture of Wheat streak mosaic virus (WSMV). The genome size of WSMV is 9.3–9.4 kb and has a single open reading frame, which is transcribed into a large polyprotein. This polyprotein is composed of 10 proteins: P1 (P1 protein: 40 kDa); HC-Pro (helper component protease: 44 kDa); P3 (P3 protein: 32 kDa); 6K1 and 6K2 (6 kDa protein); CI (cytoplasmic inclusion protein: 73 kDa); VPg (viral protein genome-linked proteinase: 23 kDa); NIa (nuclear Inclusion putative protease: 26 kDa); NIb (Nuclear Inclusion putative polymerase: 57 kDa) CP (coat protein: 37 kDa). nt, nucleotides; UTR, untranslated region.

CAUSAL AGENT OF THE DISEASE

WSMV is the causal agent of wheat streak mosaic disease. WSMV is a non-enveloped, flexible, filamentous, rod-shaped virus composed of a monopartite, positive-sense, single-stranded RNA genome (ssRNA+). The WSMV genome size is ~9.3–9.4 kb and expands into a single open reading frame (ORF), which is transcribed into a large polyprotein (Fig. 1). This polyprotein is cleaved into at least 10 mature proteins: P1 (P1 protein: 40 kDa); HC-Pro (helper component protease: 44 kDa); P3 (P3 protein: 32 kDa); 6K1 and 6K2 (6 kDa protein); CI (cytoplasmic inclusion protein: 73 kDa); VPg (viral protein genome-linked proteinase: 23 kDa); NIa (nuclear Inclusion putative protease: 26 kDa); NIb (Nuclear Inclusion putative polymerase: 57 kDa) CP (coat protein: 37 kDa) (Choi *et al.*, 2002; Chung *et al.*, 2008; Stenger *et al.*, 1998). The recently described short ORF (PIPO) is expressed as a fusion protein with the N-terminal half of P3 (P3N-PIPO) (Chung *et al.*, 2008). The role of these distinct proteins has been deciphered in various processes, including suppressor of RNA silencing (Young *et al.*, 2012), genome amplification, protein–protein interactions, RNA binding and amplification of the virus genome, cell-to-cell and systemic transport, virion assembly (Rojas *et al.*, 1997; Tatineni and French, 2014; Tatineni *et al.*, 2017) and proteolytic processing (Schaad *et al.*, 1996). The 5′-terminus has a VPg and the 3′-terminus has a poly (A) tail. The RNA is infectious and serves as both the genome and viral messenger.

Protein functions

P1

The P1 protein of WSMV has serine proteinase activity, is known to mediate suppression of RNA silencing and plays a role in the enhancement of disease symptoms in WSMV-P1-expressing transgenic plants infected with *Potato virus X* (PVX) (Young *et al.*, 2012).

HC-Pro

Mutations of HC-Pro in potyviruses affect multiple functions, including disruption of polyprotein processing, aphid transmission, long-distance movement, maintenance of replication and suppression of post-transcriptional gene silencing (PTGS) (Carrington *et al.*, 1996; Llave *et al.*, 2002; Ruiz-Ferrer *et al.*, 2005). However, WSMV HC-Pro shares two functions in common with potyviruses: mediation of vector transmission and cysteine proteinase activity (Stenger *et al.*, 2005a; Young *et al.*, 2007). Moreover, deletion of the HC-Pro coding region shows no effect on WSMV virulence in wheat, oats and corn (Stenger *et al.*, 2005b). Mutation analyses of the WSMV HC-Pro protein suggest that it plays a role in replication and is dispensable for systemic movement (Stenger *et al.*, 2006). WSMV HC-Pro does not mediate suppression of RNA silencing when tested in *Nicotiana benthamiana* (Young *et al.*, 2012). WSMV HC-Pro exhibits no effect on disease synergism in maize co-infected with a WSMV HC-Pro complete deletion mutant and *Maize chlorotic mottle virus* (MCMV) (Stenger *et al.*, 2007).

CP

WSMV CP is 349 amino acids long. The C-terminal aspartic acid residues (D216, D289, D290, D326, D333 and D334) of CP are involved in host-specific virus movement and play a role in efficient cell-to-cell movement in wheat and long-distance transport in maize (Tatineni and French, 2014). WSMV CP contains three flexible linker motifs: SGSGS-1 (36–40 amino acids), SGSGS-2 (43–47 amino acids) and SGSGS-3 (53–57 amino acids). Deletion of these motifs, either individually or jointly, elicits symptoms similar to the wild-type (Tatineni *et al.*, 2017). The CP amino acids 6–27 and 85–100 are required for efficient virion assembly and/or systemic infection and cell-to-cell movement (Tatineni and French, 2014). Deletions in the N-terminal region (58–84 amino acids) of the CP enhance the accumulation of CP and genomic RNA, alter CP-specific protein profiles and cause severe symptom phenotypes in multiple cereal hosts, including wheat, maize, rye and barley (Tatineni *et al.*, 2017). The N-terminal region of WSMV CP is a host- and strain-specific long-distance transport factor (LTF) in maize. The differing amino acids (AS to EP at position 20/21; Q to L at position 30; AG to VE at position 50/52) in the N-terminus of CP between the WSMV-S81 and WSMV-T isolates are crucial for interactions with the maize inbred line SDp2 (Tatineni *et al.*, 2011). Recently, it has been reported that amino mutations of aspartic acid residues at amino acid positions 289 or 326 (D289A or D326A) at the carboxy-proximal region of CP significantly reduce mite transmission (Tatineni *et al.*, 2018).

Fig. 2 Scanning electron microscopy (SEM) image of wheat curl mite (*Aceria tosichella*) specimens on a wheat leaf.

VIRUS TRANSMISSION BY WCM

The only known vector of WSMV is an obligatory phytophagous WCM, which is amongst the most important eriophyid mite pests of agricultural crops (Navia *et al.*, 2013; Oldfield and Proeseler, 1996). This microscopic mite (Fig. 2) inhabits sheltered sites on the plant which protect it from desiccation (Navia *et al.*, 2013), and the haplodiploid single unfertilized female is capable of initiating a population (Miller *et al.*, 2012), which increases its ability to successfully spread the viruses it transmits. In addition to wheat, WCMs can transmit WSMV to barley, oats, corn, rye and many wild annual kinds of grass (Table 1). As a result of its wind-borne dispersal, the mite is widely distributed in cereal fields and grasslands, which boosts the ability of WSMV to spread within cereal-producing regions worldwide. The capability of WCMs to successfully colonize new plants is remarkable. After landing on new plants, WCMs are able to multiply very rapidly and attain, after two generations (14 days), a population density 25% higher than that on the plant from which they dispersed (Kiedrowicz *et al.*, 2017a). This confirms the great dispersal and colonization potential of WCMs, which influences the spread of wheat streak mosaic.

The WCM was identified as the agent transmitting WSMV by Slykhuis (1955), and the recent use of DNA sequence data and experimental host bioassays has shown that the WCM is, in fact, a species complex consisting of several divergent genetic lineages (probably cryptic species) (Miller *et al.*, 2013; Skoracka *et al.*, 2012, 2013). Some lineages are highly host specific to single wild-growing grass species, whereas others are less host specialized and feed on several plant species, including cereals (Skoracka *et al.*, 2013, 2017). The genetic and host range variability within the WCM complex corresponds to the virus vectoring ability amongst WCM lineages (Hein *et al.*, 2012; Schiffer *et al.*, 2009). Up to now, it has been shown that only two lineages within the complex can transmit plant viruses in wheat (Hein *et al.*, 2012).

These lineages have been designated as type 1 and type 2 in Australia (Carew *et al.*, 2009); they match the genotypes found in North America (Hein *et al.*, 2012), and correspond to European and South American MT-8 and MT-1 lineages designated by Skoracka *et al.* (2013, 2014). Laboratory-based transmission trials using these two types collected in Australia have indicated that only type 2 (MT-1) is able to transmit WSMV (Schiffer *et al.*, 2009). However, both genotypes collected in North America have been found to effectively transmit WSMV, although at varying rates (Seifers *et al.*, 2002). WCM type 2 (MT-1) transmits WSMV at an average rate of 43%–68%, depending on the vector's phenological stage, and also reproduces more rapidly in the presence of WSMV relative to type 1 (MT-8) (Siriwetwiwat, 2006). This result may suggest that a specific symbiotic relationship between WCM type 2 (MT-1) and WSMV exists, which enables higher success for both the mite and virus, e.g. better reproductive rates for the mites and therefore better chance of virus dispersal. Some arthropod-borne plant viruses exhibit close relationships with their vector, and vector fitness is often higher on infected host plants (e.g. Belliure *et al.*, 2005).

In Poland, these two WCM biotypes also differ in colonization strategy, and biotype 1 (MT-8) has a uniform distribution, whereas biotype 2 (MT-1) occurs unexpectedly in only a few localities within the country, but attains very high densities there (about 30% higher than MT-8) (Skoracka *et al.*, 2017). All results obtained to date now suggest that biotype 2 (MT-1) is able to multiply more rapidly and transmit WSMV more efficiently than biotype 1 (MT-8). These differences in virus transmission efficiency therefore indicate that these two biotypes may require different control and management strategies. As they are divergent phenotypes, they may respond differently to control measures. The next steps directed towards WCM management should focus on genotyping methods to enable straightforward and rapid identification of the biotype in the field.

Virus transmission rates may be determined not only by mite genotype, but also by virus genetic strain. It has been shown that virus isolate and mite genotype, but not source location or WCM colony age, have a significant influence on WSMV transmission, and the existence of cryptic species within WCMs and numerous genotypes of WSMV complicates the epidemiology and poses a challenge to the management of this virus (Wosula *et al.*, 2016).

Undoubtedly, the existence of divergent WCM lineages has implications not only for the management of the WCM and WSMV, but also for the study of the biology and genetics of virus transmission. All further research on the relationships between the WCM and WSMV should be based on the molecular identification of WCM lineages and should focus on particular lineages instead of WCM *sensu lato*.

Table 1 Host range of *Wheat streak mosaic virus*.

	Host	Common name	Reference
Cereals			
	Avena barbata	Bearded oat	Coutts *et al.* (2014)
	Avena sativa	Oat	Brakke (1971)
	Hordeum vulgare	Barley	Brakke (1971)
	Panicum millaceum	Broomcorn millet	Sill and Agusiobo (1955); Vacke *et al.* (1986); Ellis *et al.* (2004)
	Pennisetum glaucum	Pearl millet	Seifers *et al.* (1996)
	Secale cereale	Cereal rye	Vacke *et al.* (1986); Ito *et al.* (2012)
	Setaria italica	Foxtail millet	Truol *et al.* (2010)
	Sorghum bicolor	Sorgum	Seifers *et al.* (1996)
	Triticum aestivum	Wheat	Brakke (1971)
	Zea mays	Maize	Brakke (1971)
Wild grasses			
	Aegilops cylindrica	Jointed goatgrass	Sill and Connin (1953)
	Agropyron repens	Couch grass	Dráb *et al.* (2014); Singh and Kundu (2017)
	Agrostis capillaris	Common bent	Chalupníková *et al.* (2017)
	Alopecurus pratensis	Meadow foxtail	Dráb *et al.* (2014)
	Anthoxanthum odoratum	Sweet vernal-grass	Chalupníková *et al.* (2017)
	Arrhenatherum elatius	False oat-grass	Dráb *et al.* (2014)
	Austrostipa compressa	Speargrass	Vincent *et al.* (2014)
	Avena fatua	Wild oat	Vacke *et al.* (1986)
	Avena strigesa	Wild oats	Vacke *et al.* (1986)
	Brachypodium distachyon	Purple false brome	Mandadi *et al.*, (2014)
	Briza maxima	Blowfly grass	Coutts *et al.* (2014)
	Bromus arvenis	Field brome	Sill and Connin (1953)
	Bromus japonicus	Japanese brome	Wegulo *et al.* (2008)
	Bromus rigidus	Brome grass	Coutts *et al.* (2014)
	Bromus secalinus	Cheat grass	Sill and Connin (1953)
	Bromus tectorum	Downy brome	Sill and Connin (1953)
	Cenchrus longispinus	Mat sandbur	Connin (1956)
	Cenchrus pauciflours	Sandbur	Wegulo *et al.* (2008)
	Cynodon dactylon	Couch grass	Ellis *et al.* (2004)
	Digitaria sanguinalis	Hairy crab grass	Vacke *et al.* (1986)
	Echinochloa crus-galli	Barnyardgrass	Sill and Connin (1953)
	Echinochloa colonum	Junglerice	Khadivar and Nasrolahnejad (2009)
	Elymus repens	Quackgrass	Ito *et al.* (2012)
	Eragrostis cilianensis	Stink grass	Connin (1956)
	Eragrostis curvula	African lovegrass	Ellis *et al.* (2004)
	Eriochloa acuminata	Tapertip cupgrass	Seifers *et al.* (2010)
	Eriochloa contracta	Prairie cupgrass	Christian and Willis (1993)
	Eleusine tristachya	Spike goosegrass	Ellis *et al.* (2004)
	Elymus canadensis	Canada wild rye	Ito *et al.* (2012)
	Holcus lanatus	Soft-grass	Chalupníková *et al.* (2017)
	Holcus mollis	Creeping soft-grass	Chalupníková *et al.* (2017)
	Hordeum leporinum	Barley grass	Coutts *et al.* (2014)
	Lagurus ovatus	Hare's-tail	Vacke *et al.* (1986)
	Lolium mitiflorum	Annual ryegrass	Vacke *et al.* (1986); Ellis *et al.* (2004)
	Lolium rigidum	Ryegrass	Coutts *et al.* (2014)
	Panicum dichotomiflorum	Fall panicgrass	Sill and Connin (1953)
	Panicum capillare	Witch grass	Coutts *et al.* (2008a,b)
	Phalaris aquatica	Phalaris	Ellis *et al.* (2004)
	Phleum pratense	Timothy-grass	Dráb *et al.* (2014)
	Poa pratensis	Bluegrass	Ito *et al.* (2012); Dráb *et al.* (2014)
	Setaria viridis	Green bristlegrass	Sill and Connin (1953)
	Tragus australianus	Small burr grass	Coutts *et al.* (2008a,b)

However, to date, the fundamental knowledge about relationships between the WCM and WSMV has been based solely on WCM *sensu lato*. The WCM acquires WSMV during feeding, when it penetrates the epidermal cells using thin, dagger-like chelicerae. The mites are subsequently infective for up to 9 days at 20–25 °C after they have been removed from an infected plant or after

moulting to the next developmental stage (Navia *et al.*, 2013; Orlob, 1966). Mites can remain infective for up to 2 months at 3 °C, which indicates that overwintering specimens can be a source of WSMV inoculum (Navia *et al.*, 2013). All mobile stages of the WCM (larva, nymph and adult) can be infective. However, virus transmission efficiency differs amongst stages, with immature stages having a higher efficiency than adults. Moreover, for adults to be effectively infective, they must acquire the virus as an immature stage (del Rosario and Sill, 1965; Orlob, 1966; Siriwetwiwat, 2006; Slykhuis, 1955) and, to acquire the virus, the mite requires 15–30 min of feeding on the plant (Orlob, 1966). It has been suggested that WSMV circulates, but does not multiply, in its vector (Paliwal, 1980).

WCMs can transmit other viruses apart from WSMV, such as *Triticum mosaic virus* (TriMV) and *Wheat mosaic virus* (WMoV), and can cause mixed infections (Byamukama *et al.*, 2014; Seifers *et al.*, 2011; de Wolf and Seifers, 2008). Such double or even triple infections have been found more frequently (47%) than single infections of winter wheat by WSMV (5%) in the Central Great Plains of the USA (Byamukama *et al.*, 2016). In another experiment, yield loss was 96% when a susceptible wheat cultivar was co-inoculated with WSMV and TriMV, compared with single inoculation (yield losses of 53% and 50% caused by single inoculation of wheat by TriMV and WSMV, respectively) (Byamukama *et al.*, 2014). Oliveira-Hofman *et al.* (2015) found that transmission of WSMV by WCM genotype 2 (MT-1) was higher from singly infected source plants than from those co-infected with TriMV.

The high level of infection of a wheat crop with WSMV is associated with the presence of abundant grasses and volunteer wheat plants which serve as hosts for WCMs and WSMV and provide an effective 'green bridge' refuge for WCMs between harvesting of the current season's crop and planting of the next season's crop (Somsen and Sill, 1970). When the quality of green bridge food decreases because of host maturity or overcrowding, WCMs start their aerial movement by wind currents into wheat fields from nearby grass vegetation or fields with volunteer wheat that harbour viruliferous mites (Kiedrowicz *et al.*, 2017b; Somsen and Sill, 1970). For example, in Australia, a 40% WSMV incidence and about 5000 WCMs per spike were found at the margin of a wheat crop associated with abundant grasses and volunteer wheat plants in an adjacent pasture (so-called 'edge effect') (Coutts *et al.*, 2008a,b). However, Byamukama *et al.* (2016) have shown that viruliferous WCMs can be found in any part of the field by the end of the growing season, not only at the edges of wheat fields. Hunger *et al.* (1992) and Somsen and Sill (1970) found that, as plants mature, they become more resistant to virus infection and develop fewer and milder symptoms. WCMs usually attain high population densities at the end of the wheat growing season, which ensures the infestation and subsequent virus infection of various green bridge hosts, including volunteer wheat and grasses. If conditions allow the survival of these hosts until autumn-planted winter wheat emerges, the probability of WSMV transmission to autumn-planted wheat increases, resulting in some level of disease and yield loss every year (Byamukama *et al.*, 2016). The control of grasses and volunteer cereals before the planting of winter wheat and the use of resistant cultivars have been suggested as effective strategies for WCM and WSMV management (Coutts *et al.*, 2008a,b).

Apart from green bridges, climate and weather conditions may influence the levels of WCM infestation and WSMV infection. It has been suggested that high temperatures are the most preferable for MT-1 and MT-8 WCM lineages (Kuczyński *et al.*, 2016). According to Orlob (1966), dry and hot conditions favour the development of WCM populations. Indeed, in Nebraska, USA, the drier western regions are more conducive for WCM population build up than are the less dry eastern regions. In addition, in a year with dry and warm conditions, considerably more WCMs were trapped during a field experiment (Byamukama *et al.*, 2016). Conversely, in Australia, wet summers and autumns, as well as westerly frontal winds, provide good conditions for WCM development and spread, which increases the probability of virus outbreaks (Coutts *et al.*, 2008a,b).

Virus–vector interactions may also be altered by nutrient availability. It has been shown that enrichment of CO_2 concentration has no observable effects on WCM populations, which suggests that increases in atmospheric CO_2 may not directly alter WCM populations and WSMV spread (Miller *et al.*, 2015). Interestingly, nitrogen fertilization increased WCM population growth rates when mites were WSMV infested, but had the opposite effect on non-viruliferous mites. This outcome was interpreted as a virus–vector mutualism that is conditional on nitrogen limitation. Although, at high nitrogen rates, the interaction between virus and vector was mutually beneficial, at low nitrogen rates the transmission was beneficial for the virus, but detrimental for the vector (as the vector is expected to be nitrogen limited). Therefore, the increase in population growth rate of a viruliferous vector associated with nitrogen may result in virus outbreaks. From a disease management perspective, these results provide a recommendation about the timing and amount of fertilization, suggesting that fertilization should be avoided at the time of year at which the WCM disperses to green bridge plants (Miller *et al.*, 2015).

It has also been suggested that there might be a host-dependent trade-off in virus transmission capability by the WCM. Mites reared on western wheatgrass (*Agropyron smithii* Rydb.) transmit WSMV at significantly lower rates than mites reared on wheat. Once these mites have adapted to wheat, they transmit WSMV at rates comparable with those of colonies that have always been reared on wheat (del Rosario and Sill, 1965). Undoubtedly, given the increasing prevalence and spread of WSMV in many continents, there is still a demand to better understand the biology,

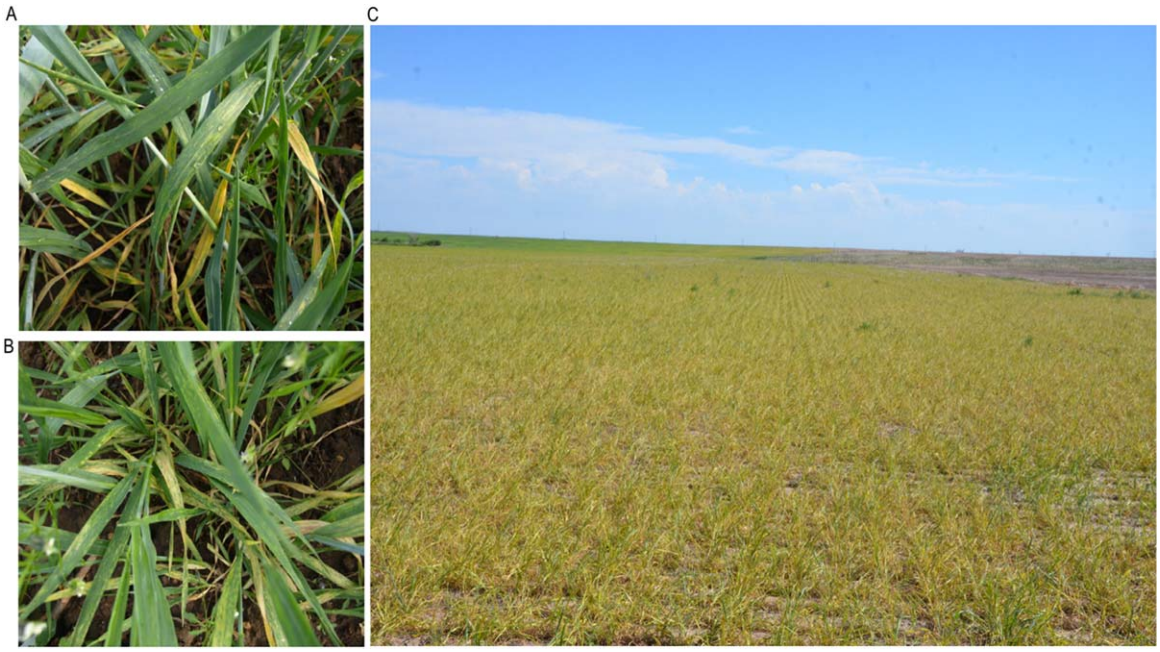

Fig. 3 *Wheat streak mosaic virus* (WSMV) disease symptoms on hosts. (A) WSMV-infected wheat cv. Cubus showing advanced symptoms with linear streaks coalescing into almost solid yellow areas. (B) WSMV-infected wheat cv. Vlada mechanically inoculated with WSMV isolate (CZlab, accession no. FJ216408). (C) A section of a wheat field affected by a severe epidemic of wheat streak mosaic in western Nebraska, USA in May 2017. Note the intense yellowing and stunting of the wheat crop.

ecology and genetics of the WCM complex in order to design effective management strategies for the WCM and associated viruses.

VIRUS TRANSMISSION BY SEEDS

WSMV transmission by seed was first described in maize in seed production fields in Iowa, and a very low percentage of seed transmission (0.1%) of the virus was found (Hill *et al.*, 1974). Jones *et al.* (2005) identified WSMV seed-borne infection in eight wheat genotypes by testing for the virus in seedlings. They found 0.2%–0.5% seed transmission across genotypes and up to 1.5% transmission in individual genotypes, indicating that the rate of transmission was lower across the wheat breeding collection tested and higher in individual genotypes. Such a low seed transmission rate is likely to have little significance epidemiologically in an individual field. However, the epidemiological significance is amplified when one considers the increased probability of global spread of the virus through local, regional and international exchange of germplasm.

HOST RANGE OF WSMV

WSMV has a wide host range, including cereals and other grass species. Wheat (*Triticum aestivum*) is a major host for the virus and the preferred host for the vector mite, *A. tosichella* biotypes 1 and 2 (lineages MT-8 and MT-1), which are known to vector the virus. Other cereal hosts include oats (*Avena sativa*), barley (*Hordeum vulgare*), rye (*Secale cereale*), maize (*Zea mays*), foxtail millet (*Setaria italica*), broom-corn millet or millet (*Panicum miliaceum*) (Table 1) (Brakke, 1971; Coutts *et al.*, 2014; Vacke *et al.*, 1986), and the mite also feeds and reproduces on these cereals. However, some cereals are susceptible to the virus, but are not good hosts for mites, for example barley (*Hordeum vulgare*) and rye (*Secale cereale*). Various annual and perennial grasses serve as hosts of WSMV, including *Agropyron repens*, *Agrostis capillaris*, *Avena fatua*, *Bromus japonicus*, *Brachypodium distachyon* and *Holcus mollis* (Table 1) (Chalupníková *et al.*, 2017; Dráb *et al.*, 2014; Mandadi *et al.*, 2014; Singh and Kundu, 2017; Wegulo *et al.*, 2008).

DISEASE SYMPTOMS

WSMV on young leaves starts as light green streaks which elongate to form discontinuous yellow to pale green stripes, forming a mosaic pattern running parallel to the leaf veins as symptoms progress in spring (Vacke *et al.*, 1986) (Fig. 3A). These symptoms are often difficult to diagnose as they can be easily confused with nutritional disorders, environmental effects or chemical damage. Plants in field margins closest to the source of WCMs are often the first, and may be the only ones, to show symptoms. With low to moderate levels of infection, a gradation of the intensity of symptoms may be seen across a field, with the most severe symptoms at the edge of the field closest to the WCM source. In severe

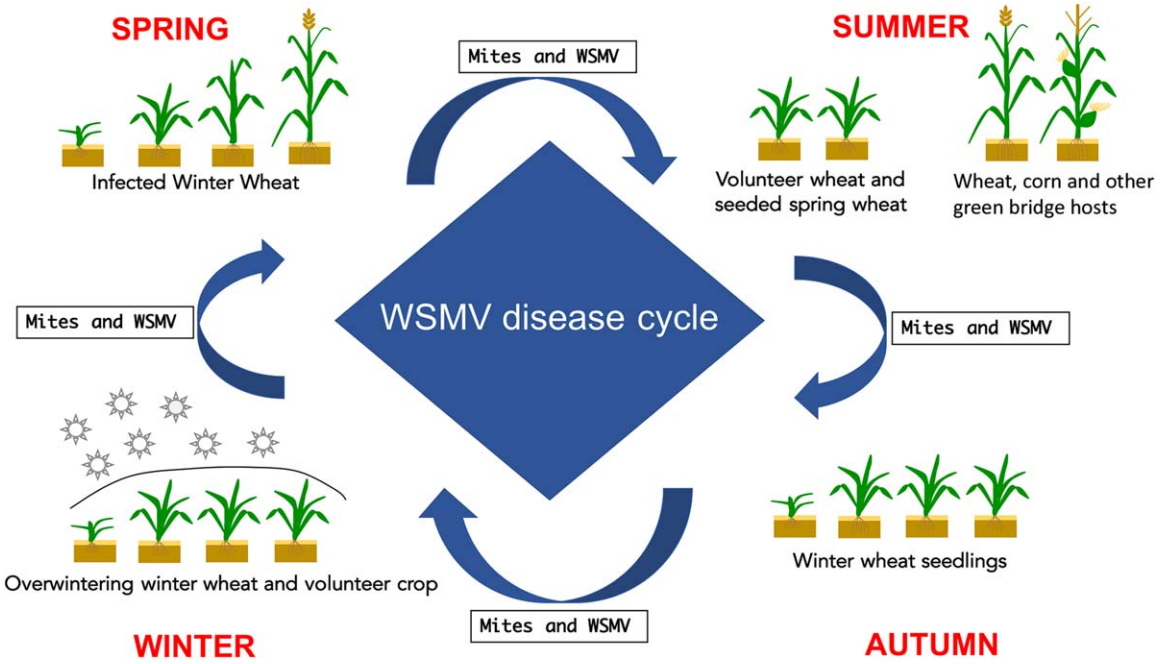

Fig. 4 The life cycle of *Wheat streak mosaic virus* (WSMV).

epidemics, plants in entire fields can become symptomatic (Fig. 3B,C). In winter wheat, infections that cause serious yield losses occur in the autumn. However, symptoms usually appear the following spring, except when there are prolonged warm temperatures late into the autumn, in which case symptoms can appear in the autumn. The appearance of symptoms in the autumn is an indication that severe epidemics may develop in the following spring. In the spring, plants infected in the autumn appear stunted, yellow, less upright than healthy plants and poorly tillered if infections occur early in the autumn. Yellowing intensifies as the temperatures become warmer. Spikes may not develop in severely infected plants or may be poorly filled with shrivelled kernels in less severely infected plants. The effects of spring infections on symptom development and yield are usually subtle (Somsen and Sill, 1970). Recent studies by Tatineni *et al.* (2017) have shown that deletion of CP amino acids 58–84 leads to the development of serious chlorotic streaks and spots, followed by acute chlorosis in wheat, maize, barley and rye, compared with mild to moderate chlorotic streaks and mosaic symptoms caused by wild-type WSMV.

DISEASE CYCLE

The only known vector of WSMV is the WCM, biotypes 1 and 2 (lineages MT-8 and MT-1) (Slykhuis, 1955). The preferred host for these lineages is wheat. However, several other cereal crops (e.g. cereal rye, maize, barley and oat) and wild grasses (e.g. couch grass, false oat-grass), which are WSMV hosts, are also hosts to the mite (Table 1). In winter wheat, initial infections occur during the autumn when viruliferous mites move from WSMV-infected volunteer wheat and other cereal and grass hosts, aided by wind, to the newly emerged wheat on which they feed and, during this process, transmit WSMV (Fig. 4).

Infections that occur in the autumn cause the most significant yield losses. The amount of yield loss is determined by the following factors: the presence of volunteer wheat and other mite and virus hosts proximal to wheat fields during planting, the density of mite populations, time of infection in the autumn, prevailing temperatures during the autumn and cultivar susceptibility. The higher the population densities of mites and mite and virus hosts near a wheat field during planting, the earlier infections occur in the autumn. The milder and more prolonged the temperatures remain in the autumn, and the higher the susceptibility of the wheat cultivar planted, the greater the yield loss (Hunger *et al.*, 1992; Slykhuis *et al.*, 1957). The mites overwinter as eggs, larvae, nymphs and adults in the crown and WSMV overwinters in the live tissues of wheat plants and other hosts.

In the spring, when temperatures warm up, mites become active and are spread by wind within and between fields. They feed and transmit the virus to healthy plants. During and after heading, mites move from the leaves and other above-ground parts of the wheat plants to sites within the spikes, in which they feed and are protected. Their populations build up to high levels during spike development. When the wheat crop matures and starts to dry down, the mites must find new hosts with green tissue on which they can feed and survive during the summer. Hence, they move to volunteer wheat and other grass hosts,

which serve as a green bridge for the mites and virus between harvesting and planting in the autumn. Following planting in the autumn, the mites move onto the newly emerged wheat and transmit WSMV, completing the disease cycle.

The WSMV disease cycle in spring wheat is similar to that in winter wheat, except that initial infections occur in spring after wheat emergence and the disease cycle is completed in the following spring, when mites move onto the newly emerged wheat and transmit the virus. Because of the timing of planting, the risk for significant losses as a result of WSMV in spring wheat is less than that in winter wheat. However, depending on the environmental conditions and proximity to spring wheat of infected winter wheat and other virus hosts with high mite populations, losses can be as significant in spring wheat as in winter wheat.

WSMV DIAGNOSIS AND QUANTIFICATION

WSMV infection has historically been detected by means of symptoms on leaves. However, symptoms on leaves are not a reliable method for the confirmation of WSMV because other viruses can cause similar symptoms. Two near-identical serological methods are available for the detection of WSMV which are based on enzyme-linked immunosorbent assay (ELISA): double antibody sandwich-ELISA (DAS-ELISA) and triple antibody sandwich-ELISA (TAS-ELISA). ELISA is the most established method for the monitoring of viruses, but is less effective than methods based on cDNA amplification (polymerase chain reaction, PCR) because of its low sensitivity (Izzo *et al.*, 2012), its inability to recognize all related viral strains (Coutts *et al.*, 2011) and its inefficiency to interpret viral accumulations (Schubert *et al.*, 2015).

WSMV has been detected by molecular methods, such as reverse transcription-polymerase chain reaction (RT-PCR) or quantitative RT-PCR (RT-qPCR) (Dráb *et al.*, 2014; Gadiou *et al.*, 2009; Schubert *et al.*, 2015). Most of the PCR-based detection protocols have targeted the viral CP gene (Gadiou *et al.*, 2009; Singh and Kundu, 2017). European isolate WSMV-ΔE has been detected by polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP) targeting the conserved *ClaI* restriction site in the core CP gene sequence (Gadiou *et al.*, 2009). Multiplex RT-PCR is being used not only for the detection of viral pathogens, but also for strain identification of viral pathogens. RT-PCR and multiplex RT-PCR provide indications of the presence or absence of WSMV, rather than the virus titre in a sample using RT-qPCR (Chalupníková *et al.*, 2017; Price *et al.*, 2010b).

In contrast, RT-qPCR has enabled the quantification of the virus concentration of several plant RNA viruses, including WSMV (Chalupníková *et al.*, 2017; Dráb *et al.*, 2014). The method is preferred for absolute virus quantification to study virus biology, virus gene expression, and virus–host and virus–vector interactions. Using RT-qPCR, Tatineni *et al.* (2010) quantified WSMV concentrations in wheat with single and double infections by WSMV and TriMV, and revealed that the two viruses induced cultivar-specific disease synergism in wheat. Using FAM (Fluorescein) and ATTO-labelled (bright fluorophores) sequence-specific probes in RT-qPCR, Schubert *et al.* (2015) revealed a higher accumulation of RNA in the USA PV57 strain compared with European isolates. Overall, RT-qPCR is preferred for absolute virus quantification to study virus biology, virus gene expression, and virus–host and virus–vector interactions.

GENETIC DIVERSITY OF WSMV

WSMV is widely distributed in the wheat-growing regions of the world, including North and South America, Australia, Asia, Europe and Russia (Table S1, see Supporting Information). The extent of the genetic diversity of WSMV has been evaluated between various isolates with different origins. Variability based on the whole genome divided WSMV isolates into three major clades, namely clade A, clade B and clade D (Schubert *et al.*, 2015) (Fig. 5A). Clade A represents isolates from Mexico, known as El-Batán. Clade B contains isolates from Europe, Russia and Turkey (Gadiou *et al.*, 2009) (Table S1). Clade B isolates from Europe, also known as *WSMV-ΔE*, are characterized by a deletion of triplet codon GCA at nucleotide position 8412 to 8414, resulting in deletion of the glycine amino acid at position 2761 in the sequence of the CP (Gadiou *et al.*, 2009). Whole-genome comparative analyses of clade B isolates revealed differences in the putative protein P1/HC-Pro cleavage site in addition to the CP gene between European, American and Asian isolates (Choi *et al.*, 2002; Schubert *et al.*, 2015). The P1/HCPro cleavage site for clade A isolates is HGLRWY/GDS, clade B isolates contain the motif HGLRWY/C(G)EP(S) and isolates from America and Asia possess the motif HGL(F)RWY/GDQ (Schubert *et al.*, 2015).

Clade D includes isolates from North and South America, Australia, Canada and Turkey (Dwyer *et al.*, 2007; Robinson and Murray, 2013). Clade D isolates of American origin are divided into four subclades: D1 contains isolates from the American Pacific Northwest (APNW); D2 contains isolates from Kansas and Colorado; D3 contains isolates from Kansas, Kentucky, Ohio and Missouri; and D4 contains isolates from Kansas and Nebraska, including Sidney 81 (French and Stenger, 2002). The characteristic triplet deletion in the CP, similar to *WSMV-ΔE* isolates from Europe, was later identified in clade D isolates originating from North America (Robinson and Murray, 2013). Earlier phylogenetic analysis based on the CP gene showed the existence of clade C, in addition to clade A, clade B and clade D (Robinson and Murray, 2013; Stenger and French, 2009) (Fig. 5B). Clade C comprises isolates from Iran (Dwyer *et al.*, 2007) (Table S1). More recent analysis of the WSMV whole genome from Iran revealed that one isolate (Iran_Saadat) clustered with clade B, and another isolate (Iran_Naghadeh) aligned together with clade D, resulting in the hypothesis of three distinct genotypes coexisting in Iran (Schubert *et al.*, 2015).

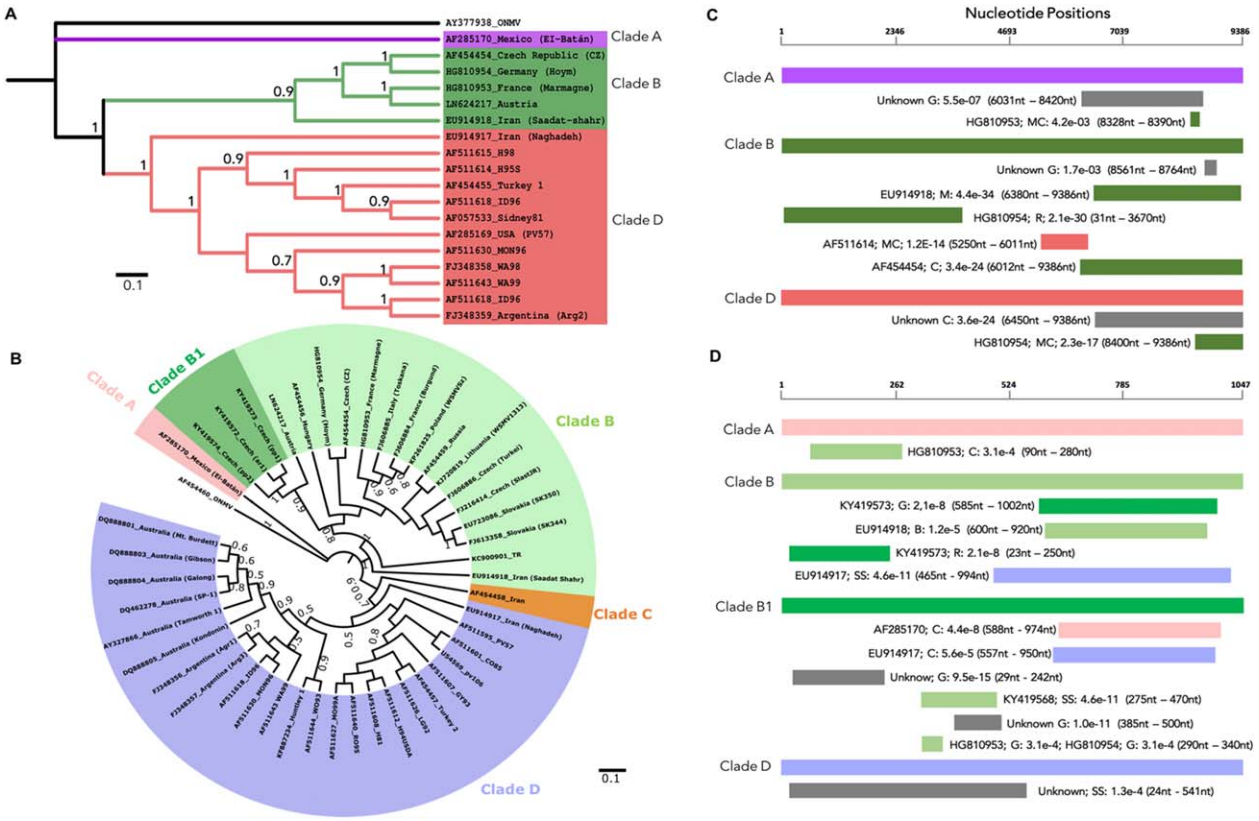

Fig. 5 Genetic diversity of various types of *Wheat streak mosaic virus* (WSMV). (A) Based on the whole genome of WSMV. (B) Based on the coat protein gene sequence of WSMV. An isolate from Mexico (El-Batán) represents clade A; isolates from Europe represent clade B and include an Asian isolate from Iran (Saadat-shahr); WSMV grass isolates from the Czech Republic are classified into type B1; an isolate from Iran (AF454458) represents clade C; isolates from the USA, Argentina, Turkey and Australia represent clade D. *Oat necrotic mottle virus* (ONMV) (AY377938_ONMV) was used as an outgroup. For the generation of the tree, nucleotide sequences were aligned using ClustalX2 (Larkin *et al.*, 2007) and the tree was constructed using MEGA 7 (Kumar *et al.*, 2016) as described previously (Singh *et al.*, 2018). The tree was viewed using ITOL (https://itol.embl.de/). The neighbour-joining method was used for the construction of the tree and the reliability of the branches was inferred from a bootstrap analysis of 1000 replicates. The dataset supporting the results for the study has been submitted to the TreeBASE repository (http://treebase.org/treebase-web/home.html) and is publicly accessible at http://purl.org/phylo/treebase/phylows/study/TB2:S22140. (C, D) Recombinant analysis of WSMV based on the full genome and coat protein nucleotide sequences. Analyses were performed using various algorithms included in the RDP software package (Martin *et al.*, 2015) as described previously (Singh and Kundu, 2017). The type strain AF285169_PV57 (USA) was used as a reference. The order of the designation of the recombination events is as follows: accession number, algorithm used (R, RDP; G, GENECONV; C, Chimaera; MaxChi; B, Bootscan; SS, SiScan; 3seq; LARD), *P*-value and nucleotide position. Only recombination events with $P < 0.05$ detected by at least three different algorithms are shown. Numbers of events with $P > 0.05$ are given in parentheses. Recombination events were observed in five algorithms: RDP, GENECONV, Bootscan, Chimaera and SiScan. Algorithms MaxChi, 3seq and LARD did not detect significant recombination events.

WSMV has a diverse host range, and grasses serve as one of the important natural reservoirs of the virus. It has been revealed recently that WSMV which infects grasses from the Czech Republic shares high similarity with clade B isolates from other countries in Europe. Therefore, a new clade has been introduced, and is known as clade B1 (Singh and Kundu, 2017). Based on the CP gene sequence, clade A isolates share ~79% (nucleotides) and 83% (amino acids) with clade B isolates, 73% (nucleotides) and 76% (amino acids) with clade B1 isolates (grasses) and 78% (nucleotides) and 84% (amino acids) with clade D isolates. Clade B isolates share a high similarity of ~92% (nucleotides) and 94% (amino acids) with clade B1 isolates and ~90% (nucleotides) and 95% (amino acids) with clade D isolates. Clade B1 isolates, represented by grasses from the Czech Republic, show a similarity of 85% (nucleotides) and 88% (amino acids) to clade D isolates (Singh and Kundu, 2017).

Whole-genome recombinant analysis of various WSMV clades has shown that recombination mainly occurs at the 3′ end of the sequence (Schubert *et al.*, 2015). Clade A isolates recombine with clade B isolate, *HG810953_Marmagne*, from France (8328–8390 nucleotides) (Fig. 5C). Clade B isolates (Europe/Asia) recombine only with isolates from within this cluster, as well as with clade D isolates (5250–6011 nucleotides) (Fig. 5C). Clade D isolates show recombination events with clade B isolate *HG810954_Hoym* from

Germany (8400–9386 nucleotides). In addition, recombination prediction based on a partial CP gene sequence has revealed that clade A isolates recombine with isolates from clade B (isolate *HG810953_Marmagne* from France at 90–280 nucleotides) (Singh and Kundu, 2017) (Fig. 5D). Clade B isolates recombine within this cluster, with clade B1 (isolate KY419573_P. pretense at 585–1002 nucleotides; 23–250 nucleotides) and clade D (Fig. 5D). Clade B1 isolates are predicted to recombine with all three clades: clade A, clade B and clade D (Singh and Kundu, 2017) (Fig. 5D). There is an unknown recombination that occurs in clade D. However, further experimental investigations are required to decipher the potential outcome of recombinant analysis to expand our understanding of the various clades of WSMV.

PLANT RESISTANCE TO WSMV

Resistance to WSMV was first reported in perennial Triticeae relatives, such as *Thinopyrum intermedium* and *Thinopyrum ponticum* (Chen *et al.*, 2003; Friebe *et al.*, 1993; Harvey *et al.*, 1999). Three resistance genes have been identified: *Wsm1*, *Wsm2* and *Wsm3* (Fahim *et al.*, 2012b; Friebe *et al.*, 2009; Haley *et al.*, 2002; Lu *et al.*, 2012). These resistance genes have been introduced into cultivated wheat lines. The resistance gene *Wsm1* is associated with chromosome 4D and has led to the release of the winter wheat cultivar Mace (Graybosch *et al.*, 2009). The resistance gene *Wsm2* is associated with chromosome arm 3BS and has led to the release of several cultivars of wheat, including RonL (Seifers *et al.*, 2006), Snowmass (Haley *et al.*, 2011), Clara CL (Martin *et al.*, 2014) and Oakley CL (Zhang *et al.*, 2015). However, both *Wsm1* and *Wsm2* are ineffective at higher temperatures (Seifers *et al.*, 2013). The third true resistance gene, *Wsm3*, has recently been identified and has been proven to be effective at higher temperatures than *Wsm1* and *Wsm2* (Fahim *et al.*, 2012b). However, *Wsm3* is not yet available in any commercial wheat cultivars (Richardson *et al.*, 2014). The commercially available WSMV-resistant wheat cultivars were developed in the USA and there are no reports of WSMV-resistant cultivars or other cereal species in Europe.

Resistance genes to the WCM vector have been identified in grass species: *Aegilops tauschii* ($2n = 2x = 14$, DD), *Thinopyrum ponticum*, ($2n = 10x = 70$, JJJJsJs) and *Th. intermedium* ($2n = 6x = 42$, JJsS) (Fahim *et al.*, 2011; Fedak and Han, 2005; Qi *et al.*, 1979). The grass genes intercross to hexaploid wheat, but very few wheat cultivars possess effective resistance against the WCM because of virulent WCM populations (Hakizimana *et al.*, 2004; Martin *et al.*, 1976; Murugan *et al.*, 2011). However, WCM resistance remains a compelling approach to reduce losses caused by WSMV. Two distant hybrids between spring wheat and the grass *Agropyron glaucum*, Zhong1 and Zhong2, show effective resistance towards both WSMV and its WCM vector (Chen *et al.*, 2003; Han *et al.*, 2003; Qi *et al.*, 1979).

DISEASE MANAGEMENT

The management of WSMV is aimed at the minimization or elimination of the risks of infection of wheat. The highest risk is volunteer wheat which emerges in a wheat field just before harvest following a hailstorm. Other risks include: volunteer wheat in summer crops other than wheat; crops or grassy weeds that are hosts of WCMs or WSMV, e.g. maize, that are allowed to grow past autumn wheat emergence; a cool, wet summer which favours the growth of volunteer wheat and other hosts, as well as the survival and reproduction of WCMs, and also prolongs the period of growth of summer host crops; a prolonged autumn with above normal temperatures; and early planting of wheat.

Because WSMV cannot be controlled by chemicals and the chemical control of WCMs is ineffective (Fritts *et al.*, 1999), the most effective strategy for the management of WSMV is to use cultural practices. Pre-harvest volunteer wheat, especially volunteer wheat that emerges in a wheat field as a result of a hailstorm, should be controlled with herbicides or tillage. Post-harvest volunteer wheat should also be controlled. To be effective, volunteer wheat should be completely dead at least 2 weeks before planting. Grassy weeds in and close to fields in which wheat will be planted in the autumn should be controlled with tillage or herbicides. Early planting of wheat should be avoided. The combined effects of mites and virus when wheat is planted early include heavy and widespread infections in the autumn, leading to severe epidemics the following spring that result in substantial yield losses.

Wheat should not be planted next to late-maturing summer crops that are hosts to WCMs or WSMV, such as maize, foxtail millet, sorghum or small grain cover crops. When available, wheat cultivars with greater resistance or tolerance to WSMV that are adapted to the local area or region should be planted. High-risk wheat fields should be planted last. These are the fields adjacent to grassy weeds and late-maturing host crops. An integrated disease management approach that combines as many as possible of these strategies and tactics will most effectively reduce losses caused by WSMV, as illustrated in McMechan and Hein (2016), who showed that cultivar resistance and delayed planting improved the yields of three winter wheat cultivars under high WSMV intensity.

CONCLUSIONS AND FUTURE DIRECTION

WSMV continues to be a threat to wheat production worldwide. Research is needed that will provide information to enhance our understanding of the biology, ecology and epidemiology of the disease and its WCM vector, including the knowledge that the WCM constitutes a species complex. Improved techniques for rapid detection and diagnosis will be essential to growers in making timely and informed management decisions. These techniques include the use of molecular tools, such as RT-PCR and RT-qPCR.

In addition, the increasingly common whole-genome sequencing approach provides the opportunity to search for signatures of polyphagy, detoxification and WSMV vectoring abilities in different WCM biotypes, which offers new possibilities for the development of wheat protection strategies. The genetic engineering of resistance to WSMV in wheat, for example through the expression of artificial polycistronic microRNA (Fahim *et al.*, 2012a) and gene silencing (Li *et al.*, 2005), will complement traditional resistance breeding strategies to achieve higher and more effective levels of resistance. One recent addition to genetic engineering is the development of the characteristic clustered regularly interspaced short palindromic repeats/CRISPR-associated 9 (CRISPR/Cas9) protein that has emerged as a potent genome-editing tool to confer resistance against geminiviruses (Baltes *et al.*, 2015). Therefore, it will be intriguing to implement CRISPR/Cas9 to modify WSMV/WCM genes in order to develop effective resistance against virus and vector.

WSMV mutants with deletion in amino acids in the CP region are capable of systemic infection, although with delayed and milder symptoms (Tatineni and French, 2014). Therefore, the availability of a series of viable CP deletion mutants of WSMV will greatly facilitate our understanding of the complexity of WSMV–host interactions. Furthermore, it will be interesting to identify the different amino acids in different strains of WSMV that are vital for the interactions with the vector and hosts using molecular approaches. The interaction of viruses with their hosts is a rather complex and dynamic process, involving numerous interactions amongst viral proteins and host proteins. An improved understanding of the complex interactions of WSMV-derived proteins that alter the host cellular machinery, as well as the identification of host genes, will contribute to the development of novel sources of resistance and other control measures. New technology, such as next-generation sequencing (RNA-sequencing) of hosts infected with WSMV, will provide valuable insights into host factors that differentially interact with the virus, thus enhancing our understanding of the mechanisms of host–virus interaction, as well as the nature and mechanisms of long-distance transport of viruses in monocot plants.

Climate change poses new challenges because of its influence on the biology, ecology and epidemiology of WSMV and its WCM vector. The current trend in climate change is towards warmer temperatures globally. The implications of this trend are that there will be more frequent outbreaks of severe WSMV epidemics over larger areas or regions. The increased frequency of outbreaks of severe epidemics, coupled with an increased probability of long-distance dispersal through the exchange of infected germplasm amongst researchers locally, regionally and globally, means that greater yield losses will be expected. Concerted efforts will be needed to mitigate these losses. These will include breeding for resistance to WSMV and the WCM using traditional methods, as well as molecular tools; vigilance in implementing management tactics, especially the control of volunteer wheat and other crop and grass hosts of WSMV and the WCM; modification or adaptation of management tactics to account for climate change and differences in the biology and ecology of WCM biotypes; and educating growers, crop consultants, extension educators and the public about the disease and how to manage it to protect yields.

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SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article.

Table S1 Diversity of *Wheat streak mosaic virus* isolates.