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Genetics and mechanisms underlying transmission of *Wheat streak mosaic virus* by the wheat curl mite

Satyanarayana Tatineni¹ and Gary L Hein²

Wheat streak mosaic virus (WSMV, genus *Tritimovirus*; family *Potyviridae*) is the most economically important virus of wheat in the Great Plains region of the USA. WSMV is transmitted by the eriophyid wheat curl mite (WCM), *Aceria tosichella* Keifer. In contrast to Hemipteran-borne plant viruses, the mode and mechanism of eriophyid mite transmission of viruses have remained poorly understood, mostly due to difficulty of working with these ~200 μ m long microscopic creatures. Among eriophyid-transmitted plant viruses, relatively extensive work has been performed on population genetics of WCMs, WSMV determinants involved in WCM transmission, and localization of WSMV virions and inclusion bodies in WCMs. The main focus of this review is to appraise readers on WCM, WSMV encoded proteins required for WCM transmission and further details and questions on the mode of WSMV transmission by WCMs, and potential advances in management strategies for WCMs and WSMV with increased understanding of transmission.

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Introduction

Transmission by arthropod vectors is the most effective means of disseminating plant viruses. Viruses have evolved precise vector-specific transmission interactions between viral and vector proteins that determine the nature and efficiency of transmission. Arthropod transmission of plant viruses is divided into three broad categories based on the interactions between the virus and vector. These categories are nonpersistent, semipersistent, and persistent transmission [1^{••},2^{••}]. In

nonpersistent transmission or stylet-borne transmission, the virus can be transmitted only for a few minutes after a relatively brief period of acquisition (<5 min). In semipersistent or foregut-borne transmission, vectors acquire viruses after minutes to hours of feeding and transmission is retained for hours to days. The site of virus interaction with the vector in these methods is the stylet or the foregut. Because the lining of the stylet and foregut are lost in the molting process, retention of these viruses is limited, and viruses are not retained through the molt. Persistent transmission is defined as transmission with the most intimate interactions between virus and vector and is subdivided into circulative and propagative [3,4^{••}]. In circulative transmission, viruses move through the vector, from the gut lumen into the hemolymph or other tissues and finally into the salivary glands, where they are introduced back into the plant during vector feeding. In propagative transmission, viruses also circulate through the vector, but they are able to replicate in various tissues within the vector. Because of their prevalence within the vector, they often can be transmitted to progeny of the vector via transovarial transmission.

Hemipteran insects (e.g. aphids, leafhoppers and whiteflies) are the most common vectors of plant viruses, but the family Eriophyidae (Subclass Acari) includes eleven species of mites that serve as vectors of several plant viruses [5[•]]. Wheat curl mite (WCM; *Aceria tosichella* Keifer) belongs to the family Eriophyidae in the superfamily Eriophyoidea [6]. Approximately 4000 species of eriophyid mites have been reported [7,8]; however, all known eriophyid species that serve as vectors of plant viruses belong to the family Eriophyidae [5[•],6]. Recently, Stenger *et al.* [5[•]] presented a review of eriophyid mites and the viruses transmitted by these mites. In this review, we will focus specifically on the WCM and its transmitted viruses, with special emphasis on *Wheat streak mosaic virus* (WSMV).

Wheat viral diseases

Wheat viral diseases in the Great Plains region of North America can cause 3–5% annual yield loss [9,10]. Viruses infecting wheat in the Great Plains include *Agropyron mosaic virus* (AgMV), *Barley yellow dwarf virus*, *High Plains wheat mosaic virus* (HPWMoV), *Soil-borne wheat mosaic virus*, *Triticum mosaic virus* (TriMV), *Wheat American striate mosaic virus*, and WSMV [10–12]. Among these viruses, HPWMoV, TriMV, and WSMV are transmitted by the WCM [13–15]. It appears that the transmission capabilities of HPWMoV and TriMV by WCM are similar to those of

WSMV (see below) ([14–17]; G. Hein, unpublished]. However, further studies are needed to unravel the transmission characteristics of TriMV and HPWMoV. Wheat infections by WSMV, TriMV and HPWMoV are widespread across the Great Plains, and co-infection of wheat by two or all three viruses is common in growers' fields with exacerbated yield loss [18–20]. WCM-transmitted viruses are the most economically important viral pathogens of wheat in the Great Plains region with an estimated annual yield loss of ~5.44 million quintals valued at \$120 million (at \$6/27.2 kg) [9,10], and these losses are often expressed as severe infections with up to 100% yield loss in localized areas.

Biology and genetics of wheat curl mites

WCMs, like other eriophyid mites, are much smaller in size than other arthropod vectors with an average length of ~200 μm (Figure 1) [21,22]. The short stylets of WCMs limit the depth of penetration of the plant to about 5 μm ; thus, they can penetrate only the epidermis of the plant [23,24]. Besides their size, the most distinctive feature of WCMs is that they possess only two pair of well-developed legs located near the front end of their elongated body (Figure 1). WCMs have a relatively simple life cycle. After hatching from the egg, they undergo two immature stages (larval and nymphal), each lasting about 2–3 days, before becoming adults. Between each of these stages, the mites spend a short quiescent period (<1 day) before molting to the next stage. The time period from egg to adult requires 7–9 days. Eriophyid mites undergo arrhenotokous reproduction with the males being haploid and a single female capable of establishing a colony [8]. The rapid developmental rate coupled with this reproductive strategy results in

tremendous reproductive potential. The WCM relies primarily on wind currents for movement, and the spread of mite-transmitted viruses benefits from the mite's extreme reproductive capabilities.

Analyses of portions of ribosomal ITS1 and mitochondrial DNA sequences of WCM populations from Australia and the Great Plains demonstrated the presence of two comparable genotypes, each present in both Australia and North America [25,26]. Hein *et al.* [26] identified the two genotypes of WCMs from five populations collected across the Great Plains: Type 1 (collected from Kansas, Texas, Montana and South Dakota) and Type 2 (collected from Nebraska). Additional work has shown that these genotypes are both commonly present across the Great Plains [27], and little evidence has been found in either North America or Australia to suggest extensive interbreeding of these two genotypes even though they commonly occur together [25–27]. These two genotypes have also commonly been found in Europe and other global regions [28]; however, Skoracka *et al.* [29] and Szydło *et al.* [30] have revealed even greater genetic diversity in WCMs from Poland and Turkey and identified the wheat curl mite as a cryptic species complex.

The genetic differences in WCM populations correspond to variable transmission efficiencies of different wheat viruses. For example, the same five WCM populations identified by Hein *et al.* [26] from the Great Plains areas differentially transmitted HPWMoV isolates, but not WSMV [31]. However, Wosula *et al.* [32] found both Type 1 and Type 2 mites from the Great Plains to be effective at transmitting WSMV, but Type 2 mites consistently transmitted WSMV at higher rates. Alternatively, Schiffer *et al.* [33] found that Type 2 mites in Australia transmitted WSMV well, but Type 1 mites were not able to transmit WSMV. McMechan *et al.* [17] showed that Type 2 but not the Type 1 WCM effectively transmitted TriMV. WCMs exhibited mutualistic (Type 2 only) and antagonistic (both Type 1 and 2) effects on WCM reproduction with WSMV and TriMV, respectively, in infected wheat [17,27,34]. The observed negative effects of TriMV infection on WCM population might explain the lower incidence of TriMV most often found in growers' fields [35]. Additional work on comparison of transmission capabilities of various genotypes of wheat curl mites across world regions or continents would be ideal, especially if they could be tested with the same virus isolates for best comparison.

WSMV and its transmission characteristics by WCM

WSMV is the type species of the genus *Tritimovirus* within the family *Potyviridae* [36]. WSMV virions are flexuous filaments (690–700 nm $\times$ 11–15 nm) (Figure 2a) encapsidating a single positive-strand genomic RNA of 9384 nucleotides. The genomic RNA is polyadenylated at the 3' end and probably with a VPg (virus protein linked

Figure 1

Scanning electron micrograph of an adult wheat curl mite (*Aceria tosichella* Keifer) feeding on a wheat leaf. Note that two pairs of legs attached to the front end of the elongate mite and also see a pair of eggs located next to the mite.

Figure 2

(a) Electron micrograph picture showing flexuous filamentous virion particles of WSMV. Bar indicates 100 nm. **(b)** Schematic diagram of *Wheat streak mosaic virus* (WSMV) genomic organization with a large open reading frame (ORF) encoding for a polyprotein. Individual mature proteins of polyprotein encoded by WSMV are indicated inside or above the genome organization. Vertical lines within large ORF indicate the location of cleavage peptides for proteolytic processing by three virus-encoded proteinases. Expanded views of HC-Pro and CP citrons required for WSMV transmission by the wheat curl mite (WCM). Black boxes within HC-Pro and CP citrons indicate deletion of indicated amino acids abolished WCM transmission. Location of cysteine residues in HC-Pro citron at amino acid positions 16, 46 or 49 that are required for WCM transmission of WSMV are indicated. Location of aspartic acid residues in the CP citron for efficient WCM transmission of WSMV are also indicated.

to the genome) covalently attached to the 5' end [36]. The genomic organization of WSMV is similar to that of members of the aphid-transmitted *Potyvirus* genus of the family *Potyviridae* with a single large open reading frame encoding a polyprotein of ~350 kDa (Figure 2bA). The polyprotein is processed by three virus encoded proteinases (P1, HC-Pro and NIa-Pro) into at least 10 mature proteins analogous to potyviruses. Young *et al.* [37] reported that P1 of WSMV, but not HC-Pro as in potyviruses, is the suppressor of RNA silencing.

Under experimental conditions, WCMs efficiently transmit WSMV up to 50% and 100% with single and 5–10 viruliferous mites per test plant, respectively [32]. The virus cannot be transovarially transmitted, and only immature wheat curl mites can acquire the virus. Acquisition can occur in as little as 15 min with transmission effectiveness increasing with acquisition time up to about 16 hours [24,27,38]. All stages of mites can transmit virus, but immature mites are more effective at transmission [24,27]. The virus persists for at least seven days in adult

mites kept on virus-immune plants [24], but transmission efficiency of adults decreases with time [24,27].

WSMV determinants for transmission by the WCM

The availability of an efficient reverse genetics system facilitated the examination of functions of various WSMV-encoded proteins in virus biology [39–42,43^{••},44,45]. In contrast to the multi-functional HC-Pro of members of the genus *Potyvirus* [46–49], WSMV HC-Pro is dispensable for systemic infection of wheat [42]. This facilitated the examination of the role of HC-Pro in vector transmission. Replacement of WSMV HC-Pro with that of *Turnip mosaic virus* (an aphid-borne potyvirus), AgMV (a different eriophyid mite-transmitted rymovirus), or *Oat necrotic mottle virus* (a tritrovirus with no known vector) abolished mite transmissibility of WSMV [43^{••}]. In contrast, a chimeric WSMV genome with HC-Pro of a divergent WSMV isolate El Batán 3 was efficiently transmitted by WCMs. A series of 5'-proximal nested deletions in the HC-Pro cistron of WSMV revealed that deletion of as few as eight amino acid codons (codons 3–10) at the 5' end of HC-Pro cistron completely abolished WCM transmission (Figure 2bA; [44]). Further analyses of the HC-Pro cistron through alanine substitution of cysteine residues at amino acid positions 16, 46, or 49 abolished WCM transmission (Figure 2bA). This suggests a zinc-finger-like motif (His₁₃-X₂-Cys₁₆-X₂₉-Cys₄₆-X₂-Cys₄₉) in WSMV HC-Pro is required for WSMV transmission by WCMs [50].

The CP cistron of WSMV is unique in that it tolerates extensive deletions and point mutations at the amino-proximal and carboxy-terminal regions for systemic infection of wheat [45,51,52]. Moreover, WSMV mutants lacking CP amino acids 36–84 accumulated more efficiently in wheat compared to wild-type virus [51,53]. The role of CP in WSMV transmission by the WCM was examined using deletion and point mutations and found that CP amino acids 58–100 are required for WCM transmission (Figure 2bA; [54[•]]). This revealed that WSMV CP is a determinant of WSMV transmission by the WCM, similar to members of the genus *Potyvirus*. Additionally, the aspartic acid residues at amino acid positions 289 and 326 were found to be required for efficient mite transmission (Figure 2bA). Interestingly, the WCM-transmitted progeny of mutants D289A and D326A harbored a second-site mutation of R131C and N275H, respectively, but these second-site mutations were not found in wheat infected by *in vitro* transcripts of mutant viruses [54[•]]. This raises the question of the origin of the second-site mutations occurring only through mite transmission and suggests that these mutations arise during virus replication within the mite. These data together with the fact that only juveniles acquire the virus and mites remain viruliferous through molting and as adults suggest that WCM transmit WSMV in a persistent manner (see below).

Mechanism of WCM transmission of WSMV

CP and HC-Pro are identified as vector transmission determinants of both WCM-transmitted WSMV [43^{••},54[•]] and aphid-transmitted potyviruses [47,55], but these viruses differ in the mode of vector transmission. In the potyvirus bridge model, HC-Pro provides a link between virus particles and aphid stylets through interaction with CP, and this facilitates virion attachment to aphid stylets [56,57]. In potyviruses, HC-Pro and CP interaction was mediated by the PTK motif in HC-Pro and the DAG motif in CP [58–60], and the KITC motif of HC-Pro mediates interaction with the aphid stylet [56,61]. However, these motifs were absent in the HC-Pro and CP cistrons of WSMV [36], and interaction between HC-Pro and CP was not observed in the yeast two-hybrid system and pull-down assay [40]. This indicates the actual mechanism behind the HC-Pro- and CP-mediated WCM transmission of WSMV is different from that of aphid-transmitted potyviruses. These differences are reinforced by the fact that WSMV transmission by WCMs is not stylet or foregut borne as WCMs remain viruliferous after molting.

The accumulation of WSMV virions in wheat curl mites was observed in the sac-like posterior midgut [62], not in the hindgut as reported earlier [63[•],64[•]] due to misidentification of the posterior midgut. In addition to virus-like particles, two types of inclusion bodies were found in the digestive tract of viruliferous mites [63[•]]. These observations lead to several unanswered questions regarding the nature of WSMV transmission by mites: What is the nature of these inclusion bodies? Are these inclusion bodies produced as the result of overexpression of WSMV proteins due to virus replication, as observed in WSMV-infected plants? Recently, we also found that WSMV in WCMs exhibited 0.3% sequence diversity in 0.4–4.2% of virus-specific RNA-seq reads (A. Gupta and S. Tatineni, unpublished). Collectively, the observed second-site mutations in mite-transmitted progeny of CP aspartic acid mutants [54[•]], sequence diversity in WSMV genome from wheat curl mites (unpublished data), and detection of virions and inclusion bodies in WCMs [63[•]] suggests that WSMV replicates in wheat curl mites. Thus, it was suggested that WCMs transmit WSMV in a 'circulative' manner [62].

The mode of transmission of WSMV by WCM has been difficult to fully classify using the current categories. The mites' ability to retain WSMV through the molt supports its classification as a persistent virus. In contrast, vectors of semipersistently transmitted *Beet yellows virus*, *Cauliflower mosaic virus*, *Strawberry mottle virus*, and *Strawberry vein banding virus* lose infectivity after molting [1^{••},65,66]. However, the short acquisition times as demonstrated for the mite are not characteristic of persistent viruses, although Hogenhout *et al.* [4^{••}] suggests shorter acquisition times may result from feeding on epidermal cells. A

significant latent period (hours to weeks) is identified as a characteristic of a persistent virus [1^{••},4^{••}]. Because of its short acquisition times, however, there does not appear to be a latent period for WSMV transmission by WCM. There is evidence accumulating that the virus may replicate in the mite ([62,63[•],64[•]]; unpublished data) which would place this transmission interaction in the persistent propagative category. However, the virus is not retained for the life of the mite as transmission efficiency decreases for adults and also decreases with adult age [24,27]. The classification of this mite-virus transmission relationship does appear to mostly fit the category of a persistent virus, but there are clear distinctions. These distinctions may not be surprising when considering the minute size of the mite and its restricted feeding characteristics. Further work is needed to unequivocally confirm the nature of WCM transmission of WSMV.

Management strategies of WSMV

Diseases caused by WCM-transmissible WSMV, HPWMoV, and TriMV are economically important on wheat in the Great Plains. Symptoms elicited by all three viruses are similar on wheat, hence diseases caused by individual- or mixed-infections by these viruses are called wheat streak mosaic (WSM) disease complex. These viruses primarily infect winter wheat through viruliferous WCMs moving from over-summering host plants (e.g. volunteer wheat, maize and other grasses). Hence, cultural practices and deployment of WCM- and virus-resistant wheat cultivars are needed for effective management of the WSM disease complex. Management of over-summering hosts for the mite is the primary cultural practice for controlling this disease complex. Delayed planting can also avoid virus infections for the management of WSM disease complex [67,68]. However, delayed planting has potential to reduce wheat yield due to fewer growing days before the onset of winter.

WCMs not only transmit economically important wheat viruses but nonviruliferous mites can cause yield loss in wheat when spikes are infested with high mite population due to their enormous reproductive capabilities [69]. However, these losses are not nearly as extreme as potential losses from virus impact. The development of WCM-resistant wheat cultivars can aid in management of WCMs and WCM-transmitted viruses. Thus far, four curl mite colonization (*Cmc*) genes have been identified, and others are being developed in breeding programs. Wheat cultivars or lines with DNA transferred from *Aegilops tauschii*, (*Cmc1*, *Cmc4*), *Agropyron elongatum* (*Cmc2*) and *Secale cereal* (*Cmc3*) have provided resistance to WCMs [70–75]. However, Harvey *et al.* [76] reported that after extensive deployment of the resistance gene (*Cmc3*) in cv. TAM 107, WCM populations adapted to this resistance gene. This suggests that multiple lines of resistance may need to be deployed to provide durable WCM resistance.

Limited resistance to WSMV has been found in wheat through the years; however, efforts to transfer resistance from close relatives of wheat have resulted in some recent successes. Thus far, two wheat streak mosaic resistant genes have been transferred on chromosomal translocations from *Thinopyrum intermedium* (*Wsm1* and *Wsm3*) [77–79]. However, these translocations remain problematic in the breeding process with wheat and have limited their usefulness. A third gene (*Wsm2*) was identified by Haley *et al.* [80]. The origin of this gene is not clear but it is suspected to have originated from wheat, and it has now been incorporated into several commercial lines [81,82]. Recent successes in identifying and incorporating virus resistance genes into wheat are encouraging. However, recent studies showing tendencies for greater mite buildup in virus-resistant wheat cultivars because the plants remain healthy may demonstrate an increased importance for pyramiding mite-resistance with virus-resistance in wheat (G. Hein, unpublished).

Future research directions

The advent of modern technologies will facilitate studies on the identification of WCM gene products involved in virus transmission, followed by silencing of such genes through RNA interference (RNAi) technology. The same techniques can also be applied to study interactions between WCM and wheat, and this information can be utilized to achieve improved resistance of WCMs on wheat. Additionally, the actual mechanisms of WCM transmission of WSMV are not clear. Experiments on temporal accumulation of negative-sense genomic RNA copies and nonstructural proteins of WSMV in WCMs would answer whether WSMV can replicate in WCMs. These studies would facilitate unraveling WCM-WSMV interactions and expand our search for potential targets for developing improved and durable resistance.

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References and recommended readings

Papers of particular interest, published within the scope of this review have been highlighted as:

- of special interest
- of special interest

1. Ng JCK, Falk BW: **Virus–vector interactions mediating nonpersistent and semipersistent transmission of plant viruses.** *Annu Rev Phytopathol* 2006, **44**:183-212.

Excellent review describing nonpersistent and semipersistent transmission of plant viruses.

2. Whitfield AE, Falk BW, Rotenberg D: **Insect vector-mediated transmission of plant viruses.** *Virology* 2015, **479**:278-289. Excellent review describing vector transmission of plant viruses.
3. Gray S, Gildow FE: **Luteovirus-aphid interactions.** *Annu Rev Phytopathol* 2003, **41**:539-566.
4. Hogenhout SA, Ammar E, Whitfield AE, Redinbaugh MG: **Insect vector interactions with persistently transmitted viruses.** *Annu Rev Phytopathol* 2008, **46**:327-359. Excellent review on persistent transmission of plant viruses.
5. Stenger DC, Hein GL, Tatineni S, French R: **Eriophyid mite vectors of plant viruses.** In *Vector-mediated Transmission of Plant Pathogens*. Edited by Brown JK. The American Phytopathological Society; 2016:263-274. Excellent review on eriophyid mite vectors of plant viruses.
6. Oldfield GN: **Diversity and host plant specificity. Evolution of eriophyid mites in relation to their host plants.** In *World Crop Pests. Vol. 6, Eriophyid Mites: Their Biology, Natural Enemies and Control*. Edited by Lindquist EE, Sabelis MW, Bruin J. Amsterdam: Elsevier Science Publishing; 1996:199-216.
7. Amrine JW Jr, Stasny TA: *Catalog of the Eriophyidae (Acarina: Prostigmata) of the World*. West Bloomfield, MI: Indira Publishing House; 1994.
8. de Lillo E, Skoracka A: **What's cool on eriophyid mites?** *Exp Appl Acarol* 2010, **51**:3-30.
9. Appel J, DeWolf E, Todd T, Bockus W: **Preliminary 2015 Kansas wheat disease loss estimates.** *Kansas Cooperative Plant Disease Survey Report*. 2015 <https://agriculture.ks.gov/docs/default-source/pp-disease-reports-2012/2015-ks-wheat-disease-loss-estimates4ec4d4002e2e1aa5bfff0000620720.pdf?sfvrsn=0>.
10. Brakke MK: **Virus disease in wheat.** In *Wheat and Wheat Improvement*, 2nd ed.. Edited by Heyne EG. Madison, WI: American Society of Agronomy, Crop Science Society of America, Soil Science Society of America; 1987:85-603.
11. Jensen SG, Lane LC, Seifers DL: **A new disease of maize and wheat in the high plains.** *Plant Dis* 1996, **80**:1387-1390.
12. Seifers DL, Martin TJ, Harvey TL, Fellers JP, Stack JP, Ryba-White M, Haber S, Krokshin O, Spicer V, Lovat N, Yamchuk A, Standing KG: **Triticum mosaic virus: a new virus isolated from wheat in Kansas.** *Plant Dis* 2008, **92**:808-817.
13. Slykhuis JT: **Aceria tulipae Keifer (Acarina: Eriophyidae) in relation to spread of wheat streak mosaic virus.** *Phytopathology* 1955, **45**:116-128.
14. Seifers DL, Harvey TL, Martin TJ, Jensen SG: **Identification of the wheat curl mite as the vector of the High Plains virus of corn and wheat.** *Plant Dis* 1997, **81**:1161-1166.
15. Seifers DL, Martin TJ, Harvey TL, Fellers JP, Michaud JP: **Identification of the wheat curl mite as the vector of Triticum mosaic virus.** *Plant Dis* 2009, **93**:25-29.
16. Knoch EA: **Transmission Characteristics of Triticum mosaic virus by the Wheat Curl Mite *Aceria tosichella* Keifer and Ecology of the Wheat-Mite-Virus Complex on Field Corn.** . Dissertations and Student Research in Entomology University of Nebraska-Lincoln; 2018 . 114 pp. <https://digitalcommons.unl.edu/entomologydiss/53/>.
17. McMechan AJ, Tatineni S, French R, Hein GL: **Differential transmission of Triticum mosaic virus by wheat curl mite populations collected in the Great Plains.** *Plant Dis* 2014, **98**:806-810.
18. Burrows M, Franc G, Rush C, Blunt T, Ito D, Kinzer K, Olson J, O'Mara J, Price J, Tande C, Ziems A, Stack J: **Occurrence of viruses in wheat in the Great Plains region, 2008.** *Plant Health Prog* 2009 <http://dx.doi.org/10.1094/PHP-2009-0706-01-RS>.
19. Byamukama E, Tatineni S, Hein GL, Graybosch RA, Baenziger PS, French RC, Wegulo SN: **Quantification of yield loss caused by Triticum mosaic virus and Wheat streak mosaic virus in winter wheat under field conditions.** *Plant Dis* 2014, **98**:127-133.
20. Tatineni S, Graybosch RA, Hein GL, Wegulo SN, French R: **Wheat cultivar-specific disease synergism and alteration of virus accumulation during co-infection with Wheat streak mosaic virus and Triticum mosaic virus.** *Phytopathology* 2010, **100**:230-238.
21. Lindquist EE: **External anatomy and notation of structures.** In *World Crop Pests. Vol. 6, Eriophyid Mites: Their Biology, Natural Enemies and Control*. Edited by Lindquist EE, Sabelis MW, Bruin J. Amsterdam: Elsevier Science Publishing; 1996:3-31.
22. Staples R, Allington WB: **Streak mosaic of wheat in Nebraska and its control.** *University of Nebraska College of Agriculture, Agricultural Experiment Station. Res Bull* 1956, **178**:40.
23. Oldfield GN: **Toxemias and other non-distorting feeding effects.** In *World Crop Pests. Vol. 6, Eriophyid Mites: Their Biology, Natural Enemies and Control*. Edited by Lindquist EE, Sabelis MW, Bruin J. Amsterdam: Elsevier Science Publishing; 1996:243-250.
24. Orlob GB: **Feeding and transmission characteristics of *Aceria tulipae* Keifer as vector of Wheat streak mosaic virus.** *Phytopathol Z* 1966, **55**:218-238.
25. Carew M, Schiffer M, Umina P, Weeks A, Hoffmann A: **Molecular markers indicate that the wheat curl mite, *Aceria tosichella* Keifer, may represent a species complex in Australia.** *Bull Entomol Res* 2009, **99**:479-486.
26. Hein GL, French R, Siriwetwivat B, Armine JW: **Genetic characterization of North American populations of the wheat curl mite and dry bulb mite.** *J Econ Entomol* 2012, **105**:1801-1808.
27. Siriwetwivat B: **Interactions between the wheat curl mite *Aceria tosichella* Keifer (Eriophyidae), and wheat streak mosaic virus and distribution of wheat curl mite biotypes in the field.** . (Ph.D. dissertation) Lincoln: University of Nebraska; 2006.
28. Skoracka A, Kuczyński L, Rector B, Amrine JW: **Wheat curl mite and dry bulb mite: untangling a taxonomic conundrum through a multidisciplinary approach.** *Biol J Linnean Soc* 2014, **111**:421-436.
29. Skoracka A, Kuczynski L, de Mendonca RS, Dabert M, Szydio W, Knihinicki D, Truol G, Navia D: **Cryptic species within the wheat curl mite *Aceria tosichella* (Keifer) (Avari: Eriophyoidea), revealed by mitochondrial, nuclear and morphometric data.** *Invertebr Syst* 2012, **26**:417-433.
30. Szydio W, Hein GL, Denizhan E, Skoracka A: **Exceptionally high levels of genetic diversity in wheat curl mite (*Aceria tosichella*) populations from Turkey.** *J Econ Entomol* 2015, **108**:2030-2039.
31. Seifers DL, Harvey TL, Louie R, Gordon DT, Martin TJ: **Differential transmission of isolates of the High Plains virus by different sources of wheat curl mites.** *Plant Dis* 2002, **86**:138-142.
32. Wosula EN, McMechan AJ, Oliveira-Hofman C, Wegulo SN, Hein GL: **Differential transmission of two isolates of Wheat streak mosaic virus by five wheat curl mite populations.** *Plant Dis* 2016, **100**:154-158.
33. Schiffer M, Umina P, Carew M, Hoffmann A, Rodoni B, Miller A: **The distribution of wheat curl mite (*Aceria tosichella*) lineages in Australia and their potential to transmit wheat streak mosaic virus.** *Ann Appl Biol* 2009, **155**:371-379.
34. Murugan M, Cardona PS, Duraimurugan P, Whitfield AE, Schneweis D, Starkey S, Smith CM: **Wheat curl mite resistance: interactions of mite feeding with Wheat streak mosaic virus infection.** *J Econ Entomol* 2011, **104**:1406-1414.
35. Byamukama E, Seifers DL, Hein GL, De Wolf E, Tisserat NA, Langham MAC, Osborne LE, Timmerman A, Wegulo SN: **Occurrence and distribution of Triticum mosaic virus in the central Great Plains.** *Plant Dis* 2013, **97**:21-29.
36. Stenger DC, Hall JS, Choi IR, French R: **Phylogenetic relationships within the family Potyviridae: wheat streak mosaic virus and brome streak mosaic virus are not members of the genus Rymovirus.** *Phytopathology* 1998, **88**:782-787.
37. Young BA, Stenger DC, Qu F, Morris TJ, Tatineni S, French R: **Tritimovirus P1 functions as a suppressor of RNA silencing and an enhancer of disease symptoms.** *Virus Res* 2012, **163**:672-677.

38. Slykhuis JT: **Virus diseases of cereals.** *Rev Appl Mycol* 1967, **46**:4011-4429.
 39. Choi IR, French R, Hein GL, Stenger DC: **Fully biologically active *in vitro* transcripts of the eriophyid mite-transmitted wheat streak mosaic tritimovirus.** *Phytopathology* 1999, **89**:1182-1185.
 40. Choi IR, Stenger DC, French R: **Multiple interactions among proteins encoded by the mite-transmitted wheat streak mosaic tritimovirus.** *Virology* 2000, **267**:185-198.
 41. Choi IR, Horken KM, Stenger DC, French R: **An internal RNA element in the P3 cistron of Wheat streak mosaic virus revealed by synonymous mutations that affect both movement and replication.** *J Gen Virol* 2005, **86**:2605-2614.
 42. Stenger DC, French R, Gildow FE: **Complete deletion of Wheat streak mosaic virus HC-Pro: a null mutant is viable for systemic infection.** *J Virol* 2005, **79**:12077-12080.
 43. Stenger DC, Hein GL, Gildow FE, Horken KM, French R: **Plant virus HC-Pro is a determinant of eriophyid mite transmission.** *J Virol* 2005, **79**:9054-9061.
- This paper describes the HC-Pro cistron of WSMV as a determinant of wheat curl mite transmission.
44. Stenger DC, Hein GL, French R: **Nested deletion analysis of Wheat streak mosaic virus HC-Pro: mapping of domains affecting polyprotein processing and eriophyid mite transmission.** *Virology* 2006, **350**:465-474.
 45. Tatineni S, Van Winkle DH, French R: **The N-terminal region of Wheat streak mosaic virus coat protein is a host- and strain-specific long-distance transport factor.** *J Virol* 2011, **85**:1718-1731.
 46. Anandalakshmi R, Pruss GJ, Ge X, Marathe R, Mallory AC, Smith TH, Vance VB: **A viral suppressor of gene silencing in plants.** *Proc Natl Acad Sci U S A* 1998, **95**:13079-13084.
 47. Atreya CD, Pirone TP: **Mutational analysis of the helper component-proteinase gene of a potyvirus: effects of amino acid substitutions, deletions, and gene replacement on virulence and aphid transmissibility.** *Proc Natl Acad Sci U S A* 1993, **90**:11919-11923.
 48. Cronin S, Verchot J, Haldeman-Cahill R, Schaad M, Carrington JC: **Long-distance movement factor: a transport function of the potyvirus helper component proteinase.** *Plant Cell* 1995, **7**:549-559.
 49. Kasschau KD, Carrington JC: **A counterdefensive strategy of plant viruses: suppression of posttranscriptional gene silencing.** *Cell* 1998, **95**:461-470.
 50. Young BA, Hein GL, French R, Stenger DC: **Substitution of conserved cysteine residues in wheat streak mosaic virus HC-Pro abolishes virus transmission by the wheat curl mite.** *Arch Virol* 2007, **152**:2107-2111.
 51. Tatineni S, Kovacs F, French R: **Wheat streak mosaic virus infects systemically despite extensive coat protein deletions: identification of virion assembly and cell-to-cell movement determinants.** *J Virol* 2014, **88**:1366-1380.
 52. Tatineni S, French RC: **The C-terminus of Wheat streak mosaic virus coat protein is involved in differential infection of wheat and maize through host-specific long-distance transport.** *Mol Plant-Microbe Interact* 2014, **27**:150-162.
 53. Tatineni S, Elowsky C, Graybosch RA: **Wheat streak mosaic virus with deletions in the coat protein elicited more severe symptoms than the wild-type virus in multiple cereal hosts.** *Mol Plant-Microbe Interact* 2017, **30**:974-983.
 54. Tatineni S, McMechan AJ, Hein GL: **Wheat streak mosaic virus coat protein is a determinant for vector transmission by the wheat curl mite.** *Virology* 2018, **514**:42-49.
- This paper describes the CP cistron as a determinant of WSMV transmission by the wheat curl mite.
55. Atreya PL, Atreya CD, Pirone TP: **Amino acid substitutions in the coat protein result in loss of insect transmissibility of a plant virus.** *Proc Natl Acad Sci U S A* 1991, **88**:7887-7891.
 56. Peng YH, Kadoury D, Gal-On A, Huet H, Wang Y, Raccach B: **Mutations in the HC-Pro gene of Zucchini yellow mosaic potyvirus: effects on aphid transmission and binding to purified virions.** *J Gen Virol* 1998, **79**:897-904.
 57. Pirone TP, Blanc S: **Helper-dependent vector transmission of plant viruses.** *Annu Rev Phytopathol* 1996, **34**:227-247.
 58. Atreya CD, Atreya PL, Thornbury DW, Pirone TP: **Site-directed mutations in the potyvirus HC-Pro gene affect helper component activity, virus accumulation, and symptom expression in infected tobacco plants.** *Virology* 1992, **191**:106-111.
 59. Blanc S, Ammar ED, Garcia-Lampasona S, Dolja VV, Llave C, Baker J, Pirone TP: **Mutations in the potyvirus helper component protein: effects on interactions with virions and aphid stylets.** *J Gen Virol* 1998, **79**:3119-3122.
 60. Granier F, Durand-Tardif M, Casse-Delbart F, Lecoq H, Robaglia C: **Mutations in Zucchini yellow mosaic virus helper component protein associated with loss of aphid transmissibility.** *J Gen Virol* 1993, **74**:2737-2742.
 61. Huet H, Gal-On A, Meir E, Lecoq H, Raccach B: **Mutations in the helper component protease gene of Zucchini yellow mosaic virus affect its ability to mediate aphid transmissibility.** *J Gen Virol* 1994, **75**:1407-1414.
 62. Paliwal YC: **Relationship of Wheat streak mosaic and Barley stripe mosaic viruses to vector and nonvector eriophyid mites.** *Arch Virol* 1980, **63**:123-132.
 63. Takahashi Y, Orlob GB: **Distribution of wheat streak mosaic virus-like particles in *Aceria tulipae*.** *Virology* 1969, **38**:230-240.
- This paper describes localization of WSMV particles in wheat curl mites.
64. Paliwal YC, Slykhuis JT: **Localization of Wheat streak mosaic virus in the alimentary canal of its vector *Aceria tulipae* Keifer.** *Virology* 1967, **32**:344-353.
- This paper describes localization of WSMV particles in the digestive system of wheat curl mites.
65. Sylvester ES: **Virus transmission by aphids — a viewpoint.** In *Viruses, Vectors, and Vegetation*, vol. 1. Edited by Maramorosch K. New York: Interscience; 1969:59-73.
 66. Watson MA, Plumb RT: **Transmission of plant-pathogenic viruses by aphids.** *Annu Rev Entomol* 1972, **17**:425-452.
 67. Hunger RM, Sherwood JL, Evans CK, Montana JR: **Effects of planting date and inoculation date on severity of wheat streak mosaic virus in hard red winter wheat cultivars.** *Plant Dis* 1992, **76**:1056-1060.
 68. McMechan AJ, Hein GL: **Planting and variety selection for management of viruses transmitted by the wheat curl mite (*Aceria Eriophyidae*).** *J Econ Entomol* 2016, **109**:70-77.
 69. Harvey TL, Martin TJ, Seifers DL: **Resistance to the wheat curl mite (*Aceria Eriophyidae*) prevents loss in wheat yields.** *J Agric Urban Entomol* 2003, **20**:7-10.
 70. Aguirre-Rojas LM, Khalaf LK, Garcés-Carrera S, Sinha DK, Chuang WP, Smith CM: **Resistance to wheat curl mite in Arthropod-resistant Rye-wheat translocation lines.** *Agronomy* 2017, **7**:74.
 71. Carrera SG, Davis H, Aguirre-Rojas L, Murugan M, Smith CM: **Multiple categories of resistance to wheat curl mite (*Aceria Eriophyidae*) expressed in accession of *Aegilops tauschii*.** *J Econ Entomol* 2012, **105**:2180-2186.
 72. Chuang WP, Rojas LMA, Khalaf LK, Zhang GR, Fritz AK, Whitfield AE, Smith CM: **Wheat genotypes with combined resistance to wheat curl mites, Wheat streak mosaic virus, wheat mosaic virus, and Triticum mosaic virus.** *J Econ Entomol* 2017, **110**:711-718.
 73. Conner RL, Thomas JB, Whelan EDP: **Comparison of mite resistance for control of wheat streak mosaic.** *Crop Sci* 1991, **31**:315-318.
 74. Dhakal S, Tan CT, Paezold L, Fuentealba MP, Rudd JC, Blaser BC, Xue QW, Ruch CM, Devkota RN, Liu SY: **Wheat curl mite resistance in hard winter wheat in the US Great Plains.** *Crop Sci* 2017, **57**:53-61.

75. Thomas JB, Conner RL, Graf RJ: **Comparison of different sources of vector resistance for controlling Wheat streak mosaic in winter wheat.** *Crop Sci* 2004, **44**:250130.
76. Harvey TL, Martin TJ, Seifers DL, Sloderbeck PE: **Change in virulence of wheat curl mite detected on TAM 107.** *Crop Sci* 1997, **37**:624-625.
77. Graybosch RA, Peterson CJ, Baenziger PS, Baltensperger DD, Nelson LA, Jin Y, Kolmer J, Seaborn B, French R, Hein G, Martin TJ, Beecher B, Schwarzacher T, Heslop-Harrison P: **Registration of 'Mace' hard red winter wheat.** *J Plant Regist* 2009, **3**:51-56.
78. Seifers DL, Martin TJ, Harvey TL, Gill BS: **Temperature-sensitivity and efficacy of Wheat streak mosaic virus resistance derived from *Agropyron intermedium*.** *Plant Dis* 1995, **79**:1104-1106.
79. Friebe B, Mukai Y, Dhaliwal HS, Martin TJ, Gill BS: **Identification of alien chromatin specifying resistance to wheat streak mosaic and green bug in wheat germplasm by C-banding and in situ hybridization.** *Theoretical and Applied Genetics* 1991, **81**:381-389.
80. Haley SD, Martin TJ, Quick JS, Seifers DL, Stromberger JA, Clayshulte SR, Clifford BL, Peairs FB, Rudolph JB, Johnson JJ, Gill BS, Friebe B: **Registration of CO960293-2 wheat germplasm resistant to Wheat streak mosaic virus and Russian wheat aphid.** *Crop Sci* 2002, **42**:1381-1382.
81. Haley SD, Johnson JJ, Peairs FB, Stromberger JA, Heaton EE, Seifert SA, Kottke RA, Rudolph JB, Martin TJ, Bai G, Chen X, Bowden RL, Jin Y, Kolmer JA, Seifers DL, Chen M, Seabourn BW: **Registration of 'Snowmass' wheat.** *J Plant Regist* 2011, **5**:87-90.
82. Seifers DL, Martin TJ, Harvey TL, Haber S, Haley SD: **Temperature sensitivity and efficacy of Wheat streak mosaic virus resistance derived from CO960293 wheat.** *Plant Dis* 2006, **90**:623-628.