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Research Article

Inheritance of Wheat Streak Mosaic Virus Resistance in KS03HW12

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Abstract

Wheat streak mosaic caused by *Wheat Streak Mosaic Virus* (WSMV) is a serious disease in wheat (*Triticum aestivum* L.). Use of cultivars with the WSMV resistance is a primary and effective way to control this disease. 'KS03HW12' is a newly discovered wheat line with the WSMV resistance. This study was conducted to determine the inheritance of WSMV resistance in KS03HW12 and its allelic relationship with a known resistance gene *Wsm2*. KS03HW12 was crossed to a susceptible line 'KS04HW87' and a resistant cultivar 'RonL' having the *Wsm2* to obtain F_1 and F_2. Seedlings of parents, susceptible check, F_1 and F_2 were artificially inoculated and evaluated for WSMV reaction at 18°C in a growth chamber. A total of 144 $F_{2:3}$ family lines from the cross of KS03HW12 × KS04HW87 were further evaluated for WSMV reaction. Data from the cross of KS03HW12 x KS04HW87 indicated a two-gene with recessive epistasis model for the WSMV resistance in KS03HW12. The F_2 segregation ratio in the cross of KS03HW12 × RonL suggested there are different resistance genes between KS03HW12 and RonL. Therefore, resistance genes in KS03HW12 and RonL could be stacked to produce durable resistance.

Keywords: Wheat; *Wheat streak mosaic virus*; Inheritance; Epistasis; Allelism

Abbreviations

WSMV: *Wheat Streak Mosaic Virus*; WCM: Wheat Curl Mite

Introduction

Wheat Streak Mosaic Virus (WSMV: *Tritimovirus, Potyviridae*), transmitted by Wheat Curl Mite (WCM: *Aceria tosichella* Kiefer), is a destructive virus in wheat (*Triticum aestivum* L.). The WSMV causes sporadic epidemic in many regions around the world including USA, Canada, Europe, Russia, and Australia [1,2]. In the USA, WSMV has become common in the Great Plains [1]. The WSMV infected plants typically show symptoms such as yellow streaked leaves, stunted growth, reduced tillering, partially filled heads, and eventually considerable yield and quality reduction. The yield losses caused by naturally infected WSMV in Kansas have been reported as high as 13% with an average of 2% annually [3]. A WSMV epidemics occurred in Alberta in 1964 resulted in a yield reduction of 18% [4]. In artificially inoculated trials, Rahman et al. [5] reported a range of 21 to 70% in yield reduction due to WSMV infection among 38 winter/spring wheat varieties while Sharp et al. [1] showed yield losses of 41 to 74% among nine locally adapted cultivars.

There are no effective chemicals available for controlling WSMV and its vector. Host resistance is a primary and effective way to control the WSMV disease in wheat. Using wheat cultivars with vector resistance is one possible method to reduce the yield loss caused by the WSMV [6-8]. However, the ability of WCM to quickly evolve from avirulence to virulence on a resistant cultivars makes this approach less cost effective [9-11]. An alternative way to control this disease is to enhance the host resistance to the virus itself. Since WSMV was first reported in 1920s [12], great efforts have been made to find resistance sources. High level of WSMV resistance was first identified in a wild relative, *Thinopyrum intermedium* (Host) Bark worth and Dewey [*Agropyron intermedium* (Host) P. Beauv.] [13-16]. the resistance gene from *Thin.intermedium*, designated as *Wsm1*, has been introduced into cultivated wheat through translocation [17] and it has showed its effectiveness in reducing yield loss against the WSMV inoculation under field conditions [1]. However, yield penalty was observed from 11 to 28% among lines introgressed with *Wsm1*, indicating a linkage drag from the alien translocation [1]. Most recently, another resistance gene *Wsm3* from *Th. Intermedium* was identified and translocated into conventional wheat [18]. Additionally, Fahim et al. [2] also discovered WSMV resistance in another wild species *Th. scripeum*. The WSMV resistance sources in cultivated wheat were found much later than the ones in wild species. Breeding line 'CO960293-2' was the first conventional wheat line with the WSMV resistance found in the USA [19]. Later on, two more resistant breeding lines, 'KS03HW12' [20] and 'CO960333' [21], were identified. Additional conventional wheat germplasm with the WSMV resistance has also been reported [2,19,22].

All the WSMV resistance sources found so far are temperature sensitive. Most resistance sources in the conventional wheat, including CO960293-2 and KS03HW12, can resist WSMV infections at 18°C and permit a systemic infection when exposed to a higher temperature for a certain period of time [18-20]. The resistance of *Wsm1* and *Wsm3* from the wild species can tolerate a higher temperature at 20°C and 24°C, respectively [16,20]. Field tests have showed that the WSMV resistance at 18°C in CO960293-2 and KS03HW12 were effective in protecting against yield losses caused by WSMV inoculation [18,23]. The WSMV resistance in CO960293-2 is reportedly controlled by a single dominant gene, which has been mapped to chromosome 3B

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Table 1: Wheat streak mosaic virus ratings and segregation ratio in F2 from the cross of KS03HW12 x KS04HW87.

Trial	Susceptible check (T81), Parents and Progenies	No. of plants in rating scales			Expected ratio (1:2:3)	χ^2	*P*
		1	2	3			
I	T81			11			
	KS03HW12	21					
	KS04HW87		2	18			
	F_1	2					
	F_2	112	43	39	9:3:4	3.14	0.208
II	T81			10			
	KS03HW12	19					
	KS04HW87			19			
	F_2	80	29	54	9:3:4	5.87	0.053
Combined	F_2	192	72	93	9:3:4	0.92	0.629

and designated as *Wsm2* [24]. CO960293-2 has been widely utilized in breeding programs and its WSMV resistance has been introgressed into two cultivars 'RonL' (PI 648020) and 'Snowmass' (PI 658597) [25]. However, with the deployment of WSMV-resistant cultivars, the limited resistance sources in these cultivars might be broken down by the selection pressure on the virus. Therefore, it is important to discover new resistance genes, which can be stacked with the existing resistance genes to make the resistance more durable.

The WSMV resistance source KS03HW12 was developed by the Agricultural Research Center-Hays at the Kansas State University and it was derived from a three-way cross of 'KS97HW29'/'KS97HW131'//'KS96HW100-5' [23]. If KS03HW12 processes resistance gene(s) other than *Wsm1* and *Wsm2*, it could be a valuable and additional resistance source for breeding programs. It has been known that KS03HW12 did not have the *Wsm1* based on the molecular marker analysis [23]. KS03HW12 has similar WSMV resistance as CO960293-2, but they do not share any common ancestors in their pedigrees according to our records. Therefore, it might be possible that KS03HW12 has a different resistance gene than *Wsm2*. However, the allelic relationship between the resistance gene(s) in KS03HW12 and *Wsm2* has not been explored yet. It is very critical for us to define their allelic relationship in order to decide whether it is useful to pyramid these two resistance resources in breeding programs. Additionally, little is known about the genetic basis of the WSMV resistance in KS03HW12, which could hinder its utilization. By knowing its genetic control, breeding programs could choose appropriate methods to incorporate this resistance into superior cultivars more efficiently. Therefore, the objective of this project was to determine: (i) the inheritance of the WSMV resistance in KS03HW12; and (ii) the allelic relationship between the resistance gene(s) in KS03HW12 and *Wsm2*.

Materials and Methods

KS03HW12 was crossed with 'RonL' and 'KS04HW87' to obtain F_1. KS04HW87 is a WSMV-susceptible wheat breeding line and it was developed by the Agricultural Research Center-Hays at the Kansas State University. Some F_1 was selfed to obtain F_2. F_2 together with parental lines, susceptible check ('T81'), or F_1 were seeded in 30 × 50 cm metal flats filled with potting mix for the WSMV evaluation. Each flat had 22 rows and 12 seeds were planted in each row. Parental lines, susceptible check, and F_1 each were seeded in one row. The evaluation trial for the cross of KS03HW12 × KS04HW87 was conducted two times. In each trial, parental lines were planted in two replications. Due to limited number of seeds, F_1 was only planted in the first trial. After evaluation in the first trial, F_2 plants were vernalized and then transplanted into pots for generation advancement. A total of 144 $F_{2:3}$ family lines were obtained and they were seeded together with T81 and three replications of parental lines in flats for a WSMV confirmation test. Each $F_{2:3}$ family line or parental line was seeded in one row. For the cross of KS03HW12 ×RonL, parental lines, T81, and F_2 were seeded in one flat. Plants in the flats were grown in growth chambers (Percival Model PGC-15WC) under 12 h photoperiod at 18°C.Plants at the single leaf stage were mechanically inoculated with Sidney 81 isolate as described by Seifers et al. [20]. The inoculated plants were rated for symptoms three weeks after inoculation on a scale of 1 to 5 (1: no visual symptoms, 2: a few chlorotic streaks, 3: moderate mosaic, 4: severe mosaic, 5: severe mosaic, necrosis, and yellowing). The segregation ratio of WSMV rating in the F2 populations was tested by chi-square for goodness of fit. The trials for the cross of KS03HW12 × KS04HW87 were used to determine the inheritance of the WSMV resistance in KS03HW12; and the trial for the cross of KS03HW12 × RonL was used to determine the allelic relationship between the resistance gene(s) in KS03HW12 and *Wsm2*.

Results

The WSM rating for the cross of KS03HW12 × KS04HW87 is presented in Table 1. In the first evaluation trial, all 11 plants of susceptible check T81 was rated as 3 while the rating score for susceptible parentKS04HW87 was similar as T81 with 3 for all 10 plants in its first replication and 2 for two plants and 3 for eight other plants in its second replication. Both replications of KS03HW12 had no symptoms (rated as 1). Only two F_1 plants were available and they had no symptoms as KS03HW12, indicating that WSMV resistance in KS03HW12 is dominant. The F_2 plants were scored in a range of 1 to 3. The observed ratio among these three rating scores (1:2:3) fitted a 9:3:4 ratio (P=0.208), suggesting two genes with recessive epistasis conditioning the WSMV resistance in KS03HW12. To confirm the

Table 2: Segregation of resistance to Wheat streak mosaic virus in 144 $F_{2:3}$ families from the cross of KS03HW12 × KS04HW87.

$F_{2:3}$ family lines	F_2 genotype*	Observed	Expected
Resistant lines	AABB	11	9
Susceptible lines	aa__	11	36
Heterogeneous lines	Other 11 possible genotypes	122	99
Total		144	144

* aa has theepistatic effect.

Table 3: Wheat streak mosaic virus ratings and segregation ratio in F_2 from the cross of KS03HW12 × RonL.

Susceptible check (T81), Parents and Progenies	No. of plants in rating scales			Expected ratio (1:2+3)	χ^2	*P*
	1	2	3			
T81			10			
KS03HW12	19					
RonL	19					
F_2	171	17	2	15:1	4.56	0.033

segregation ratio, this F_2 population was seeded, inoculated, and evaluated for a second time. In the second evaluation trial, all plants of susceptible check T81 and susceptible parent KS04HW87 were rated as 3 while all plants of KS03HW12 were rated as 1. The observed segregation ratio (1:2:3) in the F_2 population still fitted that 9:3:4 ratio (*P*=0.053). If combining these two sets of data, the segregation ratio fitted the 9:3:4 ratios very well with a probability of 0.629. Therefore, the result from the second evaluation trial further supported a two-gene with recessive epistasis model for the WSMV resistance in KS03HW12.

In the confirmation test trial, the susceptible check (T81) and all three replications of the susceptible parent KS04HW87 were rated as 3 and all plants in the three replications of KS03HW12 had no symptoms except one plant with a score of 2 (data not shown). Thus, those family lines with only one plant scored as 2 or no diseased plants were classified as resistant lines while family lines with all susceptible plants (score of 3) were classified as susceptible. Among the 144 family lines, there were 11 resistant lines, 11 susceptible lines, and 122 heterogeneous lines (Table 2). Based on the two-gene with recessive epistasis model, 9 resistant lines (derived from F_2 plants with two homozygous dominant genes) and 36 susceptible lines (derived from F_2 plants having the homozygous recessive gene with the epistatic effect) were expected among the 144 $F_{2:3}$ family lines. The number of resistant lines observed is very close to the expected, a further demonstration of two genes controlling the WSMV resistance in KS03HW12. However, there were much less observed susceptible lines than expected. This skewness might be due to the fact that over 40% of the F_2 susceptible plants (rated as 3) in this trial did not make to the F_3 generation after their transplanting.

To study the allelic relationship with *Wsm2*, RonL was crossed with KS03HW12 since RonL has the *Wsm2* that was inherited from CO960293-2. The F_2 plants derived from the cross of KS03HW12 ×RonL were then evaluated for the WSMV reaction. All plants of T81 were susceptible with a score of 3 while all plants of KS03HW12 and RonL had no symptoms (Table 3). Most of the F_2 plants were resistant to WSMV while a total of 19 F_2 plants were rated as 2 or 3. The segregation ratio (resistant: susceptible) in this F_2 population fits a 15:1 ratio (P = 0.033), suggesting that one resistant gene in KS03HW12 might be different from the one in RonL.

Discussion

The WSMV can cause severe mosaic, yellowing, and even death if given considerable time (mostly four weeks after infection) for its development. In general, WSMV produces moderate mosaic symptom in susceptible genotypes and no visual symptom in resistant genotypes at three weeks after inoculation. But, it is possible that a few resistant plants could show minor symptom at three weeks or less after inoculation. In a previous study, Seifers et al. [20] found small percentages of plants (4, 5, and 4%) in three resistant genotypes showing systemic symptom at three weeks after inoculation. In our confirmation test, one plant of KS03HW12 was also found a few streaks and was scored as 2. These individual resistant plants might have had some favorable conditions for virus multiplication and thus showed symptoms. Our other trials also showed that more resistant plants could have minor symptoms if evaluated at four weeks after inoculation.

Several other studies have determined that the WSMV resistance in a few wheat lines was each controlled by a single dominant gene [24,26]. However, our study showed a two-gene model for KS03HW12. This discrepancy could be due to the different resistant line we analyzed or due to the way how to classify resistant and susceptible groups. In the earlier study conducted by Lu et al. [24], plants with a few streaks were also classified as resistant. However, it is not clear why they were classified in that way since there was no information in that study showing that the resistant parent has plants with a few streaks. In our study, the ratio could also fit one gene model if we classified the plants with a few streaks as resistance (data not shown). But, it does not seem like that they are the resistant parent type since we had a higher percentage of plants with a few streaks than expected. Therefore, we classified these lines in a different group than resistant and susceptible ones. According to the segregation pattern in our study, a model of two genes with an epistatic effect was determined for the WSMV resistance in KS03HW12. Hassani and Assad [27] had similar findings about epistatic effects and minor genes in controlling WSMV in two resistant lines besides a major gene.

A marker closely linked to *Wsm2* has been developed and was tested on the first F_2 population used in this study (data not shown). The marker explained about 50% of the phenotypic variation (data not shown) indicating that one resistance gene in KS03HW12 might be *Wsm2*. It has been known that RonL inherited *Wsm2* from CO960293-2. In our allelic test, the segregation ratio should fit a 3:1 ratio if RonL has only *Wsm2* and KS03HW12 has two genes including *Wsm2*. However, our allelic test showed a two-gene segregation ratio, indicating that RonL might have a second resistance gene and it is different from the ones in KS03HW12. In the genetic study for CO960293-2, Lu et al. [24] used a moderate susceptible parent to cross with CO960293-2. It might be possible that the moderate susceptible parent they used has a minor resistance gene, which is the same as the second resistance gene in CO960293-2 besides *Wsm2*. Thus, Lu et al. [24] only found *Wsm2* in their study. However, further studies need to be conducted to discover the second genes in KS03HW12 and RonL.

Conclusion

The WSMV resistance in KS03HW12 was found to be governed by two genes with a recessive epistatic effect. One resistance gene in KS03HW12 might be *Wsm2*. Based on our allelic test; RonL might also have a minor gene besides *Wsm2*, which is not allelic to the one in KS03HW12. Therefore, it is possible to gain broader or stronger resistance through pyramiding these two resistance sources.

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References

1. Sharp GL, Martin JM, Lanning SP, Blake NK, Brey CW, Sivamani E, et al. Field Evaluation of Transgenic and Classical Sources of Wheat streak mosaic virus Resistance. Crop Sci. 2002; 42: 105-110.
2. Fahim M, Mechanicos A, Ayala-Navarrete L, Haber S, Larkin PJ. Resistance to wheat streak mosaic virus-a survey of resources and development of molecular markers. Plant Pathol. 2011; 61: 425-440.
3. Sim T, Willis WG, Eversmeyer MG. Kansas plant disease survey. Plant Dis. 1988; 72:832-836.
4. Atkinson TG, Grant MN. An evaluation of streak mosaic losses in winter wheat. Phytopathology. 1967; 57: 188-192.
5. Rahman F, Ross JG, Gardner WS. Tolerance to wheat streak mosaic virus in spring and winter wheat cultivars. Crop Sci. 1974; 14: 178-180.
6. Martin TJ, Harvey TL, Bender CG, Seifers DL. Control of wheat streak mosaic virus with vector resistance in wheat. Phytopathology. 1984; 74: 963-964.
7. Conner RL, Thomas JB, Whelan EDP. Comparison of mite resistance for control of wheat streak mosaic. Crop Sci. 1991; 31: 315-318.
8. 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: 125-130.
9. Harvey TL, Martin TJ, Seifers DL. Survival of five wheat curl mite Aceriatosichilla Keifer (Acari: Eriophyidae) strains on mite resistant wheat. Exp and ApplA carol. 1995a; 19: 459-463.
10. Harvey TL, Martin TJ, Seifers DL, Sloderbeck PE. Adaptation of a wheat curl mite (Acari: Eriophyidae) to resistant wheat in Kansas. J Agric Entomol. 1995b; 12: 119-125.
11. Harvey TL, Martin TJ, Seifers DL, Sloderbeck PE. Change in virulence of wheat curl mite detected on TAM 107 wheat. Crop Sci. 1997; 37: 624-625.
12. Mckinney HH. Mosaic diseases of wheat and related cereals. US Department of Agriculture Circular number. 1937; 442: 1-23.
13. Larson R, Atkinson T. Identity of the wheat chromosome replaced by Agropyron chromosomes in a triple alien chromosome substitution line immune to wheat streak mosaic. Canadian J. Genetics and Cytology.1970; 12: 145-150.
14. Wells DG, Wong R, Chung Sze, Lay CL, Gardner WAS, Finney KF. Registration of C.I. 15092 and C.I. 15093 wheat germplasm. Crop Sci. 1973; 13: 776.
15. Wells DG, Kota RS, Sandhu HS, Gardner WS, Finney KF. Registration of one disomic substitution line and five translocation lines of winter wheat germ plasm resistant to wheat streak mosaic virus. Crop Sci. 1982; 22:1277-1278.
16. Triebe B, Mukai Y, Dhaliwal HS, Martin TJ, Gill BS. Identification of alien chromatin specifying resistance to wheat streak mosaic and greenbug in wheat germ plasm by C-banding and in situ hybridization. Theor Appl Genet. 1991; 81: 381-389.
17. Gill BS, Friebe B, Wilson DL, Cox TS. Registration of KS93WGRC27 wheat streak mosaic virus resistant T4DL4Ai#2S wheat germplasm. Crop Sci. 1995; 35: 1236.
18. Friebe B, Liu W, Qi LL, Wilson DL, Raupp WJ, Poland J, et al. Notice of release of KS12WGGRC59 wheat streak mosaic virus-and Triticum mosaic virus- resistance wheat germplasm. Annual Wheat Newsletter.2011; 57:280.
19. Haley SD, Martin TJ, Quick JS, Seifers DL, Stromberger JA, Gill BS, et al. Registration of C0960293-2 wheat germplasm resistant to Wheat streak mosaic virus and Russian wheat aphid. Crop Sci. 2002; 42:1381-1382.
20. Seifers DL, Haber S, Martin TJ, Zhang G. New sources of temperature sensitive resistance to wheat streak mosaic virus in wheat. Plant Dis. 2013a; 97: 1051-1056.
21. Seifers DL, Martin TJ, Haber S. Temperature sensitive resistance to wheat streak mosaic virus in CO960333 and KS06HW79 wheat. Plant Dis. 2013b; 97: 983-987.
22. Masumi M, Kamran R, Shirvan A, Izadpanah K. Reaction of wheat genotypes to wheat streak mosaic virus in Iran. Iran J Plant Path. 1999; 35: 9-18.
23. Seifers DL, Martin TJ, Harvey TL, Haber S. Temperature sensitive wheat streak mosaic virus resistance identified in KS03HW12 wheat. Plant Dis. 2007; 91: 1029-1033.
24. Lu HJ, Price J, Devkota R, Rush C, Rudd J. A dominant gene for resistance to wheat streak mosaic virus in winter wheat line CO960293-2. Crop Sci. 2011; 51: 5-12.
25. Haley SD, Johnson JJ, Peairs FB, Stromberger J, Heaton E, Seifert S, et al. Registration of 'Snowmass' wheat. J Plant Reg. 2011; 5: 87-90.
26. Hassani F, Assad MT. Inheritance and allelism of wheat streak mosaic virus resistance in two Iranian wheat lines. Euphytica. 2004a; 140: 213-216.
27. Hassani F, Assad MT. Inheritance of resistance to wheat streak mosaic virus in two bread wheat cultivars. Proceedings of the 4th International Iran & Russia Conference. ShahreKord, Iran. 2004b; 143-147.

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