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Generation means analysis of *wheat streak mosaic* virus resistance in winter wheat

Frederick Hakizimana[1,2], Amir M.H. Ibrahim[1,*], Marie A.C. Langham[1], Jackie C. Rudd[3] & Scott D. Haley[4]

[1]*Plant Science Department, South Dakota State University, Brookings, SD 57007, U.S.A.;* [2]*International Institute of Tropical Agriculture (IITA), Oyo Road, PMB 5320, Ibadan, Nigeria;* [3]*Texas A&M University, 6500 Amarillo Blvd, W. Amarillo, TX 79106, U.S.A.;* [4]*Soil and Crop Sciences Department, Colorado State University, Fort Collins, CO 80523, U.S.A.; (*author for correspondence: e-mail: amir_ibrahim@sdstate.edu)*

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Summary

Wheat streak mosaic virus (WSMV; Family: *potyviridae*; Genus: *Tritimovirus*) is a major threat to winter wheat (*Triticum aestivum* L. em. Thell) production worldwide, yet little is known about the genetic control of resistance. Our objective was to determine the mode of inheritance and type of gene action of WSMV resistance in two winter wheat crosses involving a resistant line, 'OK65C93-8', and two susceptible cultivars, 'Tandem' and 'Vista'. For each cross, parents, F_1, F_2, and backcross plants were inoculated and evaluated for WSMV resistance in two replicated greenhouse experiments. Generation means analysis indicated that additive, dominance, and epistatic effects were all involved in the inheritance of WSMV resistance. Broad-sense heritability estimates for visual symptom rating and ELISA values were high for both crosses (0.84–0.91). Narrow-sense heritability estimates were low in the Tandem/OK65C93-8 cross (0.43–0.45) and moderate in the Vista/OK65C93-8 cross (0.71–0.74). Due to the presence of greater non-additive gene effects combined with low narrow-sense heritability in the Tandem/OK65C93-8 cross, selecting for WSMV resistance in this cross would be complex if using conventional methods. On the other hand, the significant contribution of additive gene effects combined with moderate narrow-sense heritability in the Vista/OK65C93-8 cross suggested that it could be exploited to select for WSMV resistance. Progress from selection for WSMV resistance in early generations of winter wheat may vary among populations as indicated in this study. Therefore, evaluating genetic control of parental combinations may be warranted prior to selecting for WSMV resistance from this source.

Introduction

Wheat streak mosaic virus (WSMV) is an important wheat disease in the US Great Plains, Canada, and many other wheat-producing areas in the world. Wheat yield losses caused by WSMV are estimated at about 2% year^{-1}, but yield losses of up to 100% have been observed (McNeil et al., 1996). In Kansas, WSMV was the most important wheat disease from 1987 to 1991, with average yield losses of 13% (Friebe et al., 1996; Harvey et al., 1997).

Cultural control methods to reduce yield losses caused by WSMV include late planting and destruction of volunteer wheat (Slykhuis, 1955), which is the major over-summering host of WSMV and its vector, the wheat curl mite (*Aceria tosichella*). However, these procedures are not always effective because planting date is often dictated by the availability of soil moisture and volunteer wheat is often used for winter grazing in some states in the Great Plains region of the U.S.A. (Martin et al., 1984). Therefore, host-plant resistance to WSMV

may be the most efficient way to control the disease.

Wheat cultivars with reliable WSMV resistance are currently not available. However, high levels of WSMV resistance have been found in some perennial genera of the Triticeae, including *Secale* and *Elytrigia* (*syn. Agropyron*) species [*E. intermedia* (Host) P. Beauv. (*syn. A. intermedium*) and *E. ponticum* (Host) P. Beauv., (*syn. A. elongatum* and recently *Thinopyrum ponticum*)] (Friebe et al., 1996).

Several studies have been conducted on the inheritance of WSMV resistance using *E. ponticum* as the resistance source, but no general consensus exists on the inheritance of WSMV resistance in *Triticum* × *E. ponticum* hybrids. Some of these studies have indicated that WSMV resistance from *E. ponticum* was recessive at 27 °C. It was incompletely dominant at 22 °C and dominant in field tests (Friebe et al., 1996; Martin, 1978). Other studies have suggested that the WSMV resistance from *E. ponticum* was controlled by multiple genes (Swarup et al., 1956). OK65C93-8 is a winter wheat breeding line with a high level of WSMV resistance from *E. ponticum* developed by irradiation (Sebesta et al., 1995). It was originally released in 1971 as CI 15322 by the Oklahoma Agricultural Experiment Station in cooperation with the USDA-ARS. This line has been used in many winter wheat breeding programs as a source of WSMV resistance. Jiang et al. (1993) showed that OK65C93-8 had one entire *E. ponticum* chromosome, 1Ae-2 that substituted for chromosome 1D. They also showed that the long arm of chromosome 4D had been replaced by the distal part of 1 Ae-1L, which contained the major WSMV resistance gene(s) found in this line. Little is known, however, about the mechanisms controlling resistance in this line. Therefore, generation means analysis using parents with different reactions to the virus was used to determine the mode of inheritance and the type of gene action of WSMV resistance in the OK65C93-8 winter wheat line.

Materials and methods

Genetic material

Three winter wheat genotypes [OK65C93-8 (resistant), 'Tandem' ('Brule'/'Agate': susceptible), and 'Vista' (NE68513/NE684457//'Centurk'/3/Brule: susceptible)] were chosen as parents for this study (Hakizimana, 2001). The source of WSMV resistance in OK65C93-8 originated from a wheat × *E. ponticum* hybrid line designated P3-19 (Sebesta et al., 1995). The three genotypes were crossed in all combinations during the 1997–1998 and 1998–1999 greenhouse cycles to produce F_1, F_2, reciprocal, and backcross seed.

Experimental design

Two separate experiments were conducted in a greenhouse at South Dakota State University in November 2000 and January 2001. The experimental material consisted of six populations, namely P_1, P_2, F_1, F_2, BC_1P_1, and BC_1P_2. The progeny derived from backcrossing the F_1 to the female parent were designated as BC_1P_1 and those from backcrossing to the male parent as BC_1P_2. Each experimental unit consisted of 5 plants of each parent, 5 plants of each F_1, 25 plants of the F_2 population, 15 plants of BC_1P_1, and 25 plants of BC_1P_2. A total of 20 plants for each parent, 20–40 plants of the F_1, 185–196 plants of the F_2, 50–100 plants of BC_1P_1, and 11–57 plants of BC_1P_2 were evaluated for WSMV resistance. Non-segregating populations (parents and F_1's) were represented by fewer plants, whereas segregating populations (F_2's and backcross) were represented by more plants to compensate for the greater variability in error variance usually associated with segregating populations versus non-segregating populations (Hallauer and Miranda, 1988).

Plants were grown in the greenhouse in a randomized complete design with two replications in each experiment. The pots were 13 cm in diameter and contained a sieved mixture of topsoil, vermiculite, peat moss, and sand in a 3:1:1:1 ratio. Pots were placed on greenhouse benches with a 16 h photoperiod, a light intensity of 200 μmol m^{-2} s^{-1}, and 20 °C day and 16 °C night temperature.

Inoculum production and plant inoculation

Inoculum was produced by mechanically infecting seedlings of susceptible greenhouse-grown 'Arapahoe' winter wheat at the 2–3 leaf stage with a wheat streak mosaic virus isolate (WSMV-SD) collected in South Dakota. WSMV-SD is serologically reactive with antiserum to several known WSMV isolates and has been well characterized in field studies (M.A.C. Langham, unpublished). A comparison of WSMV-SD with other WSMV isolates is incomplete at this time. Seedling plants of Arapahoe were inoculated 7 days after emergence using an air-blast inoculation technique (Wu and Langham, 1996). Two weeks after inoculation, foliage

was cut approximately 2.5 cm above the soil surface to prepare inoculum. Inoculum was prepared by blending infected Arapahoe wheat in three cycles (15, 10, 5 sec) with 0.02 M potassium phosphate buffer [1:3 tissue (g): buffer (ml) at pH 7.0 in a 1:6 ratio] at high speed in a Waring blender. The resulting extract was filtered through cheesecloth and 1% silica carbide was added (Bottacin and Nassuth, 1990; Wu and Langham, 1996). Plants were inoculated by hand rubbing plants using cheesecloth saturated with the sap extract of the WSMV-SD isolate. Virus-free inoculation buffer with 1% silica carbide was used on control plants (parents).

Disease assessment

Symptoms were recorded at 14 and 21 days following inoculation. Infected plants were rated for WSMV symptoms on a scale of 1–5, where 1 = no visible symptoms to light green flecks; 2 = broken light green and a few yellow streaks; 3 = mixed green and yellow streaks; 4 = yellow streaks; and 5 = severe yellow streaking and necrosis (Wu and Langham, 1996). In both experiments, the reciprocal crosses gave similar WSMV reaction; therefore, they were grouped together for statistical and genetic analyses.

Enzyme-linked Immunosorbant assay (ELISA)

WSMV infected leaves were collected from each plant at the 4–5 leaf stage to confirm the presence of the virus in every plant of the population by the ELISA (enzyme-linked immunosorbant assay: Agdia, Elkhart, IN) method proposed by Clark and Adams (1977).

Crude extracts from WSMV infected and non-infected leaves were prepared with a sap extractor (Erich Pollahne: Hanover, Germany) and were diluted in a 1:10 ratio (v:v) with extraction buffer, which was composed of phosphate buffered saline (PBS) containing 2% polyvinylpyrrolidone (PVP), 2% Tween-20, 0.2% ovalbumin, 0.02% sodium azide, and 0.13% sodium sulphite. This extraction buffer was adjusted to pH 7.4.

After the plates were loaded with the appropriate concentration of sap extract and buffer (100 μl), they were then incubated at 4 °C overnight. Plates were then washed five times by filling each well with PBS-Tween and allowing it to soak for 5 min between rinses. After washing, the plates were drained upside down on paper towels and then PBS with 0.05% Tween, 0.2% bovine serum albumin, 2% PVP, and 0.02% sodium azide containing WSMV antibody conjugate in a 1:100 ratio was added to the wells (100ul/well).

Following the addition of WSMV antiserum conjugated with alkaline phosphatase, plates were incubated for 3 hr, washed with PBS-Tween, and drained again upside down on paper towels. Then, 100 (l of p-nitrophenyl phosphate (PNP) solution (1 mg/ml of PNP) in 9.7% diethanolamine, 0.02% sodium azide and 0.01% magnesium chloride was added to each well. The plates were then allowed to develop at room temperature for 2 hr until the wells containing WSMV-infected sap turned yellow.

Absorbance was measured four times (at 30 min intervals) at 405 nm using an ELISA micro-plate reader (Dynext Technologies, Chantilly, VA). Absorbance values were considered positive if they were twice those of the equivalent control.

Statistical and genetic analyses

Comparisons of the reciprocal crosses did not show any significant difference ($P > 0.05$), suggesting the absence of maternal or cytoplasmic effects for WSMV resistance in the OK65C93-8 winter line. Therefore, the reciprocal crosses were combined for subsequent analyses.

Following confirmation of error variance homogeneity ($X^2 = 1.58$, $P < 0.05$), data were analyzed over experiments (Gomez and Gomez, 1984). Statistical analyses were performed using the Statistical Analysis System (SAS) program (SAS, 1985). Individual scaling tests were computed following the methods of Mather and Jinks (1971). Visual symptom ratings were used to classify plants as either resistant or susceptible. Plants with visual symptom ratings of 1 were considered resistant, while those with a rating higher than 1 were considered susceptible.

The three-parameter model (mean, additive, and dominance effects) was first tested using both visual symptom ratings and ELISA values from the two crosses. We used the individual scaling tests of Ketata et al. (1976) and Mather and Jinks (1971) with $A = 2BC_1P_1 - F_1 - P_1$, $B = 2BC_1P_2 - F_1 - P_2$, and $C = 4F_2 - 2F_1 - P_1 - P_2$ to test the fitness of our data to the additive-dominance model.

Due to the presence of epistatic effects, the generational means analysis proposed by Gamble (1962) was used to estimate genetic parameters using a six-parameter model. Each of the six generations used in this study was expressed in terms of the following effects: m = overall mean; a = pooled additive effect;

d = pooled dominance effect; aa = pooled additive-by-additive; ad = pooled additive-by-dominance; and dd = pooled dominance-by-dominance epistatic effects. The equations used to estimate the genetic parameter effects were the following:

$$P_1 = m + a + aa$$
$$P_2 = m - a + aa$$
$$F_1 = m + d + dd$$
$$F_2 = m + 0.5d + 0.25dd$$
$$BC_1P_1 = m + 0.5a + 0.5d + 0.25aa + 0.25ad + 0.25dd$$
$$BC_1P_2 = m - 0.5a + 0.5d + 0.25aa + 0.25ad + 0.25dd$$

The genetic parameters in this equation and their respective standard errors were estimated using a least squares analysis on the transformed data. The significance of the genetic parameters was estimated based on their corresponding *P*-values obtained from the least squares analysis.

Heritability estimates

Narrow-sense heritability (h^2_{ns}) was estimated following the method proposed by Warner (1952): $h^2_{ns} = [2V_{F2} - (V_{B1} + V_{B2})]/V_{F2}$, where: V_{F2}, V_{B1}, and V_{B2} are the variances of the F_2, $BC_1 1$, and $BC_1 2$ generations. The standard error for the narrow-sense heritability was estimated as described by Ketata et al. (1976). Broad-sense heritability (h^2_{bs}) was estimated as proposed by Burton (1951) and utilizes the F_1 data to estimate the environmental variance: $H^2_{bs} = (V_{F2} - V_{F1})/V_{F2}$, where V_{F1} and V_{F2} are the variances of F_1 and F_2 generations.

Results and discussion

For both WSMV symptom ratings and ELISA values, the effects due to experiments and generations × experiments were not significant (Table 1) in either cross, indicating the absence of environmental variation for these traits. Highly significant differences ($P < 0.01$) were observed among generations in both crosses.

The means, ranges, and variances of the parental lines (P_1, P_2), F_1, F_2, BC_1P_1, and BC_1P_2 of both crosses evaluated for WSMV symptom ratings and ELISA values in two experiments are shown in Table 2. The performance of the parents conformed to expectations. In both crosses, OK65C93-8 was highly resistant (low symptom ratings and ELISA values), while Tandem and Vista were highly susceptible to WSMV infection (high-symptom ratings and ELISA values). The F_1 plants from both crosses were all susceptible (Table 2), indicating recessive inheritance of the gene(s) controlling resistance to WSMV in the OK65C93-8 winter wheat line. The F_2 and backcross plants were either resistant or susceptible with the majority of plants being susceptible, again suggesting recessive inheritance of the gene(s) controlling resistance to WSMV.

Table 1. Mean squares from the generation means analysis for wheat streak mosaic virus symptom ratings and enzyme-linked immunosorbant assay values of Tandem/OK65C93-8 and Vista/OK65C93-8 winter wheat crosses evaluated across seasons

Source	df	Symptom ratings	ELISA values
Tandem/OK65C93-8			
Experiment (Exp)	1	0.12	0.24
Reps within Exp	2	0.09	0.19
Generation (Gen)	5	2.65**	0.99**
Gen × Exp	5	0.13	0.11
Error	401	0.05	0.07
CV%		12.63	23.65
Vista/OK65C93-8			
Experiment (Exp)	1	0.19	0.10
Reps within Exp	2	0.10	0.16
Generation (Gen)	5	2.41*	0.93**
Gen × Exp	5	0.06	0.10
Error	303	0.05	0.04
CV%		13.65	18.05

*,**Significant at the 0.05 and 0.01 probability levels, respectively.

Individual scaling tests (A, B, and C) were used to test the fitness of the three-parameter model (mean, additive, and dominance) in explaining the variability observed among the progeny from both crosses and both traits measured as suggested by Ketata et al. (1976). Based on the individual scaling tests, the model did not fit the data in the Tandem/OK65C93-8 cross for WSMV symptom rating and ELISA values or ELISA values in the Vista/OK65C93-8 cross (Table 3), indicating that epistasis was involved in the inheritance of the two characters. Consequently, the data were transformed according to Mather and Jinks (1971).

Square root transformation resulted again in significance of the scaling tests, suggesting that the three-parameter model was not sufficient to explain the genetic differences for ELISA values and symptom rating

Table 2. Means, ranges, and variances across seasons of WSMV symptom ratings and ELISA values of the parents, F_1, F_2 and backcrosses generations of two resistant/susceptible winter wheat crosses

	Symptom ratings[¶]					ELISA values[†]		
Population	N_1	N_2[§]	Mean	Range	Var.	Mean	Range	Var.
Tandem/OK65C93-8								
OK65C93-8 (P_1)	20	20	1.2	1.0–1.8	0.2	0.7	−0.1–2.3	0.2
Tandem (P_2)	20	20	3.7	3.4–4.0	0.2	2.4	1.6–3.4	0.6
F_1 ($P_2 \times P_1$)	40	39	3.5	1.5–4.0	0.5	2.7	1.8–3.5	0.6
F_2 (F_1 selfed)	196	193	2.9	1.0–4.4	0.8	2.0	0.0–3.5	0.9
BC_1P_1 ($F_1 \times P_1$)	57	40	2.9	1.0–4.6	1.1	2.0	0.0–3.5	0.2
BC_1P_2 ($F_1 \times P_2$)	100	100	3.5	2.4–4.4	0.4	2.0	0.0–3.2	0.8
Vista/OK65C93-8								
OK65C93-8 (P_1)	20	20	1.2	1.0–1.8	0.2	0.7	−0.1–3.4	0.2
Vista (P_2)	20	20	4.1	3.8–4.5	0.2	2.5	1.2–3.4	0.7
F_1 ($P_2 \times P_1$)	20	20	3.5	3.0–4.0	0.3	2.5	1.4–3.4	0.7
F_2 (F_1 selfed)	185	182	3.1	1.0–4.6	0.9	2.1	0.0–3.5	0.9
BC_1P_1 ($F_1 \times P_1$)	11	11	2.6	1.1–4.0	0.9	2.5	0.1–3.5	1.1
BC_1P_2 ($F_1 \times P_2$)	50	41	3.6	1.8–4.4	0.1	2.8	1.3–3.5	0.5

[¶]Symptom ratings on a scale 1–5 scale; 1 = resistant and 5 = necrotic plant.
[†]ELISA values are averages of absorbance readings recorded after 30 min.
N_1 = number of plants evaluated for WSMV symptom ratings in each generation.
[§]N_2 = number of plants evaluated for ELISA values in each generation.

Table 3. Significance of the individual scaling list (A, B, and C) for WSMV symptom ratings and ELISA values in Tandem/OK65C93-8 and Vista/OK65C93-8 winter wheat crosses, using transformed data

		Scaling test		
Trait	Cross	A	B	C
Symptom ratings	Tandem/OK65C93-8	**	**	**
	Vista/OK65C93-8	NS	NS	NS
ELISA Values	Tandem/OK65C93-8	NS	**	**
	Vista/OK65C93-8	*	**	NS

*,** =Significant at the 0.05 and 0.01 probability levels, respectively.
NS = Not significant at $\alpha = 0.05$

in the Tandem/OK65C93-8 cross. This indicated that epistasis was involved in the inheritance of WSMV resistance. Consequently, the six-parameter model (additive, dominance, and epistatic interactions) was used to determine the type and magnitude of gene action involved in the inheritance of WSMV resistance.

The estimates of the genetic effects and their magnitudes are presented in Table 4 for the three- and six-parameter models. The three-parameter model showed that additive and dominance effects were highly significant ($P < 0.01$) for both characters in both crosses, indicating that they contributed significantly to the inheritance of WSMV resistance. In both crosses, additive effects were significant and negative for both characters. Dominance effects were significant and positive.

The six-parameter model revealed that epistatic effects contributed significantly to the genetic variation of WSMV resistance using ELISA values in both crosses. On the other hand, the epistatic effects for symptom rating were significant only in the Tandem/OK65C93-8 cross. The major contribution of the gene effects to the variation was indicated by the relative magnitude of the parameter as related to the overall mean (Gamble, 1962). In the Tandem/OK65C93-8 cross, estimates of the additive genetic effects (fixable effect) were negative and quite small relative to the overall mean and also to dominance variation for both symptom ratings and ELISA values. However, these additive genetic effects were highly significant ($P < 0.01$), indicating that additive genetic variation was a factor in the resistance to WSMV in these crosses. The dominance effects were non-significant for symptom rating in the Vista/OK65C93-8 cross, indicating the contribution of additive gene effects in the inheritance of resistance to WSMV in this cross. The lack of dominance and epistatic effects in this cross indicated that

Table 4. Genetic parameters and standard errors for WSMV symptom ratings and ELISA values estimated based on the three-and six parameter models on means of parents, F_1, F_2, and backcrosses in Tandem/OK65C93-8 and Vista/OK65C93-8 winter wheat crosses

Parameter	Symptom rating	ELISA Values
3-parameter model		
	Tandem/OK65C93-8	
m	$1.52^{**} \pm 0.03$	$0.94^{**} \pm 0.03$
a	$-0.35^{**} \pm 0.03$	$-0.16^{**} \pm 0.03$
d	$0.38^{**} \pm 0.05$	$0.33^{**} \pm 0.06$
	Vista/OK65C93-8	
m	$1.55^{**} \pm 0.03$	$1.01^{**} \pm 0.03$
a	$-0.44^{**} \pm 0.03$	$-0.25^{**} \pm 0.03$
d	$0.32^{**} \pm 0.06$	$0.29^{**} \pm 0.06$
6-parameter model		
	Tandem/OK65C93-8	
m	$1.15^{**} \pm 0.01$	$1.40^{**} \pm 0.12$
a	$-0.43^{**} \pm 0.04$	$-0.26^{**} \pm 0.04$
d	$1.43^{**} \pm 0.30$	$-0.87^{**} \pm 0.31$
aa	$0.35^{**} \pm 0.10$	$-0.41^{**} \pm 0.11$
ad	$0.45^{**} \pm 0.10$	$0.31^{**} \pm 0.13$
dd	$-0.69^{**} \pm 0.20$	$0.75^{**} \pm 0.20$
	Vista/OK65C93-8	
m	$1.46^{**} \pm 0.18$	$0.60^{**} \pm 0.17$
a	$-0.47^{**} \pm 0.04$	$-0.27^{**} \pm 0.04$
d	$0.65^{NS} \pm 0.51$	$1.57^{**} \pm 0.49$
aa	$0.08^{NS} \pm 0.17$	$0.39^{*} \pm 0.17$
ad	$0.32^{NS} \pm 0.17$	$0.27^{*} \pm 0.17$
dd	$0.26^{NS} \pm 0.35$	$-0.93^{**} \pm 0.35$

*,**Significant at the 0.05 and 0.01 probability levels, respectively; NS Non-significant at the 0.05 probability level; †m = overall mean; a = pooled additive effects; d = pooled dominance effect; aa = pooled additive × additive epistatic effect; ad = pooled additive × dominance epistatic effect; and dd = pooled dominance × dominance epistatic effect.

there would be a high probability of success in selecting for resistance to WSMV in the early generations using symptom ratings.

Epistatic effects were also important contributors to the genetic variation for both symptom ratings and ELISA values in the Tandem/OK65C93-8 cross (Table 4). Additive-by-additive and additive-by-dominance interactions were significant and positive for symptom rating in the Tandem/OK65C93-8 cross, suggesting a reducing effect in the expression of WSMV resistance in this cross. On the other hand, both types of interaction were not significant for symptom rating in the Vista/OK65C93-8 cross (Table 4). Dominance-by-dominance interactions were significant and negative for symptom rating in the Tandem/OK65C93-8 cross (Table 4), indicating their enhancing effect in the expression of WSMV resistance; while they were non-significant in the Vista/OK65C93-8 cross.

It is evident that the magnitude of non-allelic interactions and absolute total of non-fixable gene effects (dominance, additive-by-dominance, and dominance-by-dominance) were greater than the fixable effects (additive and additive-by-additive gene effects) in both crosses and both characters.

The analysis of generation means was a useful method in determining the gene action involved in the inheritance of WSMV resistance using both the three- and six-parameter models. The former was instrumental in the estimation of unbiased additive gene effects, while the latter was useful in providing information on the kind and magnitude of epistatic effects involved in the genetic control of resistance.

Narrow-sense heritability estimates were less than broad-sense heritability estimates for both crosses (Table 5). In the Tandem/OK65C93-8 cross, narrow-sense heritability estimates observed for symptom ratings and ELISA values were low to moderate (h^2_{ns} symptom rating = 0.43; h^2_{ns} ELISA Value = 0.45). The low narrow-sense heritability observed in this cross may have been caused by large epistatic effects shown in the generation means analysis.

In the Vista/OK65C93-8 cross, the narrow-sense heritability was relatively high for both symptom ratings and ELISA values (Table 5). This confirms the presence of additive genetic variability for WSMV resistance, as was observed in the generation means analysis. The broad-sense heritabilities for both crosses and both characters were very high (Table 5).

Table 5. Narrow-sense and broad-sense heritability estimates for WSMV symptom ratings and ELISA values derived from Tandem/OK65C93-8 and Vista/OK65C93-8 winter wheat crosses

	Heritability	
Trait	Narrow-sense	Broad-sense
	Tandem/OK65C93-8	
Symptom rating	0.43 ± 0.10§	0.91
ELISA values	0.45 ± 1.56	0.85
	Vista/OK65C93-8	
Symptom rating	0.71 ± 0.26	0.91
ELISA values	0.74 ± 0.34	0.84

§Heritability estimate ± S.E.

The results from the generation means analysis showed that additive, dominance, and epistatic effects are all involved in the inheritance of WSMV resistance in the OK63C93-8 winter wheat line. Due to the presence of higher magnitudes of non-additive gene effects (dominance, additive-by-dominance, and dominance-by-dominance) combined with small narrow-sense heritability in Tandem/OK65C93-8, selecting for WSMV resistance in the segregating populations of this cross will be complex. Progress from selection for WSMV resistance in early generations of winter wheat will vary from one population to another, as indicated in this study. Therefore, our results suggest that determining relative magnitude of additive and non-additive gene effects of parental combinations may be warranted prior to selecting for WSMV resistance.

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