Effects of Wheat Soilborne Mosaic Virus on Hard Red Winter Wheat

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ABSTRACT

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The effects of wheat soilborne mosaic (WSBM) on 13 hard red winter wheat cultivars were evaluated using disease severity, enzyme-linked immunosorbent assay (ELISA), and virion concentration. The wheats were grown for two seasons in a location with no history of the disease and in a location with a history of severe WSBM. Resistant cultivars (Hawk, Mustang, Newton, Plainsman V, and Tam 108) showed lower disease severity and lesser reductions in height, grain yield, and 1,000 kernel weight (TKW) than susceptible cultivars (Chisholm, Danne, Payne, Sage, Tam 101, Tam 105, Triumph 64, and Vona). Mustang showed the least reduction in number of tillers (8.3%), height (4.2%), grain yield (31.6%), and TKW (0.8%). ELISA was useful to validate the presence of the virus. Results suggest that some mechanism(s) inhibits or slows capsid protein production and virion accumulation or production in cultivars resistant to WSBM.

Wheat soilborne mosaic virus (WSBMV), which causes a serious disease on wheat, is considered to be transmitted by the soilborne fungus *Polymyxa graminis* Ledingham (4,12). WSBM was first reported in the United States in the eastern soft red winter wheat growing areas (9) but has since become one of the major virus diseases of hard red winter wheat in the plains states.

The first record of WSBM in Oklahoma was in 1952, and reductions in yield of 32–61% and reductions in test weight of 0–3% were reported (16). Subsequent studies (2,7,11,13) have evaluated the effect of WSBM on grain yield and reported reductions from 0 to 85%. Other, more comprehensive studies (3,5,10,15) have examined the effects of WSBMV on specific components of wheat production. For example, Nykaza et al (10), using five near-isogenic lines and the two wheat cultivars Centurk and Eagle, reported that reductions in grain yield, kernel weight, tiller number, test weight, and plant height averaged 22.0, 11.8, 11.8, 3.4, and 4.7%, respectively. They concluded that losses due to WSBM varied considerably from season to season and by location.

The results from all of these studies indicate that WSBMV affects yield and growth parameters of wheat. However, no studies have been published that report the effects of WSBMV on cultivars of hard red winter wheats currently being grown in the central and southern plains states and that have been released since the mid-1970s. Also, no studies have been reported that address the use of ELISA and isolation of sedimentable virions to monitor reaction of wheat cultivars to WSBMV in the field, although these techniques have been used in growth chamber and greenhouse studies of WSBMV (6). Therefore, this study was initiated to determine the effect(s) of WSBMV on selected hard red winter wheat cultivars currently grown in the central and southern plains, to evaluate the reaction of these cultivars to WSBMV, and to further examine the mechanism of resistance using symptomatology, ELISA, and virion concentration.

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MATERIALS AND METHODS

Experiment location and design. Trials were conducted at the Plant Pathology Department Experimental Farm west of Stillwater, Oklahoma, during 1984–1985 and 1985–1986. Plots were planted in two locations approximately 150 m apart and situated so neither area drained into the other. One location had a consistent history of severe WSBM (Norge loam) and the other location had no history of the disease (Easpur loam). Soil tests for pH, nitrogen, phosphorus, and potassium were conducted each year in each location. Preplant fertilization and/or liming were used to provide appropriate pH and N-P-K for wheat production in north central Oklahoma and to result in comparable fertility for the two locations. Additional nitrogen (38.1 kg/ha, in the form of ammonium nitrate) was topdressed onto each area in March or early April of each season. Chlorsulfuron (Glean), 9.46 g in 18.9 L/0.405 ha (0.3 oz in 20 gal/acre), was applied in the fall to control weeds, and triadimefon (Bayleton), 113.5 g in 14.2 L/0.405 ha (4.0 oz in 15 gal/acre), was used during the spring as needed to maintain a low incidence of foliar fungal diseases. Plots were planted during September in each season and irrigated with 2.5–5.1 cm of water as coleoptiles were emerging through the soil.

Ten hard red winter wheat cultivars—Chisholm, Danne, Hawk, Newton, Payne, Sage, Tam 101, Tam 105, Triumph 64, and Vona—were tested during 1984–1985. Twenty kernels of each cultivar were planted into each of two 5-m rows replicated five times in each location in early September 1984. Early in March 1985, replications contained seven to nine plants as a result of poor emergence or were thinned to 10 plants. During 1985–1986, nine cultivars were evaluated. Six (Chisholm, Hawk, Newton, Sage, Tam 101, and Vona) had been evaluated during 1984–1985, and three (Mustang, Plainsman V, and Tam 108) were evaluated for the first time. Agronomic practices and treatment of plots were the same as during 1984–1985, but plot design differed. Plots in each location consisted of three 3.05-m rows, solid planted, with four replications per cultivar.

Disease assessment. Individual plants (considered subsamples for each replication) were assessed for disease reaction on 14 and 28 March and 15 April 1985, using a scale of 0 = no mosaic or stunting symptoms, 1 = slight mosaic and slight or no stunting, 2 = moderate mosaic with some stunting, and 3 = severe mosaic and stunting. A disease severity index (DSI) was calculated for each cultivar in each area using the formula of Sherwood and Hagedorn (14). Severity of WSBM in the middle row of each replication was determined on 20 March 1986, using the same scale as described for 1984–1985, but the entire row rather than individual plants were evaluated and no DSI was calculated.

After each visual assessment during

1984–1985, 5 g of foliage was collected from each replication and stored at −20 C until evaluations could be conducted. Collections were made sufficiently early in the season so that no flag or flag minus-1 leaves were collected; this was done to minimize impact on grain yields from the individual plants. From this 5-g sample, 2-g and 1-g subsamples were used for evaluation by electrophoresis and ELISA, respectively. Following the ELISA evaluation of foliage by replication, foliage was combined from replications of each cultivar in each location and evaluated again by ELISA. During 1984–1985, ELISA values obtained from averaging replications resulted in the same information regarding the presence or absence of WSBMV capsid protein as did analyzing the combined samples of five replications. Therefore, samples for analysis by ELISA during 1985–1986 were obtained by combining foliage collected from the outer two rows of each replication nine times, starting in November 1985 and ending in May 1986, and reserving the middle row to collect data pertaining to grain yield. ELISA was conducted in both years using a direct sandwich ELISA with polyclonal antibodies as previously described (1). In each microtiter plate, three wells of a sample from greenhouse-grown Blue Jacket was added as a known negative control to zero the plates prior to reading. Samples were given in relation to these zero values, and therefore small negative values were obtained in some instances. Based on previous experiments, a value greater than 0.100 was considered positive for WSBMV. Isolation and determination of concentration of sedimentable virions by electrophoresis were conducted as previously reported (6), using Hawk, Newton, Sage, and Tam 101 from the 1984–1985 season.

Grain yield and TKW were obtained for each replication during 1984–1985 by combining grain harvested from individual plants and using the mean for the value of the replication. Results were analyzed using an unpaired *t* test to compare the grain yield and TKW from the same cultivar grown in the two different locations. Regression analysis was conducted to determine the usefulness of visual assessment of symptoms (DSI) and ELISA for predicting the effect of WSBMV on yield. For this analysis, data from the 10 plots (five from each location) were used for each cultivar, and analysis determining coefficients of determination was obtained for each assessment date. During 1985–1986, the middle row (not sampled for ELISA) was used to gather data pertaining to tiller count, plant height, grain yield, and TKW. These data were analyzed using an unpaired *t* test as described for 1984–1985.

RESULTS AND DISCUSSION

Symptoms indicative of WSBM were uniformly observed on plants during both years in the location with a history of severe WSBM. Nonuniform symptoms of WSBM were observed in the location with no history of the disease, resulting in considerable statistical variation. However, disease severity in the plots in the location with no history of the disease was consistently lower, especially for the resistant cultivars, than in the plots in the location with a history of severe WSBM (Tables 1 and 2). Grain yields are not comparable between the two years because yields reported during 1984–1985 (Table 1) were obtained from five replications, each consisting of seven to 10 plants, and values for 1985–1986 (Table 2) were obtained using four replicate rows, each 3.05 m long. However, yields were consistently greater in the location with no history of WSBM (Tables 1 and 2), with yield reductions ranging from 31.6% (Mustang, 1985–1986) to 69.4% (Triumph 64, 1984–1985). Resistant and susceptible cultivars had average yield decreases of 40.2 (SD = 10.97) and 54.3% (SD = 10.66) over the two years. Reductions in TKW ranged from 0.8 to 18.8%, with Hawk and Chisholm showing increases (Table 1 and Table 2, respectively). Plant height and tiller counts also were consistently greater in the location with no disease history

Table 1. Reaction of 10 hard red winter wheat cultivars to wheat soilborne mosaic (WSBM) in 1985 in a location with no history of the disease and in one with a history of severe WSBM

	14 March			28 March			15 April				
Cultivar[a]	DSI[b]	ELISA[c]	μg/gfw[d]	DSI	ELISA	μg/gfw	DSI	ELISA	μg/gfw	Yield[e] (g)	TKW[f] (g)
Hawk (R)	0.7	0.058	0.0	0.0	0.311	0.0	0.7	1.180	0.0	102	27
Hawk (R)	6.7*	0.413	2.1	4.0	0.724	7.8	4.0*	0.826	0.8	67*	32*
Newton (R)	0.0	0.102	0.0	0.0	0.440	0.0	0.0	1.400	0.0	73	22
Newton (R)	8.7*	0.505	9.2	12.0	0.967	28.1	3.9	1.290	2.8	49*	22
Chisholm (S)	44.0	1.126	···	18.6	1.238	···	20.0	1.846	···	74	28
Chisholm (S)	62.7	1.367	···	39.3*	1.345	···	40.0*	1.673	···	37*	27
Danne (S)	32.7	0.681	···	22.0	1.332	···	12.7	1.390	···	73	29
Danne (S)	75.0*	1.156	···	40.6	1.161	···	40.0*	1.305	···	27*	25
Payne (S)	16.7	0.845	···	14.6	0.919	···	8.5	1.163	···	116	32
Payne (S)	76.6*	1.444	···	42.6*	1.508	···	39.3*	1.953	···	55*	26*
Sage (S)	41.6	0.999	60.3	33.3	1.330	36.5	24.7	1.356	29.5	57	27
Sage (S)	78.6	1.730	72.7	64.9	1.511	89.0	48.3*	1.512	51.5	28*	25
Tam 101 (S)	44.0	0.924	72.2	32.6	1.207	78.3	24.6	1.230	47.0	46	29
Tam 101 (S)	63.3	1.501	74.2	44.3	1.648	100.1	43.3*	1.589	50.5	28	26
Tam 105 (S)	29.9	0.741	···	22.5	1.096	···	13.4	1.716	···	61	25
Tam 105 (S)	76.7*	1.323	···	55.8*	1.359	···	39.4*	1.771	···	31*	22*
Triumph 64 (S)	32.8	0.495	···	17.3	1.159	···	17.6	1.409	···	85	32
Triumph 64 (S)	81.3*	1.378	···	58.7*	1.428	···	42.2*	1.418	···	26*	26
Vona (S)	16.7	0.387	···	15.3	0.974	···	12.6	1.013	···	68	23
Vona (S)	80.7*	1.449	···	58.0*	1.477	···	45.9*	1.342	···	23*	19*

[a] First listing of each cultivar is for location with no history of WSBM, second listing is for location with a history of severe WSBM. R = resistant, S = susceptible.

[b] Disease severity index = Σ(class × no. plants in class × 100)/(total no. plants × 3), where 0 = no mosaic or stunting symptoms, 1 = slight mosaic and slight or no stunting, 2 = moderate mosaic with some stunting, and 3 = severe mosaic and stunting. Values are the means of seven to 10 plants rated in each of five replications. * = Significant ($P = 0.05$) differences as determined by an unpaired *t* test.

[c] Values are the means obtained from foliar samples collected from five replications with two readings per replication.

[d] Micrograms of sedimentable virions per gram fresh weight of foliar sample.

[e] Average amount of grain collected from seven to 10 plants from each of five replications. * = Significant ($P = 0.05$) differences as determined by an unpaired *t* test.

[f] Thousand kernel weight determined by weighing 500 kernels from each of five replications and multiplying by 2. * = Significant ($P = 0.05$) differences as determined by an unpaired *t* test.

(Table 2), but because of variation among replications, only the values for plant height were consistently significantly different. Hawk, Mustang, and Newton showed the least reduction in yield ($\bar{x}$ = 34.3%, SD = 2.44), and Mustang showed the least reduction in tillers (8.3%), height (4.2%), grain yield (31.6%), and TKW (0.8).

Regression analysis of data from 1984–1985 to determine the relationship between visual assessment of symptoms (DSI) and grain yield and between ELISA and grain yield revealed that visual assessment was the best indicator of the effect of WSBMV on yield. Coefficients of determination (r^2) between DSI and yield for the susceptible cultivars were significant at 14 March 1985 ($P = 0.01$) and at 28 March and 15 April 1985 ($P = 0.05$). However, no significant r^2 values were obtained with the resistant cultivars at any assessment date. Coefficients of determination between ELISA and yield were significant ($P = 0.05$) only with the susceptible cultivars at the first assessment date. Occasionally, high ELISA values were obtained from resistant cultivars that showed low disease severity, e.g., Hawk and Newton, 1984–1985 (Table 1). These high ELISA values may have occurred because only one or a few plants of the resistant cultivars were infected with WSBMV. Thus, visual assessment would indicate a low severity because only one or a few plants were expressing symptoms of WSBM, but there would be sufficient virus capsid in the foliage sample to result in a high ELISA value. This helps explain the poor correlation between ELISA and yield observed in the regression analysis. We feel that visual assessment of symptoms used in conjunction with ELISA to assure presence of the virus in plants is the best approach to ascertaining the effect of WSBMV on yield and to identifying resistant germ plasm.

ELISA values from the location with no history of WSBM during 1984–1985 initially were lower than comparable values from the location with a history of severe WSBM, and ELISA values obtained from resistant cultivars initially were much lower than values from susceptible cultivars (Table 1). By the final assessment on 15 April 1985, ELISA values of all cultivars were comparable. No virus particles were obtained from

Table 2. Disease severity, tiller count, height, yield, and thousand kernel weight (TKW) of nine hard red winter wheat cultivars planted in 1985 in a location with no history of wheat soilborne mosaic (WSBM) and in one with a history of severe WSBM

Cultivar[a]	Disease severity[b]	Tiller count[c]	Height[d] (cm)	Yield[e] (g)	TKW[f] (g)
Hawk (R)	0.3	133	78.5	351	25
Hawk (R)	0.8	105*	70.0*	230	22*
Mustang (R)	1.5	96	72.0	309	26
Mustang (R)	1.8	88	69.0	211*	26
Newton (R)	0.0	129	83.0	327	24
Newton (R)	1.0*	84	71.3*	202*	23
Plainsman V (R)	0.5	92	71.0	451	24
Plainsman V (R)	1.3	63	62.0*	173*	24
Tam 108 (R)	0.0	119	82.5	444	25
Tam 108 (R)	1.0	99	71.3*	229*	21*
Chisholm (S)	0.8	121	74.3	427	27
Chisholm (S)	1.8*	87	64.8*	265*	29
Sage (S)	1.0	105	80.0	257	24
Sage (S)	2.5*	87	66.5*	137*	20*
Tam 101 (S)	1.0	105	75.5	325	29
Tam 101 (S)	2.3	74	58.8*	132	26*
Vona (S)	0.5	120	76.5	493	23
Vona (S)	2.3*	78*	64.3*	163*	21*

[a]First listing of each cultivar is for location with no history of WSBM, second listing is for location with a history of severe WSBM. R = resistant, S = susceptible.
[b]Determined 20 March 1986 by averaging ratings of four replicate plots, each a single 3.05-m row; 0 = no mosaic or stunting symptoms, 1 = slight mosaic and slight or no stunting, 2 = moderate mosaic with some stunting, and 3 = severe mosaic and stunting. * = Significant ($P = 0.05$) differences as determined by an unpaired t test.
[c]Number of tillers with fertile heads in 0.305 m of each row in each of four replications. * = Significant ($P = 0.05$) differences as determined by an unpaired t test.
[d]Determined by measuring from the ground to the base of ears several times within each of four replications and using the average for each replication. * = Significant ($P = 0.05$) differences as determined by an unpaired t test.
[e]Average amount of grain collected from four 3.05-m rows. * = Significant ($P = 0.05$) differences as determined by an unpaired t test.
[f]Thousand kernel weight determined by weighing 500 kernels from each of four replications and multiplying by 2. * = Significant ($P = 0.05$) differences as determined by an unpaired t test.

Table 3. Detection of wheat soilborne mosaic virus capsid protein by ELISA in nine hard red winter wheat cultivars during 1985–1986 in a location with no history of wheat soilborne mosaic (WSBM) and in one with a history of severe WSBM

	Date of assessment by ELISA[b]								
	1985		1986						
Cultivar[a]	24 Nov.	22 Dec.	19 Jan.	16 Feb.	2 Mar.	16 Mar.	30 Mar.	20 Apr.	11 May
Hawk (R)	−0.030	0.019	−0.010	−0.005	0.002	0.013	−0.043	0.046	0.215
Hawk (R)	−0.004	0.012	0.026	−0.005	0.013	0.040	0.021	0.109	0.368
Mustang (R)	−0.039	0.008	−0.021	−0.030	0.002	−0.014	0.055	−0.007	0.085
Mustang (R)	−0.006	0.023	0.048	0.615	0.309	0.460	0.666	0.008	0.503
Newton (R)	−0.005	−0.013	0.004	−0.022	0.022	0.012	−0.012	−0.002	0.151
Newton (R)	−0.007	0.008	0.297	0.009	0.054	0.504	−0.014	−0.003	0.217
Plainsman V (R)	−0.020	−0.025	−0.019	0.002	0.000	0.033	0.042	0.043	0.032
Plainsman V (R)	−0.008	−0.023	0.070	0.489	0.692	0.031	0.232	0.027	0.332
Tam 108 (R)	−0.030	0.007	−0.048	−0.005	0.046	0.015	−0.046	0.085	0.076
Tam 108 (R)	−0.035	0.156	0.415	0.436	0.298	0.128	−0.048	0.271	0.562
Chisholm (S)	−0.045	0.061	2.080	0.262	0.174	0.628	0.681	0.902	0.062
Chisholm (S)	1.175	0.234	2.415	1.015	1.310	1.124	1.373	1.643	0.679
Sage (S)	−0.040	−0.001	0.775	0.208	0.018	0.525	0.454	0.424	0.078
Sage (S)	1.728	0.226	1.871	0.889	1.268	0.696	0.646	0.549	0.826
Tam 101 (S)	−0.002	0.026	0.954	1.018	0.761	0.253	0.879	0.025	0.485
Tam 101 (S)	1.374	1.595	2.168	1.220	0.883	1.463	0.889	1.089	0.914
Vona (S)	−0.082	0.143	−0.015	−0.052	−0.005	0.008	0.564	0.012	0.016
Vona (S)	0.783	1.268	1.446	1.047	0.702	0.778	0.656	0.471	0.651

[a]First listing of each cultivar is for location with no history of WSBM, second listing is for location with a history of severe WSBM. R = resistant, S = susceptible.
[b]Values are average of seven readings obtained from a composite foliar sample collected from four replicate plots in each location.

the resistant cultivars Hawk and Newton growing in the location with no history of WSBM (Table 1), although virions were obtained from these cultivars growing in the location with a history of severe WSBM. ELISA values obtained during 1985–1986 were more sporadic over a longer period of time than those obtained during 1984–1985. No ELISA values were considered positive from resistant cultivars planted in the location with no history of WSBM until the final reading of the season on 11 May 1986 (Table 3). Positive ELISA values from resistant cultivars in the location with a history of severe WSBM were obtained as early as 22 December 1985 (e.g., Newton and Tam 108). In contrast, positive ELISA values were obtained from susceptible cultivars as early as 24 November and 22 December 1985 from the locations of severe history and no history of WSBM, respectively (Table 3). These results from two seasons indicate that WSBMV capsid protein and virions are produced in resistant wheat cultivars, but at a lower concentration and/or reduced rate than in susceptible cultivars. In this and in a previous study (6), amounts of virus obtained from resistant cultivars never equaled amounts obtained from susceptible cultivars, indicating that virus accumulation may be inhibited or replication may proceed at a slower rate in resistant cultivars. Larsen et al (8) reported greater sensitivity to foliar inoculation with WSBMV by cultivars field-resistant to WSBM and thus felt that tolerance and resistance to the virus at the cellular level were not possible mechanisms of plant resistance. Our results indicate that some mechanism is present in resistant cultivars that slows capsid protein and virus accumulation. This could result from a reduction in some aspect of the replicative cycle of the virus, although further work is indicated to ascertain more fully the mechanisms of resistance to WSBMV.

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