

Temperature-Influenced Virus Movement in Expression of Resistance to Soilborne Wheat Mosaic Virus in Hard Red Winter Wheat (*Triticum aestivum*)

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Journal Article J6350. Oklahoma Agricultural Experiment Station, Oklahoma State University, Stillwater. Support from the Oklahoma Center for Advancement of Science and Technology, and the Oklahoma Wheat Research Foundation is gratefully acknowledged. Accepted for publication 30 January 1993.

ABSTRACT

Myers, L. Drumm, Sherwood, J. L., Siegerist, W. C., and Hunger, R. M. 1993. Temperature-influenced virus movement in expression of resistance to soilborne wheat mosaic virus in hard red winter wheat (*Triticum aestivum*). *Phytopathology* 83:548-551.

Soilborne wheat mosaic (SBWM) disease, caused by the soilborne wheat mosaic furovirus (SBWMV), is a major disease of hard red winter wheat (*Triticum aestivum*) in the plains states. The virus is transmitted by the soilborne fungus *Polymyxa graminis*. Although cultivars expressing resistance to SBWM have been developed, information regarding the mechanism(s) of resistance is lacking. When resistant (Hawk and Newton) and susceptible (Sage and Vona) cultivars were planted in soil containing viruliferous *P. graminis* under conditions conducive for fungal and subsequent viral infection, SBWMV was readily detected by ELISA in roots of all cultivars but was detected in the foliage of only the susceptible

cultivars. When the cultivars were mechanically inoculated with SBWMV the virus was detected in the foliage and later in the roots of all cultivars, indicating downward translocation of virus was not impeded in any of the cultivars. The reaction of resistant and susceptible cultivars to SBWMV was similar when the cultivars were initially grown at 15 C, so infection by viruliferous *P. graminis* occurred, and then transferred to 23 C. Virus was detected in the roots and foliage of all cultivars. Thus, resistance to SBWMV may be related to differential virus movement in resistant versus susceptible cultivars with temperature modulating the expression of the resistance.

Additional keywords: enzyme-linked immunosorbent assay (ELISA), serology.

Soilborne wheat mosaic (SBWM) disease, caused by the soilborne wheat mosaic furovirus (SBWMV), is a major disease of hard red winter wheat (*Triticum aestivum* L.) in the plains states (7). Losses caused by SBWM range to 80% and vary with cultivar, geographical area, and growing conditions (5,13,17,23). SBWMV has a bisegmented genome consisting of RNAs of 7,090 and 3,593 nucleotides encapsidated individually into particles of 281 × 20 nm or 142 × 20 nm (30). Both components are required for infection (29). The virus is transmitted by the soilborne fungus *Polymyxa graminis* Led. (10). Viruliferous resting spores of *P. graminis* can survive many years in the soil and cannot be eliminated in an economically feasible manner.

Infection of wheat by SBWMV is accomplished by colonization of the roots by viruliferous *P. graminis* zoospores, transfer of some infectious form from the fungus to the roots, and subsequent replication, movement, and assembly of virus. How this occurs in the interaction between *P. graminis*, SBWMV, and wheat has not been demonstrated. An open reading frame (ORF) on RNA2, analogous to one in beet necrotic yellow vein virus, has been proposed to be involved in fungal transmission of SBWMV (31).

Efforts to control SBWM have focused on the use of resistant cultivars. Although cultivars expressing resistance to SBWMV have been developed, information regarding the mechanism(s) of resistance is lacking. The roots of both susceptible and resistant cultivars can be colonized by *P. graminis* (18,19), suggesting that resistance is directed at aspects of virus infection rather than the vector. Larsen et al (19) proposed that field resistance to SBWMV could be due to reduced susceptibility to viruliferous zoospores, a root-localized hypersensitive response, or reduced virus movement in roots of resistant cultivars. The detection of virus and viral coat protein in foliage of both resistant and susceptible cultivars following spring growth as temperature increases suggests that resistance does not involve localized inhibition of viral replication or movement (2) and that viral movement from the roots and/or viral replication is environmentally modulated.

A better understanding of resistance to SBWM would facilitate evaluation and development of germplasm with resistance to SBWM, and perhaps other viruses of wheat that are transmitted by a soilborne vector. Here we report results that indicate resistance is expressed in the cultivars Hawk and Newton as an inhibition of virus movement from the roots and that temperature modulates the expression of the resistance. Preliminary reports have been published (26-28).

MATERIALS AND METHODS

Wheat cultivars, virus source, and planting conditions. Two susceptible (Vona and Sage) and two resistant (Hawk and Newton) cultivars were selected based on field performance (15). Soil infested with SBWMV-viruliferous *P. graminis* was obtained from a field with a history of severe SBWM located near Stillwater, OK (15). The soil in this location is classified as a Norge loam (fine-silty, mixed, thermic Udic Paleus-toll). The soil used over a 12-mo period was collected in September, sifted through a large mesh screen to remove root and straw debris, and then stored at field moisture at 4 C. A commercial soil mix was used for soil not infested with viruliferous *P. graminis*. Inoculum for mechanical inoculations was prepared from symptomatic Vona foliage grown in the same field from which soil containing the viruliferous *P. graminis* had been collected. The foliage was collected in February or March because previous work (1,2,15) indicated that the titer of the virus in the foliage of Vona was highest at this time under the conditions in this area of Oklahoma. The foliage was stored at -20 C and used for inoculum during the 12-mo period after collection. In all experiments, 10 seeds of each cultivar were sown in soil in 10-cm pots producing five to seven seedlings. Subsequent environmental conditions varied with the experiment as outlined below. Temperature in the growth chambers was either 15 C or 23 C, and a mixture of fluorescent and incandescent lamps were used (180 $\mu\text{E m}^{-2} \text{sec}^{-1}$ at plant level; 11 h day/13 h night).

Inoculation by viruliferous vector. In experiments where plants were infected using viruliferous *P. graminis*, or similar control experiments, infection was obtained by maintaining high soil

moisture at 15 C during seed germination by flooding soil as previously described (6,14). A temperature of approximately 15 C is needed for establishment of infection and subsequent replication of SBWMV (6,14,19).

Mechanical inoculation with SBWMV. Mechanical inoculations with SBWMV were done using approximately 25 g of infected foliar tissue ground in 200 ml of 0.01 M phosphate buffer, pH 7.0. Plants previously dusted with 225- μ m corundum were rubbed with an inoculum-saturated cheesecloth pad and then placed in the appropriate environment.

Evaluation for SBWMV by ELISA. Samples for ELISA were taken at various intervals and stored a maximum of 2 wk after completion of an experiment at -20 C until processed. Soil was removed from root samples by washing prior to storage at -20 C. Plants were assayed using an indirect sandwich ELISA with a polyclonal capture antibody and a monoclonal probe antibody as previously described (4,15). The A_{405nm} value obtained in ELISA used for delineating a resistant and susceptible reaction, and the basis of interpretation of the values, has been described elsewhere (15). A value of <0.100 was considered a resistant reaction and a value \geq 0.100 was considered a susceptible reaction. Samples of uninoculated healthy plants were used to zero the plate reader.

RESULTS AND DISCUSSION

Reaction of resistant and susceptible cultivars to inoculation by viruliferous *P. graminis* at 15 C. From 8 to 36 days after planting seed of each cultivar in soil containing viruliferous *P. graminis*, samples from resistant (Hawk and Newton) and susceptible (Sage and Vona) cultivars were taken to determine if there were differences in the initial infection by viruliferous *P. graminis* (Table 1). SBWMV was readily detected by ELISA in roots of all cultivars by 11 days after planting and in the remainder of the root samples assayed. SBWMV was not detected in any foliage samples of Hawk and Newton. However, SBWMV was detected in foliage of Sage and Vona at 22 days and 25 days after planting, respectively. Thus, all four cultivars were initially infected by SBWMV, but there was no subsequent establishment of SBWMV in the foliage of the resistant cultivars.

In previous growth chamber (1,19) and field (18) experiments roots were not assayed for SBWMV by ELISA, but *P. graminis* was consistently found in both resistant and susceptible cultivars, indicating the resistance is most likely not directed at the fungus. In a recent field study, roots of both resistant and susceptible cultivars were found by ELISA and the reverse transcriptase-polymerase chain reaction (24; R. E. Pennington, J. L. Sherwood, and R. M. Hunger, unpublished) to be infected by SBWMV.

Reaction of resistant and susceptible cultivars to mechanical inoculation. To determine if the lack of detectable SBWMV in

the foliage of Hawk and Newton was due to the inability of these cultivars to support virus replication in the foliage, the cultivars were grown in commercial potting soil and then mechanically inoculated at 7-10 days after planting. Although successful mechanical inoculation of SBWMV to foliage of SBWMV-resistant cultivars has been reported (19), we found that if plants were inoculated and then immediately returned to 15 C virus was not subsequently detected by ELISA (27). However, infections were obtained if plants were retained at room temperature (approximately 22 C) for 18-32 h after mechanical inoculation, and then placed at 15 C.

Following this protocol, SBWMV was detected in foliage of susceptible Sage 1 day after inoculation and in foliage of the other cultivars 4 days after inoculation (Table 2). SBWMV was found in subsequent foliar samples, except one (cultivar Vona, day 18). When roots from the same plants were assayed for SBWMV, the virus was found in Newton 18 days after inoculation and in the other three cultivars 25 days after inoculation. These data indicate that the foliage of resistant Hawk and Newton supports replication of SBWMV similarly to the susceptible Sage and Vona and that the virus subsequently moves and/or replicates in the root tissue. In addition, unlike the upward translocation of SBWMV which is inhibited in Hawk and Newton when inoculated with viruliferous zoospores, the downward translocation of the virus is not impeded. Hence, resistance may result from a one-way blocking of virus translocation. Runjaum and Lapierre (25) recently reported that several cultivars of wheat could be infected by mechanical inoculation of roots.

Reaction of resistant and susceptible cultivars to inoculation by viruliferous *P. graminis* followed by temperature shift to 23 C. Results of many studies (1,2,13-15,24; R. E. Pennington, J. L. Sherwood, and R. M. Hunger, unpublished) showed that SBWMV is in the foliage of resistant Hawk and Newton during the spring as temperature increases and symptoms of SBWMV wane in susceptible Sage and Vona, which indicates SBWMV can become established in the foliage of resistant cultivars. To determine if the establishment of SBWMV in the foliage of Hawk and Newton is influenced by temperature, seed of the four cultivars were planted in field soil containing viruliferous *P. graminis* and placed at 15 C for at least 7 days to allow for establishment of infection of SBWMV. This was based on results presented in Table 1 showing that SBWMV could be detected by 11 days in plants grown in soil with viruliferous *P. graminis*. On 7, 10, 14, 17, and 21 days after planting; half the pots of plants in each treatment were transferred to 23 C. Plants in each paired treatment were then grown for another 24 days at either 15 C or 23 C (Table 3). As in the results presented in Table 1, the virus was found in the roots of all cultivars in the plants retained at 15 C; but virus was only found in the foliage of the susceptible

TABLE 1. Enzyme-linked immunosorbent assay (A_{405nm}) of roots and shoots of resistant (Hawk, Newton) and susceptible (Sage, Vona) cultivars of hard red winter wheat grown at 15 C in soil infested with soilborne wheat mosaic virus (SBWMV) viruliferous *Polymyxa graminis*^{a,b}

Days after planting	Resistant cultivars				Susceptible cultivars			
	Hawk		Newton		Sage		Vona	
	Root	Shoot	Root	Shoot	Root	Shoot	Root	Shoot
8	0.04	0.01	0.01	0.02	0.01	0.02	0.02	0.01
11	0.67	0.03	0.60	0.04	0.60	0.03	0.29	0.05
15	0.48	0.05	0.42	0.02	0.52	0.01	0.43	0.08
22	0.37	0.04	0.40	0.05	1.60	0.27	0.36	0.09
25	0.33	0.04	0.19	0.03	1.57	0.21	0.61	0.13
29	0.36	0.04	0.36	0.05	0.44	0.08	1.32	0.47
32	0.30	0.05	0.18	0.03	1.50	1.30	0.94	0.27
36	0.16	0.03	0.53	0.04	1.59	1.08	0.40	0.33

^aEach value is an average of three experiments with three replications in each experiment.

^b $A_{405nm} < 0.10$ reflects a resistant reaction for SBWMV and an $A_{405nm} \geq 0.10$ reflects a susceptible reaction for SBWMV. Samples of uninoculated healthy plants were used to zero the plate reader.

TABLE 2. Enzyme-linked immunosorbent assay (A_{405nm}) of roots and shoots of resistant (Hawk, Newton) and susceptible (Sage, Vona) cultivars of hard red winter wheat from which the foliage was mechanically inoculated with soilborne wheat mosaic virus (SBWMV), then plants retained at 23 C for 24 h, then grown at 15 C^{a,b}

Days after mechanical inoculation	Resistant cultivars				Susceptible cultivars			
	Hawk		Newton		Sage		Vona	
	Root	Shoot	Root	Shoot	Root	Shoot	Root	Shoot
1	0.00	0.00	0.00	0.00	0.00	0.55	0.00	0.00
4	0.00	0.32	0.00	0.44	0.00	0.55	0.00	0.53
8	0.00	0.43	0.00	0.36	0.06	0.71	0.00	0.40
11	0.00	0.26	0.00	0.27	0.00	0.59	0.00	0.16
18	0.00	0.37	0.21	0.58	0.00	0.90	0.00	0.05
25	0.65	0.67	0.14	1.11	0.81	1.10	0.40	0.86
32	0.32	0.84	0.53	1.15	1.24	1.83	0.59	1.63

^aEach value is an average of three experiments with three replications in each experiment.

^b $A_{405nm} < 0.10$ reflects a resistant reaction for SBWMV and a $A_{405nm} \geq 0.10$ reflects a susceptible reaction for SBWMV. Samples of uninoculated healthy plants were used to zero the plate reader.

TABLE 3. Enzyme-linked immunosorbent assay (A_{405nm}) of roots and shoots of resistant (Hawk, Newton) and susceptible (Sage, Vona) cultivars of hard red winter wheat planted at 15 C in soil infested with soilborne wheat mosaic virus (SBWMV) viruliferous *Polymyxa graminis*, then half of each experimental paired set was retained at 15 C (R-15 C) or moved to 23 C (M-23 C) at different times after germination^{a-c}

Days after planting that one-half of each paired set moved to 23 C	Resistant cultivars								Susceptible cultivars							
	Hawk				Newton				Sage				Vona			
	Root		Shoot		Root		Shoot		Root		Shoot		Root		Shoot	
	R-15 C	M-23 C	R-15 C	M-23 C	R-15 C	M-23 C	R-15 C	M-23 C	R-15 C	M-23 C	R-15 C	M-23 C	R-15 C	M-23 C	R-15 C	M-23 C
7	0.50	0.49	0.01	0.70	0.35	0.20	0.01	0.26	0.53	0.49	0.75	0.50	0.73	0.63	0.75	0.77
10	0.53	0.38	0.01	0.58	0.37	0.35	0.01	0.57	0.71	0.46	0.62	0.44	0.65	0.69	0.75	0.74
14	0.53	0.65	0.02	0.84	0.39	0.35	0.01	0.61	0.47	0.42	0.56	0.45	0.63	0.70	0.61	0.79
17	0.58	0.41	0.07	0.47	0.34	0.30	0.01	0.52	0.58	0.51	0.53	0.38	0.74	0.68	0.72	0.70
21	0.25	0.43	0.09	0.74	0.33	0.37	0.01	0.71	0.46	0.46	0.67	0.50	0.73	0.67	0.77	0.64

^a Each value is an average of five individual experiments.

^b $A_{405nm} < 0.10$ reflects a resistant reaction for SBWMV and an $A_{405nm} \geq 0.10$ reflects a susceptible reaction for SBWMV. Samples of uninoculated healthy plants were used to zero the plate reader.

^c Plants in each paired treatment were harvested 24 days after the set was moved to 23 C.

cultivars (Sage, Vona). However, SBWMV was detected in the roots and in the foliage of all cultivars when plants were shifted to 23 C. This modest shift in temperature appears to suppress expression of resistance to virus establishment in the foliage.

The inhibition of virus movement has been proposed as one of several possible mechanisms of resistance to SBWMV (19). Virus movement is an important component of the virus-host interactions (3,8), and the specific inhibition of the long-distance component of movement has been implicated as a mechanism of resistance in several other virus-host interactions. These include: maize dwarf mosaic virus (MDMV) on maize (20,21), cucumber mosaic virus (CMV) on pepper (9,22), cowpea chlorotic mottle virus (CCMV) on soybean (11), alfalfa mosaic virus on alfalfa (16), and tomato yellow top virus (TYTV) and potato leaf roll virus (PLRV) in *Lycopersicon peruvianum* (L.) Mill (12). A common feature of these results, as with results presented here, was that there was little or no difference found in the ability of susceptible and resistant plants to support initial viral infection, viral replication, and cell-to-cell spread, whereas differences were found in the subsequent movement of the virus. In the MDMV-maize system (20) it was observed that downward movement of virus, indicated by the accumulation of virus in the roots and other tissues below the inoculated leaf, occurred in both susceptible and resistant plants. Upward movement, indicated by the presence of virus in younger leaves above the inoculation point, occurred in all susceptible plants. But upward movement was restricted in resistant plants unless inoculation was performed before the emergence of the younger leaves. The authors proposed it was due to an uncoupling of upward and downward components of virus movement. Uncoupling might occur in the inoculated leaf where separate mechanisms for phloem loading or unloading of virus (or some other viral form) for upward and downward movement might exist. Alternatively, uncoupling could occur in the roots where loading for upward movement occurs. Non-Wondim et al (22) observed a similar inhibition of upward, but not downward, movement of CMV in resistant pepper lines following inoculation and attributed resistance to inhibition of viral entry or movement within the vascular system. Supporting this conclusion was an earlier study (9) where immunofluorescence microscopy showed virus to be distributed throughout all tissues and organs of susceptible plants but restricted to the inoculated leaf and only one or two phloem bundles in resistant plants. The results of immunocytochemical studies with CCMV by Goodrick et al (11) were similar in that very little viral antigen was found in the vascular tissue of inoculated plants of a resistant soybean line, whereas viral antigen was relatively abundant in the vascular tissue of inoculated susceptible plants. They proposed that nonnecrotic resistance was due to inhibition of viral entry or exit from the vascular system.

Unlike resistance to MDMV in maize, which was not affected by temperature or plant age, resistance to CMV was temperature and plant age dependent. Resistance in one of two resistant pepper lines was found to be reduced in the greenhouse during winter

or in the growth chamber at 25–12 C (day-night) as compared to summer in the greenhouse or in the growth chamber at 32–21 C (day-night). Further, resistance was expressed only in plants of either resistant cultivar that had reached the four to five true leaf stage. The resistance to TYTV and PLRV in *L. peruvianum*, which was expressed as an inhibition of virus movement, was influenced by the quality of light (12).

We propose that resistance to SBWMV in Hawk and Newton is related to inhibition of virus movement from the roots. A similar conclusion with other cultivars of wheat has recently been presented in a preliminary report (25). In addition, the results presented here indicate temperature has a modulating effect on virus movement in resistant versus susceptible cultivars. Current studies are under way to localize the RNA1, RNA2, and coat protein in the resistant and susceptible cultivars to try to elucidate how resistance is expressed at the cellular level. This may provide insight into a general mechanism whereby taxonomically different viruses are controlled by an inhibition of virus movement.

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