Relationships Between Wheat Streak Mosaic Virus and Soilborne Wheat Mosaic Virus Infection, Disease Resistance, and Early Growth of Winter Wheat

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ABSTRACT

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Hard red winter wheat cultivars resistant (Homestead and Newton) and highly susceptible (Scout-66) to field infection by soilborne wheat mosaic virus (SBWMV) were all susceptible to laboratory infection by SBWMV through sap inoculation of leaves and to root colonization by the SBWMV vector fungus, *Polymyxa graminis*. Effects of SBWMV infection on root growth were inversely related to resistance observed in the field, because Homestead and Newton were more severely affected than Scout-66. Shoot growth was unaffected for all cultivars, but secondary root growth of infected plants was reduced to 16% of control plant growth. In contrast, Homestead and Scout-66 inoculated with wheat streak mosaic virus (WSMV) had similar reductions in their root or shoot growth; shoot growth was less affected by WSMV infection than root growth, and primary root growth was less affected than secondary root growth. Resistance to *Polymyxa* zoospores carrying SBWMV or reduced movement of SBWMV within roots of cultivars resistant to SBWMV in the field are suggested as possible mechanisms of this resistance to SBWMV. Tolerance to the virus, resistance to the virus at the cellular level, and resistance to the vector fungus alone (without virus) are eliminated as possible mechanisms.

Additional key words: field resistance, root and shoot dry weight, root volume and length

Soilborne wheat mosaic virus (SBWMV) is a serious disease problem for North American producers of winter wheat (*Triticum aestivum* L.). Crop loss to SBWMV was as high as 100% in fields of susceptible varieties during the 1920s (16), and losses of 45–50% for susceptible cultivars are still reported (5,13,19). Extended cool, wet spring weather increases disease severity (18,25). At present, control is obtained primarily through later planting dates and use of cultivars resistant to SBWMV under field conditions. When planted in infested soil or when inoculated by root washings and grown in controlled-environment chambers, these resistant cultivars seldom show mosaic, stunting, or yield loss. However, they do show typical mosaic symptoms when inoculated with SBWMV by leaf rubbing (19), and we use the terms "field resistant" or "field resistance" in this paper to describe this condition.

Lommel and Willis (15) reported extensive infection of SBWMV-resistant cultivars by a combination of SBWMV and wheat spindle streak mosaic virus (WSSMV). Both SBWMV and WSSMV are carried by the same fungus, *Polymyxa graminis* Led., and their report further suggests that cultivars with field resistance to SBWMV are susceptible to the vector fungus. The report of potential breakdown in this resistance to SBWMV in the presence of WSSMV is cause for concern, particularly since the mechanism of this field resistance is unknown. Firm documentation of cultivar susceptibility to the vector fungus and a description of the effects of SBWMV and other wheat viruses on shoot and root growth of winter wheat cultivars with disparate field resistance to SBWMV should help clarify the mechanism(s) of this resistance. This study compares the effects of SBWMV and of wheat streak mosaic virus (WSMV) on early plant growth of winter wheat cultivars equally susceptible to WSMV but differing in field resistance to SBWMV. It also compares susceptibility of these cultivars to the vector fungus, *P. graminis*.

MATERIALS AND METHODS

Experiments to study the effects of SBWMV and WSMV on early plant growth were replicated four times for each virus with winter wheat cultivars Homestead (field resistant to SBWMV) and Scout-66 (highly susceptible to SBWMV in the field). Procedures were tested first with WSMV, which is easier to mechanically or rub inoculate than SBWMV (21). Each of the four treatments (WSMV-infected Homestead, WSMV-infected Scout-66, and their corresponding mock-inoculated, uninfected controls) consisted of five plants, although 10 plants for each treatment were initially included to allow for any possible loss to root or crown rot. Individual seeds were planted in 10-cm-diameter clay pots containing a pasteurized (1 hr at 80 C), sieved potting mix of loam,

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sand, peat, and vermiculite (14:7:4:3). Plants were grown in a greenhouse at 25–27 C with >12 hr natural lighting per day and watered as needed. Young plants were inoculated 10 days after planting with a type culture of WSMV (PV-57) (1). The developing second leaves (3–8 cm long) (Haun stage 1.2–1.7 [8]) were rubbed with infected leaf sap in water to which Celite (diatomaceous earth) was added as an abrasive. Control seedlings were mock-inoculated with water and Celite only. The five- to six-leaf plants (Haun stage 4.8-5.2) were harvested 29–31 days after planting with five plants free of root or crown rot randomly selected per treatment. The roots were washed free of soil and debris, and the plants were stored at 4 C pending laboratory processing and measurement. Plants were dissected to separate individual roots, which were then grouped as primary (seminal) or secondary (adventitious). Primary roots consisted of Klepper designations R, −2A, −2B, −1A, −1B, and −2Y when present (12), and the remaining leaf and tiller-associated roots (0A, 0B, 1A, 1B, etc.) were included in the secondary roots. The main axis length of each root was determined by the grid-intercept method (2). Roots of each plant were then bulked as primary or secondary, the root system volume measured by water displacement (20), and the root systems and shoots dried 3–4 days in an oven at 80 C before dry weight determination.

The procedures for the WSMV experiments were followed for the SBWMV experiments with the following modifications. A strain of SBWMV was obtained from locally grown, field-infected wheat and used to inoculate the developing leaves as in the WSMV experiments. This strain of SBWMV was subsequently determined to be similar to the Lab 1 mutant of Shirako and Brakke (24) with a preponderance of 92-nm rods and a higher rate of mechanical transmission than the wild type of SBWMV. Cultivars Homestead and Scout-66 were used in all four repetitions. Newton, a cultivar with a different gene for resistance to SBWMV (17), was included in the final repetition. Seeds were germinated for 4 days on moist filter paper at 22–23 C to facilitate more rapid growth to the inoculation stage under the cooler temperatures (15–17 C) required for optimum SBWMV replication. A single seedling was placed in each 10-cm-diameter clay pot, and the plants were placed in a growth chamber at 15–17 C with 25,000 lm/m^2 cool-white fluorescent illumination on a 12-hr diurnal cycle and watered as needed. All plants were given 5 days of darkness at 6 days postinoculation (Haun stage 1.6–1.9) to stimulate virus movement and systemic infection. They had two to three leaves at the end of the dark treatment (Haun stage 1.8–2.2). Six- to seven-leaf plants (Haun stage 5.9–6.9) were harvested 42–52 days after planting with a minimum of 7 days of growth after onset of foliar symptoms. Harvest and postharvest processing followed the same procedures for WSMV.

Susceptibility of hard red winter wheat to *P. graminis* was evaluated for cultivars Homestead, Newton, and Scout-66. A Prince Edward Island isolate of *P. graminis* (obtained from D. J. S. Barr, Ottawa) free of SBWMV was used for the study. This Canadian isolate was morphologically identical to the local viruliferous strains of the fungus. Wheat seedlings were germinated for 2 days on moist filter paper at 22–23 C, exposed for 24 hr to root washings from *P. graminis*-infected culture plants (3), and transplanted into sterile sand in 10-cm clay pots for growth at 15 C in a growth chamber with 25,000 lm/m^2 cool-white fluorescent illumination on a 12-hr diurnal cycle. Plants were watered at 2- to 3-day intervals with one-third-strength Hoagland's solution (9). Root systems of the plants were washed free of sand after 1 mo, and unstained roots were examined with a compound microscope for presence of the fungus.

Data were analyzed by standard analysis of variance methods with orthogonal contrasts (23,26). Initial data for each plant included the numbers of shoots and primary and secondary roots, total shoot dry weight, and the volume, dry weight, and main axis length for both the primary and the secondary root systems. From these were derived for each plant the total root volume, dry weight, and main axis length, and the total root:total shoot dry weight ratio. Response to virus infection (as percentage of control plant growth) was calculated for each characteristic by dividing the individual plant characteristic by the associated control plant mean (n = 4). These percentages were converted back to decimal form, divided by three to eliminate values >1.000, and arc sine-transformed before analysis. Zero values in the raw data and subsequent calculated data were assigned a value of 0.0001 to eliminate analysis problems. A confidence level of $P < 0.05$ was used for all comparisons.

Table 1. Effects of wheat streak mosaic virus (WSMV) on growth of 29- to 31-day-old Homestead and Scout-66 winter wheat grown at 25–27 C for 19–21 days after inoculation

	Means						
	Homestead[a]			Scout-66[b]			
Growth parameter	H[c]	I[d]	Resp.[e]	H[c]	I[d]	Resp.[e]	Cont.[f]
Number							
Primary roots	4.10	3.65	89	4.55	4.00	88	NS
Secondary roots	5.85	3.75	63*	8.20	3.95	44*	NS
Shoots	5.30	4.50	87	5.95	4.45	77	NS
Length (m)							
Primary root system	1.61	1.16	72*	1.84	1.05	62*	NS
Secondary root system	1.11	0.46	41*	1.41	0.49	33*	NS
Total root system	2.72	1.62	60*	3.25	1.54	52*	NS
Volume (ml)							
Primary root system	0.76	0.32	48*	0.62	0.29	52*	NS
Secondary root system	0.59	0.18	33*	0.58	0.16	27*	NS
Total root system	1.35	0.50	40*	1.19	0.45	40*	NS
Dry weight (mg)							
Primary root system	81.20	38.10	52*	81.30	33.60	46*	NS
Secondary root system	58.10	18.90	34*	60.80	16.50	24*	NS
Total root dry wt	139.00	57.00	43*	142.00	50.00	37*	NS
Total shoot dry wt	395.00	254.00	64*	408.00	254.00	66*	NS
Root:shoot biomass ratio	0.36	0.23	71	0.37	0.20	59*	NS

[a] Susceptible to WSMV, resistant to soilborne wheat mosaic virus (SBWMV) under field conditions.
[b] Susceptible to WSMV, susceptible to SBWMV under field conditions.
[c] H = healthy, mock-inoculated plants.
[d] I = WSMV-infected plants.
[e] Mean response to virus infection as a percentage of uninfected control plant growth. * = Significant ($P < 0.05$) pairwise contrast of virus-infected vs. uninfected plants.
[f] Pairwise contrasts between cultivars for response to virus infection; NS = nonsignificant contrast.

RESULTS

The course of symptom expression varied with virus type and wheat cultivar. WSMV-inoculated plants showed symptoms most quickly, with first foliar symptoms for both Homestead and Scout-66 appearing in the third-leaf stage, 6–8 days after inoculation (i.e., first leaf postinoculation; Haun stage 2.2–2.9). In contrast, SBWMV-infected plants did not show symptoms until later, and the SBWMV-resistant cultivars showed symptoms before the SBWMV-susceptible cultivar. SBWMV-inoculated Homestead and Newton typically first showed foliar symptoms in their fourth or fifth leaf 19–22 days after inoculation (second or third leaf postinoculation; Haun stages 3.8–4.9 and 3.5–4.8, respectively), whereas Scout-66 plants usually first showed foliar SBWMV symptoms in their fifth to seventh leaf 24–32 days after inoculation (third to fifth leaf postinoculation; Haun stage 4.8–6.2). Thus, WSMV-infected plants grew 12–14 days

between symptom expression and harvest, whereas SBWMV-infected Homestead, Newton, and Scout-66 grew 16–20, 13–17, and 11–15 days, respectively, between symptom expression and harvest.

Homestead and Scout-66 did not differ in their responses to WSMV, and growth of their respective uninfected controls differed only slightly after 29–31 days at 25–27 C (Table 1). WSMV infection reduced secondary root growth most, exemplified by secondary root system dry weights for infected Homestead and Scout-66 that were only about 30% of control plant values. Least affected were the numbers of shoots and primary roots (about 80 and 90% of controls, respectively). Virus infection also reduced total root dry weight for both cultivars more than total shoot dry weight (about 40 and 65% of controls, respectively). Uninfected Homestead control plants had slightly smaller root numbers and lengths but slightly greater root volumes than comparable Scout-66 control plants; mean root and shoot dry weights and root:shoot dry weight ratios for the two cultivars were almost identical.

In contrast, Homestead and Scout-66 differed greatly in most of their responses to SBWMV infection even though their respective uninfected controls differed only slightly (except for secondary root growth) after 42–52 days at 15–17 C (Table 2). Virus infection again reduced secondary root growth most and primary root numbers and shoot growth least for both cultivars, but SBWMV-infected Scout-66 had greater primary root growth (about 75% vs. about 45% of control growth) and much greater secondary root growth (about 72% vs. about 25% of control growth) than SBWMV-infected Homestead. SBWMV-infected cultivars did not differ in the numbers of shoots and primary roots produced, but infected Homestead had greater shoot dry weight than infected Scout-66. Uninfected controls of these cultivars had similar primary root growth and numbers of shoots produced, but Scout-66 controls had 1.4–1.5 times greater secondary root growth and 1.2 times greater root and shoot dry weights than Homestead controls. Root:shoot dry weight ratios for the controls again were virtually identical.

Homestead and Newton responded similarly to SBWMV infection and differed only in number of secondary roots and total shoot dry weight. However, the responses of both Homestead and Newton differed from those of SBWMV-susceptible Scout-66 in almost all aspects of root growth (Table 3). SBWMV-infected Newton had fewer secondary roots and slightly lower total shoot dry weight than infected Homestead. However, the 96 and 144% response values in these characteristics for Homestead in the final repetition of the SBWMV study were atypical of observations of those parameters for Homestead in other repetitions. Values from the three prior repetitions for Homestead were close to those obtained for Newton in the final repetition.

Cultivars Homestead, Newton, and Scout-66 all were heavily and similarly colonized by a SBWMV-free isolate of *P. graminis* even though they differ in resistance to SBWMV under field conditions. Plasmodia and zoosporangia of the vector fungus were observed most commonly in or near the zone of root maturation, whereas resting spores were found in the older portions of the roots (Fig. 1).

DISCUSSION

Cultivar responses to virus infection contrasted strongly between the two viruses studied. However, virus infection consistently reduced root growth more than shoot growth and secondary root

Table 2. Effects of soilborne wheat mosaic virus (SBWMV) on growth of 42- to 52-day-old Homestead and Scout-66 winter wheat grown at 15–17 C for 32–42 days after inoculation

	Means						
	Homestead[a]			Scout-66[b]			
Growth parameter	H[c]	I[d]	Resp.[e]	H[c]	I[d]	Resp.[e]	Cont.[f]
Number							
Primary roots	4.10	415	102	4.30	4.30	100	NS
Secondary roots	7.05	4.25	58*	10.60	9.90	93	*
Shoots	7.85	7.05	89	8.30	8.10	98	NS
Length (m)							
Primary root system	2.49	1.35	55*	2.64	2.09	79	*
Secondary root system	1.89	0.39	20*	2.87	2.07	72*	*
Total root length	4.38	1.75	41*	5.52	4.16	74*	*
Volume (ml)							
Primary root system	1.80	0.66	36*	2.19	1.60	72*	*
Secondary root system	1.03	0.19	16*	1.54	0.99	65*	*
Total root volume	2.83	0.85	29*	3.72	2.59	69*	*
Dry weight (mg)							
Primary root system	185.00	76.80	42*	207.00	166.0	77*	*
Secondary root system	94.70	18.00	17*	132.00	97.30	70*	*
Total root dry wt	280.00	94.70	34*	339.00	263.00	75*	*
Total shoot dry wt	383.00	408.00	106	457.00	395.00	86	*
Root:shoot biomass ratio	0.72	0.23	33*	0.73	0.64	88*	*

[a] Susceptible to wheat streak mosaic virus (WSMV), resistant to SBWMV under field conditions.
[b] Susceptible to WSMV, susceptible to SBWMV under field conditions.
[c] H = healthy, mock-inoculated plants.
[d] I = WSMV-infected plants.
[e] Mean response to virus infection as a percentage of uninfected control plant growth. * = Significant ($P < 0.05$) pairwise contrast of virus-infected vs. uninfected plants.
[f] Pairwise contrasts between cultivars for response to virus infection: * = Significant ($P < 0.05$) contrast; NS = nonsignificant contrast.

Table 3. Responses to infection with soilborne wheat mosaic virus (SBWMV) 32 days after inoculation for three cultivars of winter wheat grown 42 days at 15–17 C

	Percentage growth response[a]				
Growth parameter	Homestead[b]	Contrast[c]	Newton[b]	Contrast[c]	Scout[d]
Number					
Primary roots	100	NS	96	NS	104
Secondary roots	96	*	69*	*	103
Shoots	86	NS	77*	*	118
Length					
Primary root system	65*	NS	59*	*	84
Secondary root system	38*	NS	39*	*	72*
Total root length	53*	NS	49*	*	78*
Volume					
Primary root system	42*	NS	38*	*	78
Secondary root system	36*	NS	39*	*	64*
Total root volume	40*	NS	38*	*	72*
Dry weight					
Primary root system	52*	NS	47*	*	83
Secondary root system	42*	NS	43*	*	66*
Total root dry wt	48*	NS	45*	*	76*
Total shoot dry wt	141*	*	112	NS	92
Root:shoot dry wt ratio	35*	NS	40*	*	85

[a] Mean percentage of uninfected control plant growth ($n = 5$).
[b] Resistant to SBWMV under field conditions. * = Significant ($P < 0.05$) pairwise contrast between SBWMV-infected and uninfected plants of that cultivar.
[c] Pairwise contrasts of SBWMV-infected Newton vs. SBWMV-infected Homestead or Scout-66. * = Significant ($P < 0.05$) contrast; NS = nonsignificant contrast.
[d] Susceptible to SBWMV under field conditions. * = Significant ($P < 0.05$) pairwise contrast between SBWMV-infected and uninfected Scout-66 plants.

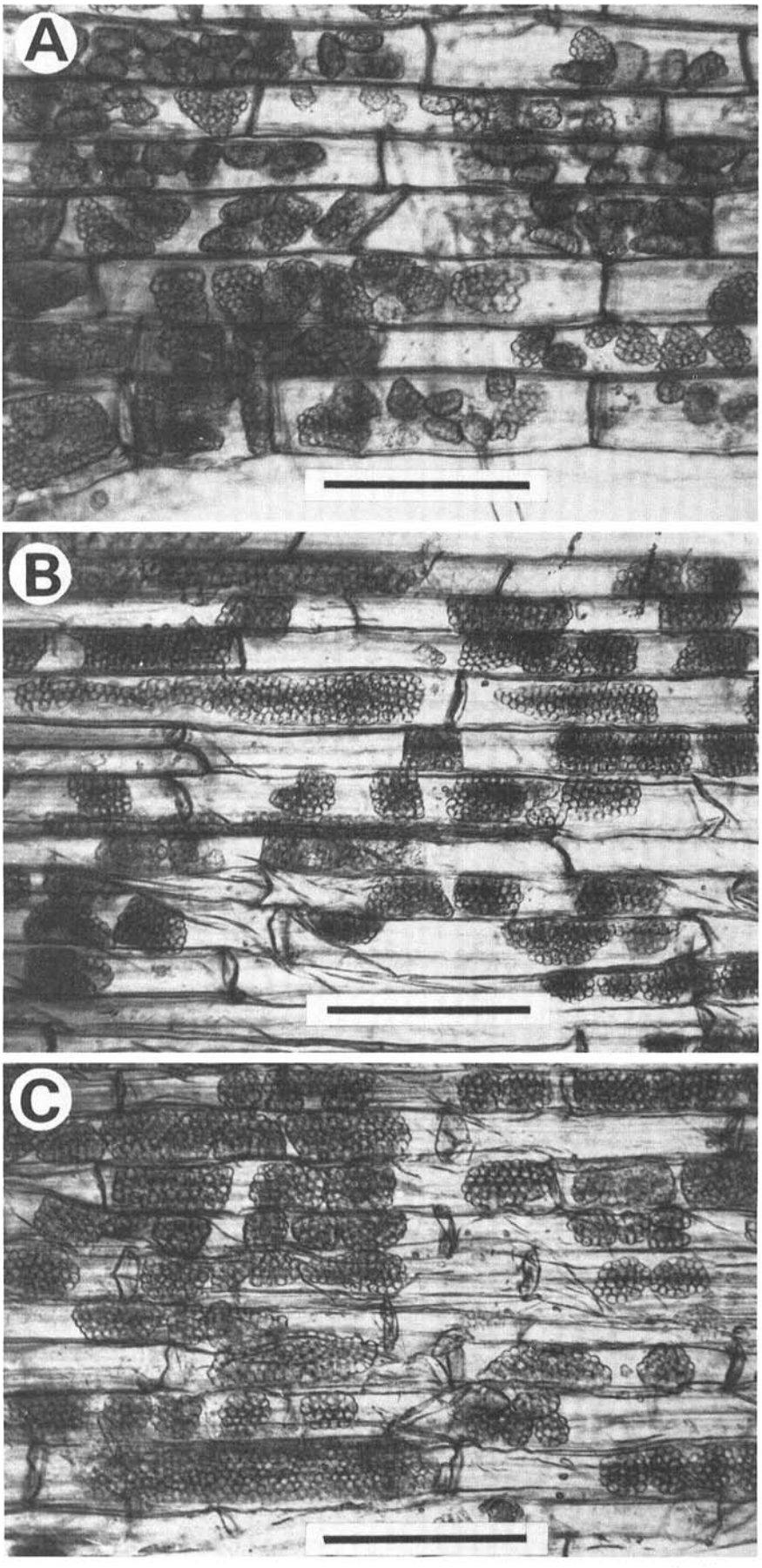

Fig. 1. Roots of 1-mo-old hard red winter wheat seedlings colonized by an isolate of *Polymyxa graminis* free of soilborne wheat mosaic virus (SBWMV). Epidermal peels from secondary root portions containing *P. graminis* resting spores. **(A)** Homestead (resistant to SBWMV in the field), **(B)** Newton (resistant to SBWMV in the field), and **(C)** Scout-66 (susceptible to SBWMV in the field). Bars = 100 μm.

growth more than primary root growth regardless of cultivar, virus type, or virus susceptibility. Cultivars with similar susceptibility to WSMV responded similarly to WSMV infection (Table 1). Cultivars with different susceptibility to SBWMV in the field responded very differently to SBWMV infection (Tables 2 and 3). This would not be unusual except for the inverse relationship between field susceptibility and cultivar response to manual inoculation with SBWMV. SBWMV-susceptible Scout-66 had a longer incubation time and much smaller reductions in root growth than SBWMV-resistant cultivars Homestead and Newton. Direct comparisons between responses to WSMV infection and responses to SBWMV infection for individual cultivars were not feasible because of different experimental conditions for the two studies. However, SBWMV infection reduced both root and shoot growth of Scout-66 less than did WSMV infection, whereas SBWMV infection in Homestead reduced root growth more and shoot growth less than did WSMV infection.

The SBWMV studies were done at constant "low" temperatures of 15–17 C, which differ somewhat from field conditions in the spring. During early spring, the mean daily temperatures are fairly cool, with soil temperatures fluctuating less than air and foliar temperatures. Thus, foliar temperatures are generally warmer in the day and cooler at night than root temperatures at corresponding times under field conditions. Foliar temperatures for plants grown under growth chamber conditions are likely to be much less variable despite a diurnal illumination regime. However, we do not believe such differences in temperature fluctuations for field-grown plants versus growth chamber-grown plants could explain our results.

Viruses are known to retard plant shoot and root growth (10,11,14,20), and reductions in root volume and dry weight range from 15 to 90% (10,14). Virus resistance also can affect symptom expression (10,11,20). Root and shoot growth of virus-infected plants most often correlates directly with resistance and inversely with susceptibility to the virus (10,11), but sometimes, virus susceptibility has little or no correlation with infected plant root or shoot growth (10,20). However, neither an inverse correlation between virus resistance and root growth in infected plants nor a greater effect of virus infection on secondary than on primary root growth has been reported previously.

Knowledge of root system development at inoculation, symptom expression, and harvest in both the WSMV and SBWMV experiments should clarify potential effects of the respective virus on root growth. All plants in both the WSMV and SBWMV experiments had initiated all of their primary roots but none of their

secondary roots by the time of inoculation. Root development at symptom onset was not measured in this study, but Klepper et al (12) provide a correlation diagram for root, leaf, and tiller development in soft white winter wheat from which root development at various growth stages can be estimated. In the WSMV experiments, both Homestead and Scout-66 expressed symptoms at Haun stage 2.6, by which time they should have about two secondary roots. Consequently, between WSMV symptom onset and harvest, infected Homestead and Scout-66 produced one or two secondary roots while their control plants were producing about three and six secondary roots, respectively. In the SBWMV experiments, Homestead and Scout-66 showed foliar symptoms at Haun stages 4.3 and 5.5, respectively, by which time they should have had about six and nine secondary roots, respectively. Because the virus-infected Homestead produced only 4.25 secondary roots by harvest, it is probable that Homestead plants either had their secondary root growth affected before expression of foliar symptoms or else had only four instead of six secondary roots at first symptom expression. Consequently, between symptom onset and harvest in the SBWMV experiments, the control Homestead and Scout-66 produced about three and two secondary roots, respectively, while their SBWMV-infected analogs produced zero and one secondary root, respectively. Since uninfected Homestead produced fewer secondary roots than uninfected Scout-66, secondary roots may grow more slowly in Homestead. Also, because these two cultivars had similar shoot dry weights, Homestead may not require the additional roots to maintain shoot growth under conditions of optimal water availability. However, this may not be true for field soils that contain suboptimal soil moisture.

The differences between the effects of virus infection on primary and secondary root growth are not surprising since secondary roots develop over a longer time period and thus would depend more on plant health (12). But the earlier onset of SBWMV symptoms and the strikingly greater SBWMV-induced growth reduction in SBWMV-resistant wheat than in SBWMV-susceptible wheat were unexpected. This could be due either to more rapid spread of the virus throughout the field-resistant plants or to a greater sensitivity to viral disruption of normal plant meristematic and physiological activities in these plants. Similarly unanticipated were the different effects of SBWMV infection on shoot and root growth for Homestead. This is reflected in the disparate root:shoot dry weight ratios of SBWMV-infected plants, reduced to 33 and 88% of control values for Homestead and Scout-66, respectively. Earlier onset of SBWMV foliar symptoms in Homestead than in Scout-66 could have caused the larger reduction in root:shoot ratios for Homestead than for Scout-66. Earlier expression of foliar symptoms could reduce photosynthetic capacity of the shoots and consequently reduce photosynthate availability for plant growth in general and secondary root growth in particular. Root:shoot ratios reflect relative allocation of photosynthate to the root and shoot systems and are sensitive to environmental factors and plant age (4,6). For example, the ratios increase with deviation from optimum growth temperatures (6) and usually decrease with increased environmental stress or plant age (4). Decreases in these ratios associated with virus infection should have been expected. Others have noted phloem dysfunction associated with infection by the phloem-limited curly-top virus (7,22), but neither SBWMV nor WSMV are phloem-limited as is evidenced by their foliar mosaic symptoms. Directly induced blockage of phloem transport by SBWMV infection is thus unlikely, but virus infection could alter photosynthate distribution through more or less direct effects on other aspects of physiology. Finally, meristematic activity and differentiation could simply be more severely disrupted in the roots than in the shoots by virus infection.

Because of the shorter time required for foliar expression of SBWMV symptoms and the greater reduction of root growth in SBWMV-infected resistant cultivars than in the susceptible cultivar, it is likely that the field resistance exhibited by Homestead and Newton acts at the root level. Potential mechanisms for such field resistance to SBWMV include 1) resistance to zoosporic infection by the vector fungus alone (whether they carry the virus or not), 2) resistance to infection by viruliferous vector zoospores but not virus-free zoospores, 3) tolerance to the virus, 4) resistance to the virus at the cellular level, 5) reduced virus movement in the roots of cultivars with field resistance, and 6) resistance to infection by the wild type of SBWMV RNA species (but not to deletion mutant RNA strains) coupled with the ability of *P. graminis* zoospores to transmit only the wild type and not the deletion mutants of SBWMV. Direct observations argue against mechanism 1, i.e., resistance to the vector fungus alone (Fig. 1). The apparently greater sensitivity to foliar inoculation with SBWMV by cultivars field resistant to SBWMV implies that this field resistance is neither tolerance to the virus nor immunity to the virus at the cellular level (mechanisms 3 and 4). The second, fifth, and sixth alternatives, however, are still possible. Reduced susceptibility to viruliferous vector zoospores could occur if the virus reduced the ability of the zoospore to penetrate the host cell wall or if a root-localized hypersensitive response killed both the viruliferous vector and the infected root cell before the virus could spread to adjacent root cells. Either alternative would allow colonization of resistant cultivar plant roots by non-viruliferous vector zoospores, and a massive virus inoculum such as that introduced by foliar inoculation could potentially overwhelm a hypersensitive response. Further studies are needed to determine whether field resistance to SBWMV is actually due to reduced susceptibility to viruliferous zoospores, a root-localized hypersensitive response, or reduced virus movement in roots of resistant cultivars. Also yet to be determined is whether *P. graminis* zoospores can transmit both the wild type and the deletion mutants of SBWMV or whether they transmit only the wild type RNAs.

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LITERATURE CITED

1. American Type Culture Collection. 1982. Catalog of Strains I. 15th ed. American Type Culture Collection, Rockville, MD. 755 pp.
2. Bohm, W. 1979. Methods of Studying Root Systems. Ecological Studies No. 33. Springer-Verlag, New York. 188 pp.
3. Brakke, M. K., and Rao, A. S. 1967. Maintenance of soil-borne wheat mosaic virus cultures by transfer through root washings. Plant Dis. Rep. 51:1005-1008.
4. Brouwer, R. 1966. Root growth of grasses and cereals. Pages 153-166 in: The Growth of Cereals and Grasses. F. L. Milthorpe and D. J. Ivins, eds. Butterworths, London. 359 pp.
5. Campbell, L. G., Heyne, E. G., Gronau, D. M., and Niblett, C. 1975. Effect of soilborne wheat mosaic virus on wheat yield. Plant Dis. Rep. 59:472-476.
6. Davidson, R. L. 1969. Effect of root/leaf temperature differentials on root/shoot ratios in some pasture grasses and clover. Ann. Bot. 33:561-569.
7. Esau, K. 1957. Phloem degeneration in Graminae affected by barley yellow-dwarf virus. Am. J. Bot. 44:245-251.
8. Haun, J. R. 1973. Visual quantification of wheat development. Agron. J. 65:116-119.
9. Hoagland, D. R., and Arnon, D. I. 1950. The water-culture method for growing plants without soil. Calif. Agric. Exp. Stn. Circ. 347. 32 pp.
10. Kainz, M., and Hendrix, J. W. 1981. Response of cereal roots to barley yellow dwarf virus interaction in a mist culture system. (Abstr.) Phytopathology 71:229.
11. Keeling, B. L. 1982. Effect of soybean mosaic virus on root volume and dry weight of soybean plants. Crop Sci. 22:629-630.
12. Klepper, B., Belford, R. K., and Rickman, R. W. 1984. Root and shoot development in winter wheat. Agron. J. 76:117-122.
13. Kucharek, T. A., and Walker, J. H. 1974. The presence of and damage caused by soil-borne wheat mosaic virus in Florida. Plant Dis. Rep. 58:763-765.
14. Kuhn, C. W. 1965. Symptomatology, host range and effect on yield of a seed-transmitted peanut virus. Phytopathology 55:880-884.
15. Lommel, S. A., and Willis, W. G. 1984. The role of wheat spindle streak and wheat soilborne mosaic virus in an epiphytotic of resistant wheat in Kansas. (Abstr.) Phytopathology 74:844.
16. McKinney, H. H. 1937. Mosaic diseases of wheat and related cereals. U.S. Dep. Agric. Bull. 442. 23 pp.
17. Merkle, O. G., and Smith, E. L. 1983. Inheritance of resistance to soil borne mosaic in wheat. Crop

Sci. 23:1075-1076.
18. Nykaza, S. M., Heyne, E. G., and Niblett, C. L. 1979. Effects of wheat soil-borne mosaic virus on several plant characters of winter wheat. Plant Dis. Rep. 63:594-598.
19. Palmer, L. T., and Brakke, M. K. 1975. Yield reduction in winter wheat infected with soilborne wheat mosaic virus. Plant Dis. Rep. 59:469-471.
20. Pratt, R. G., Ellsbury, M. M., Barnett, O. W., and Knight, W. E. 1982. Interactions of bean yellow mosaic virus and an aphid vector with Phytophthora root diseases in arrowleaf clover. Phytopathology 72:1189-1192.
21. Rao, A. S., and Brakke, M. K. 1970. Dark treatment of wheat inoculated with soil-borne wheat mosaic virus and barley stripe mosaic virus. Phytopathology 60:714-716.
22. Rasa, E. A., and Esau, K. 1961. Anatomical effects of curly top and aster yellows viruses on tomato. Hilgardia 30:469-515.
23. SAS Institite. 1982. User's Guide: Statistics. SAS Institite Inc., Cary, NC. 584 pp.
24. Shirako, Y., and Brakke, M. K. 1984. Two purified RNAs of soil-borne wheat mosaic virus are needed for infection. J. Gen. Virol. 65:119-127.
25. Shurtleff, M. C., Jacobsen, B. J., and Jedlinski, H. 1983. Soilborne mosaic of winter wheat. Report on Plant Diseases No. 102, Coop. Ext. Serv. Univ. Ill. Urbana-Champaign. 4 pp.
26. Steel, R. G. D., and Torrie, J. H. 1980. Principles and Procedures of Statistics. 2nd ed. McGraw-Hill, New York. 633 pp.