

Decline of *Cephalosporium* Stripe by Monoculture of Moderately Resistant Winter Wheat Cultivars

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Contribution 89-160-J of the Kansas Agricultural Experiment Station, Kansas State University, Manhattan.

Accepted for publication 6 June 1989 (submitted for electronic processing).

ABSTRACT

Shefelbine, P. A., and Bockus, W. W. 1989. Decline of *Cephalosporium* stripe by monoculture of moderately resistant winter wheat cultivars. *Phytopathology* 79:1127-1131.

Winter wheat cultivars were grown in monoculture for 3 yr during a *Cephalosporium* stripe epidemic to determine if the rate-reducing phenomenon observed for several polycyclic diseases could be demonstrated for a monocyclic disease. Disease incidence, severity, and yield loss were monitored yearly. The susceptible cultivars Sturdy, Norkan, and Arkan were moderately diseased and exhibited moderate yield loss each year. Disease declined in years 2 and 3 in the moderately resistant cultivars Newton and Plainsman V, such that negligible disease was observed in the final year. Dodge sustained moderate stripe in the first year, but disease declined in the next 2 yr such that Dodge reacted similarly

to Newton and Plainsman V in year 3. When disease incidence and severity were normalized for yearly differences in the environment by expression as a percentage of that observed for Sturdy, incidence dropped 13-14% for Norkan and Arkan, 23% for Plainsman V, but 65 and 78% for Dodge and Newton, respectively. This study indicates that the rate-reducing phenomenon observed for polycyclic diseases exists for monocyclic diseases as well. Instead of disease declining during one season, disease declines over several seasons with monoculture of moderately resistant cultivars. Thus, high levels of resistance are not needed to control *Cephalosporium* stripe.

Additional keywords: *Cephalosporium gramineum*, soilborne disease.

Cephalosporium stripe, incited by the fungus *Cephalosporium gramineum* Nisikado & Ikata (= *Hymenula cerealis* Ell. & Ev.) is a severe vascular disease of winter wheat (*Triticum aestivum* L.) (5,12,21,24,26,27,29). The pathogen is monocyclic (5), inhabits residue (3,6,8,14), is favored by acid soils (2,7,31), and infects through roots (1,19). Control measures for the disease include destruction of infested residue (3,9,15,18), rotation to less susceptible crops (15,18), soil liming (2,31), and late planting (20,30). Crop rotation is often uneconomical or unpopular and residue destruction favors soil erosion. Although liming has reduced *Cephalosporium* stripe incidence, wide applicability of this practice is limited in some areas because of expense and the fact that liming favors take-all, another severe disease of wheat.

Late planting reduces *Cephalosporium* stripe intensity but may also decrease yield, making this control impractical. Thus, additional control measures for the disease are desirable.

Quantitative differences among cultivars in disease development over a single growing season, because of rate-reducing effects, are known for several polycyclic foliar and stem pathogens (11). For the cereal rust diseases, this concept is referred to as "slow-rusting." Pathogen fitness characteristics and host attributes interact, resulting in a reduced rate of disease progression during the growing season. Quantitative effects have been observed for lesion number, lesion size, inoculum production, and latent period. Generally, the area under the disease progress curve is reduced, as is the apparent infection rate (10,13,16,22,23,28). However, resistance of this type has been documented only for polycyclic diseases, such as the rusts, and not for monocyclic diseases, such as *Cephalosporium* stripe.

Monocyclic diseases are characterized by the failure of plants infected during a growing season to produce inoculum capable of infecting adjacent plants during that same season (11). Generally, the pathogen survives between crops, inoculum is produced from previously diseased tissue, and plants are infected. Any factor that reduces inoculum should reduce disease. If winter wheat cultivars possessed resistance that reduced *Cephalosporium* stripe development by affecting pathogen fitness characteristics, such as capability to produce inoculum, the disease could be controlled by cultivation of the resistant cultivar in the same field several years in a row. In this research, we monitored the intensity of *Cephalosporium* stripe over several consecutive growing seasons to determine if the relative resistance of winter wheat cultivars affected disease progression when monoculture was practiced. That goal was to determine if a phenomenon similar to rate-reducing resistance in polycyclic diseases could be demonstrated for a monocyclic disease. A preliminary report has been published (25).

MATERIALS AND METHODS

A field plot for the experiment was established on the Rocky Ford Research Farm, 6 km north of Manhattan, KS. The plot area was a Chase silty clay loam soil and had a history of continuous winter wheat production. Established growing practices for winter wheat in this region had been followed before the start of the experiment, including burning of stubble and deep plowing to eliminate residue. No naturally occurring *Cephalosporium* stripe had been detected in this field in previous years.

Twelve treatment combinations from two factors were arranged in a randomized block design and replicated four times. Factor one was six winter wheat cultivars (Plainsman V, Dodge, Arkan, Newton, Sturdy, and Norkan) differing in susceptibility to *Cephalosporium* stripe. Factor two was inoculation or no inoculation with *C. gramineum* in the first year of the study. Plots of 1.2 × 7.6 m were arranged in a split-plot design, with inoculated and uninoculated treatments as subplots.

Oat kernels colonized by *C. gramineum* were used as inoculum in the first year (1985) only. One hundred grams of autoclaved, colonized oats or autoclaved, noncolonized oats were mixed with 65 g of seed of each cultivar and seeded together 7 cm deep in five rows per plot. No additional artificial inoculum was added for years 2 and 3 (1986 and 1987).

After the first year, all residue above the soil line from each plot was separately shredded with a rotary mower after grain harvest, bagged, and stored in a building. Residue from uninoculated plots was autoclaved at 6.9×10^4 Pa for 24 hr to eliminate possible trace infestation of *C. gramineum* that might have occurred the previous season. One month before planting, all residue from a single plot was uniformly rototilled into the top layer (approximately 10 cm) of soil in an equivalent-sized area in a different location from the previous year. Each cultivar was subsequently seeded into its previous year's residue. Plots were seeded in the last week of September during each year of the study, and standard wheat production practices were followed.

The experiment was repeated following the second growing season by using the residue of the second crop as an inoculum source for the third crop.

Disease was assessed yearly by measuring disease incidence, severity, and yield loss. Disease incidence and severity were assessed for each cultivar when it had completed flowering by examining 50–100 tillers per plot for striping of the top three leaves (4). Disease incidence was the percentage of tillers having at least one leaf striped. Disease severity (DS) was calculated by the formula of Bockus and Sim (4). $DS = (x + 2y + 3z)/n$, where x = number of tillers with only the third leaf down showing stripes, y = number of tillers with the second and third leaves down showing stripes, z = number of tillers with all three top leaves (e.g., flag leaf down) striped, and n = total number of tillers examined. Mature grain was harvested from each plot with a combine, and yield loss was determined by comparing yields of each set of paired plots.

Differences among cultivars in disease incidence, severity, and yield loss were assessed yearly and analyzed by analysis of variance. To remove variation from environmental differences between years, data also were expressed as a percentage of the value in that replication observed for the cultivar Sturdy. For this analysis, the values for Sturdy were excluded from the estimation of experimental error. Additionally, the experiment was analyzed as if it were a split-plot design, with time (years) considered as subplots and cultivars as whole plots.

RESULTS

Disease incidence. During all 3 yr, no *Cephalosporium* stripe was observed in uninoculated plots. Incidence of stripe in infested plots varied among years and cultivars (Table 1). Resistance to stripe was exhibited such that ranking of cultivars according to disease reaction occurred in a predictable order each year. Sturdy sustained the greatest percentage of diseased plants in all years, significantly ($P = 0.05$) more than Norkan and Arkan in 1985 and 1987, but not in 1986. Newton and Plainsman V had the least disease incidence, significantly less than Sturdy, Norkan, and Arkan in each of the 3 yr (Table 1).

The cultivar Dodge exhibited a moderate level of disease incidence in 1985, significantly lower than Sturdy but higher than Newton and Plainsman V. In 1986, only Sturdy differed significantly from Dodge. By 1987, Dodge joined Newton and Plainsman V in having significantly less disease incidence than all other cultivars (Table 1).

Disease severity. Disease severity, like disease incidence, varied with year of measurement and cultivar examined (Table 1). Nevertheless, cultivars reacted consistently, such that ranking of cultivars according to disease reaction occurred in a predictable order each year. Sturdy had the highest or equal to the highest severity each year. Newton and Plainsman V had the least severity, significantly less than that observed for Dodge and Arkan in 1985, but not in 1986. The disease severity of Dodge did not differ significantly from those of Norkan and Arkan in 1985 and 1986 but was lower in 1987. Arkan exhibited a continued moderate level of disease severity in 1987, only significantly less than that

TABLE 1. The influence of monoculture of winter wheat cultivars on percentage disease incidence (DI), disease severity (DS), and yield reduction (YR) caused by *Cephalosporium* stripe

Cultivars	Year 1 (1985)			Year 2 (1986)			Year 3 (1987)			Average	
	DI	DS	YR	DI	DS	YR	DI	DS	YR	DI	DS
Sturdy	64 a ^y	1.5 a	32 a	38 a	1.0 a	... ^z	27 a	0.6 a	16 ab	43 a	1.0 a
Norkan	51 b	1.2 ab	21 ab	24 ab	0.6 ab	...	18 b	0.4 ab	27 a	31 b	0.7 b
Arkan	49 b	0.9 b	14 bc	24 ab	0.6 ab	...	17 b	0.3 b	15 ab	30 b	0.6 b
Dodge	41 b	1.0 b	3 c	13 bcd	0.3 b	...	6 c	0.1 c	7 bcd	20 c	0.5 bc
Newton	24 c	0.5 c	11 bc	8 cd	0.1 b	...	3 c	0.0 c	-2 d	12 d	0.2 c
Plainsman V	20 c	0.3 c	11 bc	5 d	0.1 b	...	6 c	0.1 c	0 cd	10 d	0.2 c
Average	42 x	0.9 m		19 y	0.5 n		13 z	0.3 o			

^y Values in a column, or the average DI or DS in a row, followed by different letters are significantly different according to Fisher's protected least significant difference test ($P = 0.05$).

^z Values not determined.

observed for Sturdy. After the 1985 season, Dodge declined in disease severity, to a level similar to that observed for Newton and Plainsman V (Table 1).

Yield reduction. Yield reduction was variable each year, and significant differences among cultivars occurred infrequently. Yield data for the 1986 season was omitted because of bird damage to the plots before harvest. Sturdy and Norkan sustained the most yield reduction every year. In 1985, Arkan, Newton, and Plainsman V showed a moderate amount of yield reduction, whereas only Arkan had moderate loss in 1987. The small yield reduction exhibited by Dodge in 1985 was exhibited in 1987 by Dodge, Newton, and Plainsman V (Table 1).

Normalization. Normalization of disease measurements for yearly differences in the environment, by expression as a percentage of the values observed for Sturdy, did not change the results dramatically (Table 2). Significant ($P = 0.05$) differences among cultivars each year for disease incidence, disease severity, and yield reduction were very similar to those observed for nonnormalized data. However, normalizing the data allowed for easier visualization of trends since all cultivars were compared to the susceptible cultivar Sturdy.

Norkan and Arkan held their positions relative to Sturdy between years 1 and 2 (Table 2). Dodge exhibited a large decrease in disease incidence, from 65% of Sturdy in year 1 to only 39% in year 2. The disease severities for Dodge showed a similar decline. Newton and Plainsman V also showed declines in disease incidence and severity relative to Sturdy. Furthermore, a similar trend was noted between years 2 and 3, with Dodge declining in disease incidence from 39% of Sturdy to only 23% and declining in severity from 36% to 18% of Sturdy. Newton and Plainsman V seemed to maintain fairly low incidence and severity scores between years 2 and 3, whereas Norkan and Arkan maintained fairly high incidence and severity scores, similar to those in year 1 (Tables 1 and 2).

Cultivar and time effects. There were no significant cultivar \times time interactions. Averaged over the 3 yr of the study, Sturdy had the greatest disease incidence, significantly greater than all other cultivars (Table 1). Norkan and Arkan had a moderate disease incidence, significantly more than the low incidence observed for Newton and Plainsman V and moderately low incidence observed for Dodge. Similarly, Sturdy had the highest disease severity, whereas Newton and Plainsman V had the lowest disease severity. Dodge showed moderate disease severity, significantly less than that of Sturdy but not less than Norkan and Arkan. Normalization of disease incidence measurements for yearly environmental differences did not indicate cultivar and year of measurement interaction. Differences between cultivars for normalized disease incidence and severity (Table 2) were similar to those observed for nonnormalized disease incidence and severity (Table 1).

Disease incidence varied significantly with time (Table 1). Mean disease incidence and severity were greatest in 1985, significantly greater than in all other years. The lowest mean disease incidence and severity occurred in 1987, significantly less than all other years. Normalization of mean disease incidence and severity for

yearly environmental differences resulted in the same pattern of decline with time (Table 2). This decline was probably due to less conducive environments during 1986 and 1987; however, the percentage reduction in disease variables from 1985 to 1987 was highly cultivar-dependent (Table 3). Disease incidence dropped 54–65% for Sturdy, Norkan, and Arkan, 70% for Plainsman V, 85% for Dodge, and 90% for Newton. Similar reductions were seen in disease severity; 63–65% for Sturdy, Norkan, and Arkan, 75% for Plainsman V; 90% for Dodge; and 95% for Newton. Disease decline over time was more pronounced for the moderately resistant cultivars, Newton, Dodge, and Plainsman V.

When disease incidence and severity were normalized for yearly differences in the environment, the percentage reduction from 1985 to 1987 was even more cultivar-dependent (Table 3). Disease incidence dropped 13–14% for Norkan and Arkan, 23% for Plainsman V, but 65 and 78% for Dodge and Newton, respectively. A similar trend was observed for disease severity, which dropped 5–7% for Norkan and Arkan, and 18% for Plainsman V, but 74% for Dodge and 88% for Newton. Disease measurements declined more for the moderately resistant cultivars Newton, Dodge, and Plainsman V.

DISCUSSION

Results indicate that monoculture of cultivars that exhibit moderate levels of resistance to *Cephalosporium* stripe results in a decline in disease over years. The cultivars Newton and Plainsman V, which showed moderate resistance in year 1 as expressed by their incidence and severity scores, declined to even lower levels in year 2, when compared with Sturdy, a susceptible cultivar (Tables 1 and 2). The cultivar Dodge, which showed a moderately susceptible reaction in year 1, declined in year 2 to a moderately resistant reaction. This trend was repeated from year 2 to year 3. These declines were especially noticeable when normalized data were used in an attempt to eliminate environmental effects between years (Tables 2 and 3).

Complete disappearance of disease with monoculture of moderately resistant cultivars did not occur. The cultivars Newton and Plainsman V maintained a low but detectable level of disease

TABLE 3. Winter wheat cultivar effects on reduction in *Cephalosporium* stripe incidence and severity from 1985 to 1987

Variety	Percentage reduction			
	Disease incidence		Disease severity	
Sturdy	54 ^y	0 ^z	63 ^y	0 ^z
Norkan	65	14	63	5
Arkan	65	13	65	7
Dodge	85	65	90	74
Newton	90	78	95	88
Plainsman V	70	23	75	18

^yData not normalized for environmental effects.

^zData normalized for environmental effects by expression as percentage of the value observed for Sturdy.

TABLE 2. The influence of monoculture of winter wheat cultivars on the relative amount of percentage disease incidence (DI), disease severity (DS), and yield reduction (YR) caused by *Cephalosporium* stripe^x

Cultivars	Year 1 (1985)			Year 2 (1986)			Year 3 (1987)			Average	
	DI	DS	YR	DI	DS	YR	DI	DS	YR	DI	DS
Sturdy	100	100	100	100	100	...	100	100	100	100	100
Norkan	80 a ^y	83 a	75 a	76 a	81 a	...	69 a	80 a	134 a	75 a	82 a
Arkan	76 a	60 ab	50 ab	63 a	59 ab	...	66 a	64 a	64 ab	69 a	61 ab
Dodge	65 a	68 a	7 c	39 ab	36 bc	...	23 b	18 b	13 b	42 b	41 bc
Newton	38 b	32 bc	36 abc	22 b	16 c	...	8 b	4 b	–8 b	23 c	18 c
Plainsman V	31 b	23 c	31 bc	12 b	12 c	...	24 b	19 b	–5 b	23 c	18 c
Average	65 x	61 m		52 y	51 o		48 y	48 o			

^xValues are expressed as a percentage of the value observed for the cultivar Sturdy.

^yValues in a column, or the average DI or DS in a row, followed by different letters are significantly different according to Fisher's least significant difference test ($P = 0.05$). Values for Sturdy were excluded from the estimation of experimental error.

^zValues not determined.

LITERATURE CITED

after three consecutive years of planting (Table 1). Although this low level of disease has a negligible effect on yield, it is important epidemiologically. The fungus is still present to infect susceptible genotypes, if they were planted later in the same field.

Cephalosporium stripe development in Kansas has varied with time. In the years preceding 1970, little stripe was observed. By the middle 1970s, however, it had become a major disease in the central wheat region of Kansas. This increase in incidence coincided with a decline in soil pH (2) and the planting of wheat types genetically similar to Scout on the majority of hectareage. Scout types are very susceptible to Cephalosporium stripe (Bockus, unpublished). However, by the middle 1980s, Cephalosporium stripe once again became a minor disease. This decline coincided with the planting of Newton on the majority of hectareage in Kansas. That cultivar was grown on about 75% of the area where Cephalosporium had been a problem. Newton is considered moderately resistant to Cephalosporium stripe in Kansas, and our results indicate that continued cropping of Newton would maintain Cephalosporium stripe at very low levels.

Washington state has had a similar history of stripe development. The disease was important when the highly susceptible cultivar Brevor was planted between 1952 and 1962 but declined with the planting of the Gaines (1963) and Nugaines (1966) wheat types. These cultivars are moderately resistant to Cephalosporium stripe. Beginning in 1976, stripe once again became a major disease in Washington state. This may be due to planting highly susceptible cultivars, such as Hyslop, McDermid, and Stevens, on large areas (17).

Cephalosporium stripe declined with time for all cultivars during the course of this study. The environment during the 1986 and 1987 seasons was less conducive to Cephalosporium stripe than that in 1985. The winters were mild, with little soil freezing and thawing. Nevertheless, the percentage reductions in disease measurements, whether normalized or not for environmental fluctuations, were greater for moderately resistant cultivars than for the susceptible cultivars. Three possibilities may explain a cultivar-dependent drop in disease. It is possible that inoculum production from infested residue could be cultivar controlled. If Newton, Plainsman V, and Dodge limited sporulation from infested residue, the disease would be expected to decline with monoculture. Secondly, if sporulation per unit of infested residue is not cultivar dependent, inoculum density should be a function of the amount of infested residue present. Any reduction in infested residue would lead to disease decline with monoculture. Our results show that Newton, Plainsman V, and Dodge have light to moderate disease incidence and, thus, limit infested residue buildup. Thirdly, an interaction between inoculum density and cultivar reaction to *C. gramineum* could exist. Susceptible cultivars, such as Sturdy, Arkan, and Norkan, could become highly diseased even at low inoculum levels. Conversely, Newton, Plainsman V, and Dodge could become highly diseased only at moderate to high inoculum levels and slightly diseased at low inoculum levels. Reductions in disease variables with monoculture could result from reductions of inoculum densities below some critical point.

We propose that Cephalosporium stripe can be controlled by planting cultivars that exhibit properties similar to those of cultivars possessing rate-reducing resistance used to control the cereal rust diseases. The difference between resistance to a single- and multiple-cycle disease is that resistance is displayed over several seasons of growing the same cultivar instead of during a single season. Also, the observed increases and declines of stripe in Kansas and Washington may be explained to a high degree by the cultivar histories of those regions. Moderately resistant cultivars grown continuously for several years would cause a gradual reduction of the disease to low levels, whereas susceptible cultivars would cause a resurgence of the disease. Furthermore, this phenomenon, which we have quantified for Cephalosporium stripe, may have applicability to other single-cycle diseases as well. High levels of disease resistance are not necessary to provide adequate control, if monoculture of moderately resistant genotypes is practiced.

1. Bailey, J. E., Lockwood, J. L., and Wiese, M. V. 1982. Infection of wheat by *Cephalosporium gramineum* as influenced by freezing of roots. *Phytopathology* 72:1324-1328.
2. Bockus, W. W., and Claassen, M. M. 1985. Effect of lime and sulfur application to low-pH soil on incidence of Cephalosporium stripe in winter wheat. *Plant Dis.* 69:576-578.
3. Bockus, W. W., O'Conner, J. P., and Raymond, P. J. 1983. Effect of residue management method on incidence of Cephalosporium stripe under continuous winter wheat production. *Plant Dis.* 67:1323-1324.
4. Bockus, W. W., and Sim IV, T. 1982. Quantifying Cephalosporium stripe disease severity on winter wheat. *Phytopathology* 72:493-495.
5. Bruehl, G. W. 1957. Cephalosporium stripe disease of wheat. *Phytopathology* 47:641-649.
6. Bruehl, G. W., and Lai, P. 1966. Prior colonization as a factor in the saprophytic survival of several fungi in wheat straw. *Phytopathology* 56:766-768.
7. Bruehl, G. W., and Lai, P. 1968. Influence of soil pH and humidity on survival of *Cephalosporium gramineum* in infested wheat straw. *Can. J. Plant Sci.* 48:245-252.
8. Bruehl, G. W., and Lai, P. 1968. The probable significance of saprophytic colonization of wheat straw in the field by *Cephalosporium gramineum*. *Phytopathology* 58:464-466.
9. Christian, D. G., and Miller, D. P. 1984. Cephalosporium stripe in winter wheat grown after different methods of straw disposal. *Plant Pathol.* 33:605-606.
10. Fry, W. E. 1978. Quantification of general resistance of potato cultivars and fungicide effects for integrated control of potato late blight. *Phytopathology* 68:1650-1653.
11. Fry, W. E. 1982. Principles of Plant Disease Management. Academic Press, New York. 378 pp.
12. Gerdemann, J. W., and Weibel, R. O. 1960. Cephalosporium stripe on small grains in Illinois. *Plant Dis. Rep.* 44:877.
13. Johnson, D. A. 1986. Two components of slow-rusting in asparagus infected with *Puccinia asparagi*. *Phytopathology* 76:208-211.
14. Lai, P., and Bruehl, G. W. 1966. Survival of *Cephalosporium gramineum* in naturally infested wheat straws in soil in the field and in the laboratory. *Phytopathology* 56:213-218.
15. Latin, R. X., Harder, R. W., and Wiese, M. V. 1982. Incidence of Cephalosporium stripe as influenced by winter wheat management practices. *Plant Dis.* 66:229-230.
16. Lee, T. S., and Shaner, G. 1984. Infection process of *Puccinia recondita* in slow- and fast-rusting wheat cultivars. *Phytopathology* 74:1419-1423.
17. Love, C. S., and Bruehl, G. W. 1987. Effect of soil pH on Cephalosporium stripe in wheat. *Plant Dis.* 71:727-731.
18. Mathre, D. E., Dubbs, A. L., and Johnston, R. H. 1977. Biological control of Cephalosporium stripe of winter wheat. *Montana Agric. Exp. Stn., Capsule Infor. Ser.* #13. 4 pp.
19. Mathre, D. E., and Johnston, R. H. 1975. Cephalosporium stripe of winter wheat: Infection processes and host response. *Phytopathology* 65:1244-1249.
20. Raymond, P. J., and Bockus, W. W. 1984. Effect of seeding date of winter wheat on incidence, severity, and yield loss caused by Cephalosporium stripe in Kansas. *Plant Dis.* 68:665-667.
21. Roane, C. W., and Starling, T. M. 1976. Cephalosporium stripe of wheat in Virginia. *Plant Dis. Rep.* 60:345.
22. Shaner, G. 1973. Reduced infectability and inoculum production as factors of slow-mildewing in Knox wheat. *Phytopathology* 63:1307-1311.
23. Shaner, G., and Finney, R. E. 1977. The effect of nitrogen fertilization on the expression of slow-mildewing resistance in Knox wheat. *Phytopathology* 67:1051-1056.
24. Sharp, E. L. 1959. Two previously unreported fungi on cereals in Montana. *Plant Dis. Rep.* 43:12-13.
25. Shefelbine, P. A., and Bockus, W. W. 1987. Decline of Cephalosporium stripe with continuous planting of moderately resistant wheat varieties. (Abstr.) *Phytopathology* 77:1759.
26. Sim IV, T., and Willis, W. G. 1985. Ten years of wheat disease loss estimates in Kansas: 1976-1985. *Ks. St. Univ. Ext. Plant Pathol.* 1 pp.
27. Smith, N. A., Scheffer, R. P., and Ellingboe, A. H. 1966. Cephalosporium stripe of wheat prevalent in Michigan. *Plant Dis. Rep.* 50:190-191.
28. Statler, G. D., and McVey, M. A. 1987. Partial resistance to *Uromyces appendiculatus* in dry edible beans. *Phytopathology* 77:1101-1103.
29. Willis, W. G., and Shively, O. D. 1974. Cephalosporium stripe of winter wheat and barley in Kansas. *Plant Dis. Rep.* 58:566-567.
30. Wiese, M. V., and Ravenscroft, A. V. 1976. Planting date affects

disease development, crop vigor and yield of Michigan winter wheat. Res. Rep. 314, Farm Science. Michigan St. Univ. Agric. Exp. Stn. 6 pp.

31. Wiese, M. V., and Ravenscroft, A. V. 1978. Cephalosporium stripe decline in a wheat monoculture. Plant Dis. Rep. 62:721.723.