Control of Cephalosporium Stripe of Winter Wheat by Liming

TIMOTHY D. MURRAY, Associate Professor, C. C. WALTER, Agricultural Research Technologist, and J. C. ANDEREGG, Former Postdoctoral Research Associate, Department of Plant Pathology, Washington State University, Pullman 99164-6430

ABSTRACT

Murray, T. D., Walter, C. C., and Anderegg, J. C. 1992. Control of Cephalosporium stripe of winter wheat by liming. Plant Dis. 76:282-286.

In two out of 4 yr of field testing, the incidence of Cephalosporium stripe (percent infected stems), caused by *Cephalosporium gramineum*, decreased significantly ($P = 0.05$) when calcium hydroxide was added to increase soil pH from 5.1-5.3 to >6.0, and increased significantly when sulfuric acid was added to lower soil pH to 4.5; the relationship was linear in both years. In a third year, there was a nearly significant ($P = 0.07$) linear trend for decreasing incidence of disease with increasing soil pH. Disease severity, which reflects the extent of suscept colonization, was not influenced by soil pH or cultivar in any year. Grain yield and test weight increased significantly with increasing soil pH in three out of the 4 yr. The relationship of yield and test weight to soil pH was significantly linear in 2 yr and cubic in the third year. Cultivars differed significantly for disease incidence, yield, and test weight, but interactions between soil pH and cultivar for disease incidence and yield were not significant in any year. A significant interaction between soil pH and cultivar for test weight occurred in one out of 3 yr. Liming for control of Cephalosporium stripe will probably be most valuable in years when root wounding resulting from frozen soil is relatively minor.

Cephalosporium stripe, caused by *Cephalosporium gramineum* Nisikado & Ikata (sporodochial stage = *Hymenula cerealis* Ellis & Everh.), is a wilt disease of winter wheat (*Triticum aestivum* L. em. Thell.). This disease occurs in many parts of the United States including the inland Pacific Northwest (eastern Washington, northeastern Oregon, and northern Idaho). The pathogen is soilborne and survives as mycelium in parasitically colonized host residue in soil (6,8). During the autumn in the inland Pacific Northwest when temperatures decrease and rainfall begins, single-celled conidia are produced on colonized residue positioned on or near the soil surface and liberated into the soil where they serve as the primary inoculum. After infection, the pathogen colonizes the xylem and systemically invades the plant, resulting in premature death of the suscept (5,6,27). The yield of infected plants may be as low as 20% or less than that of comparable healthy plants when conditions are favorable for disease development and the pathogen colonizes the entire plant (11,20).

Cephalosporium stripe can be controlled by: 1) delaying seeding in the autumn; 2) planting a susceptible crop (winter wheat or winter barley) only 1 yr in three; 3) removing or destroying infested residue with deep plowing or burning; and 4) planting resistant cultivars (3,6,12,13,22,29). Delayed seeding results in plants with smaller root systems that are less susceptible to winter root injury and, therefore, less likely to be infected (28). Crop rotations and destruction of infested residue effectively reduce inoculum density and, therefore, disease incidence. Currently, cultivars adapted to the inland Pacific Northwest with high levels of resistance are not available, long crop rotations are not always economically feasible, and delayed seeding and residue burning are discouraged in the U.S. Pacific Northwest because of the increased potential for soil erosion. Thus,

Plant Pathology New Series 0100, College of Agriculture and Home Economics Research Center, Project 0670, Washington State University, Pullman 99164-6240.

Accepted for publication 9 September 1991 (submitted for electronic processing).

other methods of disease control are sought.

Bockus and Claassen (2) demonstrated in Kansas that Cephalosporium stripe decreases after application of lime to soil. Raising soil pH from 4.8 to 6.5 with a one-time application of lime reduced disease incidence from 36 to 22% in the first season and from 30 to 6% in the second season after liming. The yield response resulting from control of Cephalosporium stripe could not be determined because take-all, caused by *Gaeumannomyces graminis* var. *tritici*, increased significantly in the lime-amended soil and confounded the yield data.

Subsequent work in Washington by Love and Bruehl (15) and Anderegg and Murray (1) in the greenhouse demonstrated that Cephalosporium stripe can increase fivefold or more as soil pH decreases from 7.5 to 4.5. These results have significant implications for eastern Washington and northern Idaho because, by 1984, 67% of the fields in the region had soil pH <5.6 and only 6% had soil pH >6.4 (19). The increased soil acidity is associated with long-time use of ammonium-based nitrogen fertilizers (19).

The objectives of this study were to determine whether Cephalosporium stripe responds to soil pH in the field as in greenhouse studies and if there is a concomitant effect on yield.

MATERIALS AND METHODS

Plot sites, cultural practices, and experimental design. Four experiments were conducted at the Washington State University Plant Pathology Farm, Pullman, during the crop years of 1985–86, 1986–87, 1987–88, and 1988–89 (sown in the autumn of one year and harvested in the summer of the next year). These four crop years hereafter are referred to as 1986, 1987, 1988, and 1989, respectively. Winter wheat was grown in alternate years with an intervening year of fallow on each of two plot sites located approximately 30 m apart; both sites were on Thatuna silt loam (fine-silty, mixed, mesic Xeric Argialboll). Site 1 (knoll) had a native (before amendment) pH of 5.3 (1:2, w/v, 0.01 M $CaCl_2$), whereas site 2 (flat) had a native pH of 5.1 in the top 15 cm of soil.

Each experiment was organized as a randomized complete block, split-plot design, and divided into four (site 2) or six (site 1) blocks corresponding to replicates. Soil pH was the main-plot factor and cultivar was the subplot factor.

In 1986 and 1987, wheat was seeded in four-row plots (1.8 × 5.5 m with 0.4 m between rows) with a deep-furrow grain drill with John Deere HZ openers and split-packer wheels. In 1988 and 1989, wheat was seeded in eight-row plots (2.4 × 4.9 m with 0.3 m between rows) with a one-pass drill (McGregor Co., Colfax, WA) with double-disc openers (ACRA-PLANT, Garden City, KS). The seeding rates corresponded to 78.4 kg ha^{-1} (100 seed weight = 4.0 g) in 1986 and 1987, and 89.6 kg ha^{-1} in 1988 and 1989; however, the same number of seeds was planted in each subplot to account for differences in weight of seed among cultivars.

Seeding dates ranged from 9 to 12 September, which is 1 or 2 wk early relative to the production area in all 4 yr. The cultivars Lewjain (tolerant), Nugaines (tolerant), Daws (tolerant), and breeding line Selection 101 (highly susceptible) (CI 13438) were seeded in 1986; Brevor (highly susceptible), Nugaines, and Daws were seeded in 1987; and Daws and Stephens (highly susceptible) in 1988 and 1989 (9).

Soil fertility was determined by sampling each plot to a depth of 180 cm during the summer before planting and adding enough dry fertilizer (containing N, P, and S) immediately before planting in 1986 and 1987, or liquid fertilizer (also containing N, P, and S) 7.5 cm below each seed row at planting in 1987 and 1988, to satisfy crop needs for the estimated yield potential, which was based on the available water in the 120-cm profile plus anticipated rainfall. Chemical weed control was consistent with local commercial practices. Oat kernels (approximately 200 kg ha^{-1}) colonized by *C. gramineum* (9) and scattered over the plot site in October after planting served as a source of inoculum.

Soil pH adjustment. The pH of main plots within replicates was determined before planting in July or August by sampling the top 15 cm of soil with a 2.5-cm probe. Cores (10–15 per plot) were bulked, mixed, oven-dried (55 C), and passed through a sieve with 2-mm openings before pH was measured in 0.01 M $CaCl_2$ (1:2, w/v). Amendments to alter the native soil pH were made during the summer before the initial sowing in each plot. Adjustments to increase acidity of the soil were made by adding technical grade H_2SO_4 to achieve a pH 4.5. Adjustments to increase alkalinity of the soil were made by adding construction grade $Ca(OH)_2$ powder ($CaCO_3$ equivalent = 136) to achieve soil pH values of 5.5, 6.5, and 7.5. Amendments were incorporated by disking to a depth of 10 cm in 1986 and 15 cm in 1987, after which the plots were irrigated to ensure reaction of the amendments with the soil. Gypsum ($CaSO_4$) was added to plots receiving $Ca(OH)_2$ to balance the addition of sulfate added as H_2SO_4 (1). Soil samples were taken at intervals after amendment to monitor changes in soil pH.

The amount of H_2SO_4 or $Ca(OH)_2$ needed to raise or lower pH to the target value was estimated by extrapolation from a pH adjustment curve (1). The curve was constructed by adding varying amounts of 0.167 N H_2SO_4 or dry $Ca(OH)_2$ to 0.5-kg lots of soil (removed from the 0–15 cm layer of the plots) in plastic bags with a constant total volume of water and mixing thoroughly. Soil was mixed three more times corresponding, respectively, to 2, 4, and 7 days, after which samples were removed for pH determination.

Disease and yield determination. Disease incidence and severity were determined in June (postanthesis) each year by removing 0.5-m sections of row from each subplot and rating individual stems with heads for Cephalosporium stripe with a 0–5 scale; where 5 = prematurely killed stems or those having a stripe extending to the head, 4 = stems with stripes in the flag leaf, 3 = stems with stripes in the leaf subtending the flag leaf, 2 = stems with stripes in the second leaf down, 1 = stems with stripes in the third leaf down, and 0 = stems with no symptoms in the upper four leaves. Disease incidence was calculated as the percentage of tillers with symptoms in the uppermost four leaves or head, whereas disease severity, which represented the extent of suscept colonization, was the mean rating of the symptomatic tillers (32).

Yield in 1986 and 1987 was determined by harvesting the center two rows of each plot and threshing the bundles in a stationary bundle thresher. In 1988 and 1989, a 1.5-m wide swath (five rows) was harvested from the center of each plot using a Wintersteiger Nurserymaster plot combine. Test weight was determined on 0.55-L samples after chaff and debris were removed from the grain.

Statistical analysis. Data for soil pH, disease incidence (transformed with the arc sine square root function), disease severity, yield, and test weight were subjected to analysis of variance. Significances of response curves for pH were determined by partitioning the sums of squares for pH into linear, quadratic, and cubic components (14). Fisher's least significant difference was used to differentiate cultivars when appropriate.

RESULTS

1986. Native soil pH (0–15 cm) did not differ significantly ($P = 0.05$) among replicates or target pH levels on 26 July 1985 before soil amendments were added. By 28 October (7 wk postplant), the target levels for pH 4.5 and 5.5 were significantly ($P < 0.05$) different from each other and from pH 6.5 and 7.5, which were not significantly different from each other (Fig. 1). Similar relationships among target pH levels were still apparent in March and October 1986.

The incidence of Cephalosporium stripe averaged across soil pH and cultivars was low (11.7%) and tended to decrease with increasing soil pH (Fig. 2A); however, the linear trend for disease incidence across soil pH (measured in October) was significant only at $P = 0.07$.

The severity of Cephalosporium stripe (as opposed to incidence) was moderate, averaging 2.7, but was not significantly affected by soil pH or cultivar. Yield (Fig. 2B) and test weight increased with increasing soil pH, and the relationship with soil pH was significantly ($P < 0.05$) cubic for both parameters. There was a highly significant ($P < 0.001$) linear regression of grain yield on disease incidence.

Cultivars differed significantly for disease incidence and test weight ($P < 0.001$ for both) but not yield. The incidence of disease and test weight for Lewjain, Daws, Nugaines, and Selection 101 was, respectively, 20.0, 19.1, 13.7, and 11.5% infected stems (LSD [$P = 0.05$] = 7.0), and 784.7, 771.3, 800.2, and 781.7 kg m^{-3} (LSD [$P = 0.05$] = 5.0). The interaction between pH and cultivar was not significant for disease incidence, yield, or test weight.

1987. Native soil pH (15 cm depth) did not differ significantly among target levels or replicates on 24 August 1986, before the addition of amendments. However, by 21 October 1986 (6 wk postplant), all target pH levels were significantly different from each other and remained so through April 1987 (Fig. 3).

There were significant linear relationships between increasing soil pH and decreasing incidence of Cephalosporium stripe ($P < 0.05$; Fig. 4), increasing grain yield ($P < 0.05$; Fig. 5), and increasing test weight ($P < 0.001$). The linear regression of yield on disease incidence was significant only at $P = 0.10$. Disease severity was relatively high, averaging 3.7, but was not influenced by soil pH or cultivar.

The overall incidence of disease was low (9.4%), and differences among cultivars were not significantly ($P < 0.05$) different. Cultivars differed significantly ($P < 0.001$) for grain yield and test weight, with Daws and Nugaines having the greatest yield and test weight (6,685.6 and 6,660.4 kg ha^{-1}, and 799.5 and 797.8 kg m^{-3}, respectively) compared with Brevor, which had the least (3,496.6 kg ha^{-1} and 774.9 kg m^{-3}). Interactions between soil pH and cultivar for disease incidence, yield, or test weight were not significant.

1988. Soil pH values (sampled 20 July 1987) for main plots established 2 yr earlier (summer of 1985) differed from the original target levels, especially at pH 6.5–7.5, where deviations were greatest. Lime was added to some plots on 18 August to raise pH, and by 25 February 1988 soil pH for the target levels of 4.5, 5.5, 6.5, and 7.5 were 5.7, 5.9, 6.2, and 6.7, respectively. Thereafter, soil pH in main plots remained stable (did not differ significantly from soil pH determined 25 February) through 30 June 1988.

Soil pH had no significant effect on incidence of Cephalosporium stripe,

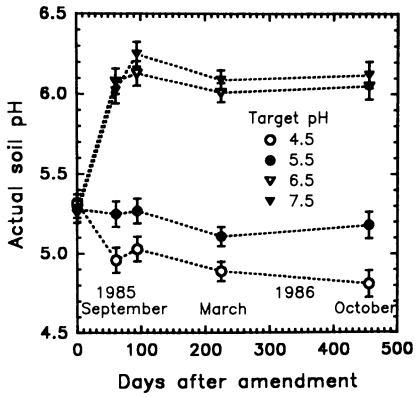

Fig. 1. Actual values of soil pH for the target levels of 4.5, 5.5, 6.5, and 7.5 at intervals after adding either H_2SO_4 (pH 4.5) or $CaOH_2$ (pH 5.5, 6.5, and 7.5) to soil on 26 July 1985 (0 days). Sample dates, respectively, are 25 September 1985, 28 October 1985, 7 March 1986, and 24 October 1986. Bars represent ± the standard error of the mean.

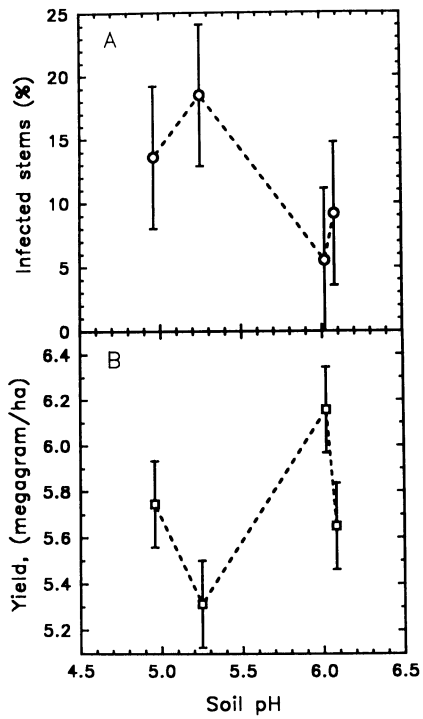

Fig. 2. Disease incidence **(A)** and yield **(B)** of winter wheat grown in plots having different soil pH values at the Washington State University Plant Pathology farm, Pullman, in 1986. The relationship between soil pH and disease incidence was not significant ($P = 0.07$), but the relationship between soil pH and yield was significantly ($P < 0.05$) cubic and described by the equation $Y = 8{,}994.0 - 4{,}760.1X + 843.9X^2 - 49.6X^3$ ($r^2 = 0.71$). Bars represent ± the standard error of the mean.

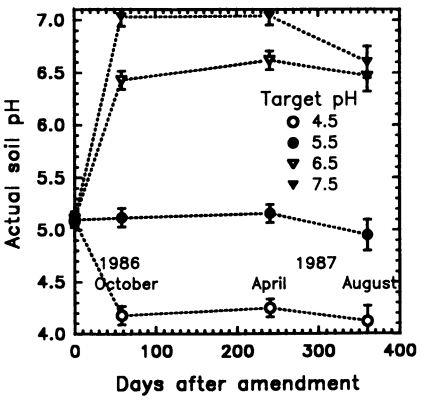

Fig. 3. Actual values of soil pH for the target levels of 4.5, 5.5, 6.5, and 7.5 at intervals after adding either H_2SO_4 (pH 4.5) or $CaOH_2$ (pH 5.5, 6.5, and 7.5) to soil on 24 August 1986 (0 days). Sample dates were 21 October 1986, 21 April 1987, and 19 August 1987. Bars represent ± the standard error of the mean.

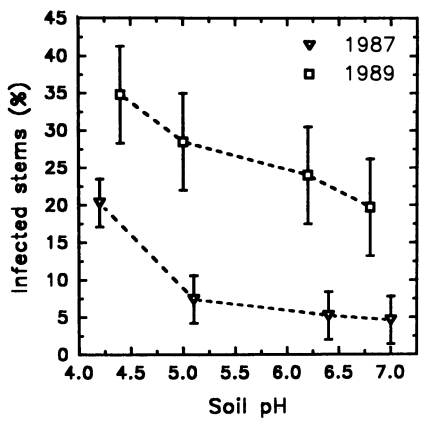

Fig. 4. Incidence of Cephalosporium stripe of winter wheat grown in plots having different soil pH values at the Washington State University Plant Pathology farm, Pullman, in 1987 and 1989. The relationship between soil pH and disease incidence was significantly ($P < 0.05$) linear in both years and described by the equations $Y = 38.2 - 5.1X$ (r^2=0.76) and $Y = 49.4 - 3.9X$ ($r^2 = 0.78$), for 1987 and 1989, respectively. Bars represent ± the standard error of the mean.

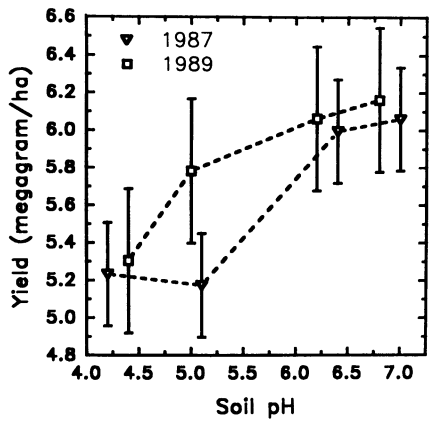

Fig. 5. Yield of winter wheat grown in plots having different soil pH values and inoculated with *Cephalosporium gramineum* at the Washington State University Plant Pathology farm, Pullman, in 1987 and 1989. The relationship between soil pH and yield was significantly ($P < 0.05$) linear in both years and described by the equations $Y = 3{,}217.0 + 360.5X$ ($r^2 = 0.85$) and $Y = 4{,}607.3 + 208.5X$ ($r^2 = 0.91$) in 1987 and 1989, respectively. Bars represent ± the standard error of the mean.

grain yield, or test weight, although disease incidence tended to decrease (41.0, 37.2, 40.3, and 29.3% infected stems) and yield tended to increase (5,222.7, 5,302.8, 5,767.5, and 5,791.1 kg ha^{-1}) with increasing soil pH (5.7, 5.9, 6.2, and 6.7, respectively). Disease severity was high, averaging 4.0, but neither soil pH nor cultivar had a significant effect.

Cultivars differed significantly for disease incidence, yield, and test weight ($P < 0.001$ for all); Daws and Stephens had, respectively, 16 and 57.8% infected stems, grain yields of 6,656.0 and 4,384.7 kg ha^{-1}, and test weights of 760.5 and 700.7 kg m^{-3}. There was a highly significant ($P < 0.001$) linear regression of yield on disease incidence.

1989. Soil pH for the target levels of 4.5, 5.5, 6.5, and 7.5 (amendments applied in the summer 1986) were 4.4, 5.0, 6.2, and 6.8, respectively, on 14 October 1988; all were significantly (LSD [$P = 0.05$] = 0.3) different from each other. The relationship between soil pH and disease incidence (Fig. 4) was significantly ($P < 0.05$) linear, as was the effect of soil pH on grain yield (Fig. 5). Soil pH had a significant effect on test weight, but there was a significant interaction between soil pH and cultivar resulting from a quantitatively larger response of Stephens ($Y = 675.8 + 11.6X$; $r^2 = 0.86$) than Daws ($Y = 742.7 + 6.4X$; $r^2 = 0.70$) to changes in soil pH. Disease severity was relatively high, averaging 3.5, but was not influenced by soil pH or cultivar. There was a highly significant ($P < 0.001$) linear regression of yield on disease incidence.

Cultivars differed significantly ($P < 0.001$) for disease incidence and yield, averaging 13.2 and 40.3% infected stems and 6,272.4 and 5,376.8 kg ha^{-1} for Daws and Stephens, respectively. The interaction of soil pH with cultivar was not significant for disease incidence or yield.

DISCUSSION

The incidence of Cephalosporium stripe was reduced significantly by liming to raise soil pH in two of the 4 yr (1987 and 1989) (Fig. 4) of this study, and grain yield (Figs. 2B, 5) and test weight increased significantly after liming in three of the 4 yr (1986, 1987, and 1989). There were significant linear regressions for increasing yield with decreasing disease incidence in 1986, 1988, and 1989, which indicated that Cephalosporium stripe was a yield-limiting factor in those years. Based on these results, it is apparent that Cephalosporium stripe can be controlled and yields increased by raising soil pH with lime, even when disease incidence is relatively low.

The lack of a significant response of Cephalosporium stripe to soil pH in 1986 and 1988 is probably related to the inherently variable nature of this disease and interactions with the environment that are not well understood (9,20). In 1986, there was a near significant ($P = 0.07$) linear trend of decreasing disease incidence with increasing soil pH, even though disease incidence was relatively low. In 1988, when environmental conditions were more conducive to disease development, the incidence of Cephalosporium stripe averaged 37%, the highest incidence in this study, yet soil pH did not affect disease incidence. There are two explanations for this apparently anomalous result. First, is the relatively narrow range of soil pH (5.7–6.7) across the target pH values in that year, which did not allow significant trends to be detected. This trend was also apparent in other years, when the change in incidence of Cephalosporium stripe was relatively small as soil pH increased from 5.7 to 6.7 (Fig. 4). The second explanation is probably related to the infection process, which is generally thought to occur after root wounding, especially wounding associated with soil freezing (7,8,20,23, 28). In the greenhouse studies by Love and Bruehl (15), Anderegg and Murray (1), and Specht and Murray (31), high incidences of Cephalosporium stripe developed when plants were exposed to acid but not frozen soil (and presumably not wounded). In another study (32), however, Cephalosporium stripe developed independently of soil pH when roots were wounded with a knife before inoculation. It appears, therefore, that soil pH has no effect on the incidence of Cephalosporium stripe when significant root wounding occurs, either by frozen soil in the field or by cutting with a knife in the greenhouse. Thus, soil pH may have the greatest influence on Cephalosporium stripe under field conditions in years when root injury is relatively minor.

Another factor related to the variable response of Cephalosporium stripe to soil pH under field conditions is the relatively small volume of soil affected by the amendments compared to the large volume of soil infested by the pathogen and occupied by host roots. In greenhouse studies (1,16,32,33), the entire soil volume in contact with plant roots and pathogen inoculum is at the same pH. In 1986, 1,561 and 3,320 kg of lime per hectare was applied for target pH levels of 6.5 and 7.5, respectively, but it was apparent after sampling that a soil pH gradient existed with depth of sample. Changes in pH in response to amendments were greatest in the 0–7.5 cm depth, and diminished in the 7.5–15 cm depth. Below 30 cm, the pH of Palouse-area soils is nearly that of the virgin soil (20). This gradient was reduced in 1987 by applying more lime (5,080 and 12,000 kg ha^{-1} for pH 6.5 and 7.5, respectively) and increasing the depth of incorporation (15 cm). Within a season, soil pH remained relatively stable among plots; however, soil pH changed over the 2-yr rotation cycle. We used powdered $Ca(OH)_2$ because it reacts more quickly than coarser materials, or those containing $MgCO_3$ or $CaCO_3$ (33). On the other hand, use of liming materials that include $MgCO_3$ and/or $CaCO_3$ of mixed particle size may provide more stable soil pH over time, because these materials would react more slowly than $Ca(OH)_2$.

In contrast to our previous work (1) in the greenhouse, disease severity was not significantly affected by soil pH or cultivar in any of the 4 yr of this study. This is probably due to the inclusion of only symptomatic stems in the disease severity calculation. Previously, both symptomatic and asymptomatic stems were included in the calculation, which resulted in a biasing of the severity rating by disease incidence (32). In light of these data, the severity rating used in these studies, which reflects the extent of host colonization, may not be related to disease resistance as previously believed (1,4). Limiting colonization of *C. gramineum* after infection is reportedly associated with resistance (21,24,25); however, the methods used to evaluate disease severity in these reports differs from ours. It appears that disease severity measured in this study (which varied from 2.7 in 1986 to 4.0 in 1988) may be more reflective of conditions during the autumn and winter when infection occurs. Specht and Murray (32) demonstrated that disease severity increased linearly with $\log_{10}$ increases in inoculum density in both root-wounded and unwounded plants, but was not influenced by soil pH. Presumably, disease severity would be high in years when inoculum density was high, which could result from favorable environmental conditions (temperature and rainfall) or cultural practices (short rotations, abundant infested residue, or early seeding). This hypothesis warrants further study, however.

Mahler and McDole (17) and Mahler (16) demonstrated 9–16% and 10–28% increases in winter wheat yields, respectively, after the application of 2,200–4,400 kg of lime per hectare. However, they did not account for potential effects of plant disease on their yield responses. We found similar yield increases in both 1987 (15.8%) and 1989 (16.2%) in response to increased soil pH (Fig. 4). Mahler and McDole (18) also calculated "minimum acceptable pH values," below which yield of winter wheat would be adversely affected. This value ranged from pH 5.2 to 5.4, depending on cultivar. Interestingly, this value agrees closely with the pH below which Cephalosporium stripe increased most dramatically in this (Figs. 2A, 4) and previous greenhouse studies (1).

Aluminum toxicity is often used to explain the decreased yield of crops grown on acid soils (10,30). This appears to be an unlikely cause of decreased yields of winter wheat in the acid Palouse

soils because the base saturation ratio of the cation exchange capacity is always greater than 80% (18), and because exchangeable aluminum has not been detected in these soils (18; T. D. Murray, *unpublished*). Acidification of soils in the Palouse region is attributable largely to the use of ammonium-based nitrogen fertilizers (19), which results in an increase in H^+ ions. How the increase in H^+ ions interacts with wheat plants and the pathogen to favor Cephalosporium stripe is unknown. Production of inoculum by *C. gramineum* on infested residue, soil inoculum density, and survival of conidia all are greater in acid than in neutral or alkaline soils (26,31), but the magnitude of these responses does not appear to be great enough to account for the observed increase in Cephalosporium stripe in acid soils (32). Because disease incidence, but not severity, was greater in acid than neutral or alkaline soils in this study, and because soil pH had no influence on disease incidence or severity in root-wounded plants (32), it appears that soil pH affects initial penetration and establishment of *C. gramineum* in the plant, but has no affect on subsequent colonization.

ACKNOWLEDGMENTS

Partial financial support of the Washington State Wheat Commission, the STEEP project, and the O. A. Vogel Wheat Research Fund is gratefully acknowledged.

LITERATURE CITED

1. Anderegg, J. C., and Murray, T. D. 1988. Influence of soil matric potential and soil pH on Cephalosporium stripe of winter wheat in the greenhouse. Plant Dis. 72:1011-1016.
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'Connor, 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, T., IV. 1982. Quantifying Cephalosporium stripe disease severity on winter wheat. Phytopathology 72:493-495.
5. Bruehl, G. W. 1956. Prematurity blight phase of Cephalosporium stripe disease of wheat. Plant Dis. Rep. 40:237-241.
6. Bruehl, G. W. 1957. Cephalosporium stripe disease of wheat. Phytopathology 47:641-649.
7. Bruehl, G. W. 1968. Ecology of Cephalosporium stripe disease of winter wheat in Washington. Plant Dis. Rep. 52:590-594.
8. Bruehl, G. W., Lai, P., and Huisman, O. 1964. Isolation of *Cephalosporium gramineum* from buried, naturally infested host debris. Phytopathology 54:1035-1036.
9. Bruehl, G. W., Murray, T. D., and Allan, R. E. 1986. Resistance of winter wheats to Cephalosporium stripe in the field. Plant Dis. 70:314-316.
10. Foy, C. D. 1984. Physiological effects of hydrogen, aluminum, and manganese toxicities in acid soil. Pages 57-97 in: Soil Acidity and Liming, 2nd ed. F. Adams, ed. ASA-CSA-SSSA Inc., Madison, WI.
11. Johnston, R. H., and Mathre, D. E. 1972. Effect of infection by *Cephalosporium gramineum* on winter wheat. Crop Sci. 12:817-819.
12. 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.
13. 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.
14. Little, T. M., and Hills, F. J. 1978. Agricultural Experimentation. Design and Analysis. John Wiley & Sons, New York. 350 pp.
15. Love, C. S., and Bruehl, G. W. 1987. Effect of soil pH on Cephalosporium stripe in wheat. Plant Dis. 71:727-731.
16. Mahler, R. L. 1986. Evaluation of soil pH manipulation on crop production in northern Idaho. Commun. Soil Sci. Plant Anal. 17:905-919.
17. Mahler, R. L., Halvorson, A. R., and Koehler, F. E. 1985. Long-term acidification of farmland in northern Idaho and eastern Washington. Commun. Soil Sci. Plant Anal. 16:83-95.
18. Mahler, R. L., and McDole, R. E. 1985. The influence of lime and phosphorus on crop production in northern Idaho. Commun. Soil Sci. Plant Anal. 16:485-499.
19. Mahler, R. L., and McDole, R. E. 1987. Effect of soil pH on crop yield in northern Idaho. Agron. J. 79:751-755.
20. Martin, J. M., Johnston, R. H., and Mathre, D. E. 1989. Factors affecting the severity of Cephalosporium stripe of winter wheat. Can. J. Plant Pathol. 11:361-367.
21. Martin, J. M., Mathre, D. E., and Johnston, R. H. 1983. Genetic variation for reaction to Cephalosporium gramineum in four crosses of winter wheat. Can. J. Plant Sci. 63:623-630.
22. Mathre, D. E., Dubbs, A. L., and Johnston, R. H. 1977. Biological Control of Cephalosporium Stripe of Winter Wheat. Capsule Information Series No. 13. Montana Agric. Exp. Stn., Bozeman. 4 pp.
23. Mathre, D. E., and Johnston, R. H. 1975. Cephalosporium stripe of winter wheat: Infection processes and host response. Phytopathology 65:1244-1249.
24. Mathre, D. E., and Johnston, R. H. 1990. A crown barrier related to *Cephalosporium gramineum* stripe resistance in wheat relatives. Can. J. Bot. 68:1511-1514.
25. Morton, J. B., and Mathre, D. E. 1980. Identification of resistance to Cephalosporium stripe in winter wheat. Phytopathology 70:812-817.
26. Murray, T. D., and Walter, C. C. 1991. Influence of pH and matric potential on sporulation of *Cephalosporium gramineum*. Phytopathology 81:79-84.
27. Nisikado, Y., Matsumoto, H., and Yamauti, K. 1934. Studies on a new *Cephalosporium*, which causes the stripe disease of wheat. Berichte d. Ohara Inst. f. Landow. Forsch. 6:275-306.
28. Pool, R. A. F., and Sharp, E. L. 1969. Some environmental and cultural factors affecting Cephalosporium stripe of winter wheat. Plant Dis. Rep. 53:898-902.
29. 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.
30. Ritchie, G. S. P. 1989. The chemical behaviour of aluminum, hydrogen and manganese in acid soils. Pages 1-60 in: Soil Acidity and Plant Growth. A. D. Robson, ed. Academic Press, Sydney.
31. Specht, L. P., and Murray, T. D. 1989. Sporulation and survival of conidia of *Cephalosporium gramineum* as influenced by soil pH, soil matric potential, and soil fumigation. Phytopathology 79:787-793.
32. Specht, L. P., and Murray, T. D. 1990. Effects of root-wounding and inoculum density on Cephalosporium stripe in winter wheat. Phytopathology 80:1108-1114.
33. Tisdale, S. L., and Nelson, W. L. 1975. Soil Fertility and Fertilizers. 3rd ed. Macmillan Publishing Co., New York. 694 pp.