Preemergence Herbicides and the Severity of Leaf Spot Caused by *Drechslera sorokiniana* on *Poa pratensis*

Clinton F. Hodges

Professor of horticulture and of plant pathology, Department of Horticulture, Iowa State University, Ames 50011.
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ABSTRACT

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Four preemergence herbicides (benefin, bensulide, dacthal [DCPA], and siduron) were evaluated for effect on the severity of leaf spot caused by *Drechslera sorokiniana* on *Poa pratensis*. On leaves of plants not previously exposed to the herbicides and inoculated with conidia in aqueous droplets containing the various herbicides, leaf spot development either was not affected or was inhibited by all concentrations tested. Leaf spot development was inhibited, not affected, or stimulated on plants previously exposed to the herbicides by soil application and inoculated with conidia in water droplets. Benefin at all concentrations inhibited leaf spot development. Concentrations of 10^{-12}, 10^{-9}, and 10^{-6} of DCPA inhibited, had no effect, and stimulated leaf spot, respectively. Siduron stimulated leaf spot at 10^{-12}M but had no effect at other concentrations. All concentrations of bensulide stimulated leaf spot. The results suggest that, except for benefin and bensulide, stimulation or inhibition of *D. sorokiniana* leaf spot by preemergence herbicides is concentration specific.

Additional key words: benfluralin, *Bipolaris, Helminthosporium.*

The ability of herbicides to stimulate or inhibit diseases induced by fungal pathogens is well documented (1,15–17). The leaf spot induced in *Poa pratensis* L. by *Drechslera sorokiniana* (Sacc.) Subram. & Jain (=*Helminthosporium sativum* P.K.& B.) generally is stimulated by auxinlike postemergent herbicides (eg, 2,4-D, 2,4,5-T, MCPP, dicamba) (9); the stimulation is associated with an interaction between the herbicides and sequential leaf senescence that enhances pathogenesis on each older leaf (10). Results of other studies show wheat to be predisposed to infection by *D. sorokiniana* (12) and corn to infection by *D. heterostrophus* (*H. maydis*) (19) in response to 2,4-D; conversely, 2,4-D reduces root rot of barley incited by *D. sorokiniana* (21).

The preemergence herbicides benefin, bensulide, dacthal (DCPA), and siduron are applied to established *P. pratensis* turf for preemergence control of annual grasses (6,18). The potential influence of these herbicides on pathogenesis by *D. sorokiniana* on leaves of *P. pratensis* is unknown; however, all inhibit root growth of grasses (3–5,20,23). Inhibition of root growth could induce stresses in the whole plant that might influence leaf spot development. The herbicides also inhibit or have no effect on conidial germination, germ-tube growth, mycelial growth, or conidial production of *D. sorokiniana* (11). Other research results show that benefin and bensulide reduce mycelium growth and delay sporulation by *Drechslera cynodontis* (14), but the abilities of these herbicides to affect the pathogenicity of this pathogen on bermudagrass were not determined.

The research presented here was initiated to determine the potential influence of benefin, bensulide, DCPA, and siduron on leaf lesion development induced by *D. sorokiniana* on leaves of *P. pratensis*.

MATERIALS AND METHODS

The preemergence herbicides evaluated for influence on leaf spot development on *P. pratensis* included *N*-butyl-*N*-ethyl-α,α,α-trifluoro-2, 6-dinitro-*p*-toluidine (benefin, benfluralin), *O,O*-diisopropyl phosphorodithioate *S*-ester of *N*-(2-mercaptoethyl) benzenesulfonamide (bensulide), dimethyl tetrachloroterphthalate (DCPA, dacthal), and 1-(2-methylcyclohexyl)-3-phenyl urea (siduron). Each herbicide was tested at concentrations of 10^{-12}, 10^{-9}, and 10^{-6}M. The highest concentration (10^{-6}) of each herbicide was equivalent to about 7.1 g a.i. per 93 m^2 (0.25 lb a.i. per 1,000 ft^2, granular formulation basis).

Poa pratensis 'Newport' was vegetatively propagated in a steamed loam-peat soil mix (2:1, v/v) in 7.6-cm (3-in.) plastic pots. All plants were grown in the greenhouse (20–30 C) for 60 days under a 16-hr daylength with supplemental incandescent lights. Cultures of *D. sorokiniana* were maintained on 20 ml of 1.0% Czapek Dox broth (10 g/L) in 3.0% (w/v) Bacto agar in 15 × 100-mm sterile, plastic petri dishes. A constant level of inoculum virulence was maintained by using conidia from 20-day-old cultures, and the pathogen was continuously cycled on *P. pratensis* tissue with individual cultures prepared from hyphal-tips isolated from diseased tissue (8).

Preemergence herbicides were applied to *P. pratensis* in droplet and soil drench treatments. Droplet treatments consisted of applying conidia in droplets of each herbicide solution at each concentration to leaf blades of plants not previously exposed to herbicides. Inoculations were conducted on the green leaves (15 cm or longer) of a single shoot. Suspensions of 10 conidia in 0.02 ml (500 conidia per milliliter) of the herbicide solutions (or water for controls) were prepared with an automatic particle counter (High Accuracy Products Corp., Montclair, CA 91763) and used for all inoculations. Each leaf blade was inoculated in five positions (10 conidia in 0.02 ml of the appropriate herbicide solution per position) 1 cm apart by using a specially designed inoculation apparatus (22). The soil of the potted plants in the soil drench treatment groups received two alternate-day applications of 20 ml (40 ml total) of the appropriate herbicide solution and were inoculated 2 days after the last application. Plants were inoculated as previously described, except that conidia were suspended in distilled water. Each treatment (droplet and soil drench) consisted of the green leaves of 20 shoots (one per plant) and was replicated five times (a total of 100 shoots for each concentration of each herbicide). Inoculated plants were incubated 6 days at 22 C under low-intensity (about 75–80 μE in) continuous fluorescent (daylight) lamps and then evaluated for the mean percentage of diseased tissue per leaf. The techniques and procedures for determining the mean percentage of diseased tissue per leaf have been described previously (9,22).

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RESULTS

Inoculation of *P. pratensis* not previously exposed to preemergence herbicides with conidia of *D. sorokiniana* in droplets of preemergence herbicides either had no effect on or inhibited leaf spot development. Bensulide at 10^{-12}M and DCPA at 10^{-6}M had no effect; other concentrations of bensulide and DCPA, and all concentrations of benefin and siduron inhibited leaf spot development (Fig. 1A–D).

Leaf spot development on plants exposed to preemergence herbicides by soil application and then inoculated with conidia in water droplets was inhibited, not affected, or stimulated. Leaf spot development was inhibited by all concentrations of benefin (Fig. 1A) and stimulated by all concentrations of bensulide (Fig. 1B). The effects of DCPA and siduron on leaf spot development were influenced by concentration. DCPA inhibited leaf spot at 10^{-12}M, had no effect at 10^{-9}M, and was stimulatory at 10^{-6}M (Fig. 1C). Siduron had no effect on leaf spot development at 10^{-9} and 10^{-6}M, but stimulated leaf spot at 10^{-12}M (Fig. 1D).

DISCUSSION

Pathogenesis by *D. sorokiniana* on leaves of *P. pratensis* differs in response to preemergence herbicides, depending on whether conidia are applied in herbicide droplets to leaves of plants not previously exposed to the herbicides or whether conidia are applied in water droplets to leaves of plants growing in soil treated with herbicide prior to inoculation. The absence of an effect or the inhibition of leaf spot development when conidia are applied to the leaves in herbicide droplets (Fig. 1A–D) coincides with in vitro inhibition of conidia germination and germ-tube growth by benefin, bensulide, DCPA, and siduron (11). The presence of the preemergence herbicides during conidial germination, whether in vitro or on the leaf surface, is inhibitory to the germination process and subsequent leaf spot development. The various herbicides examined in this study are applied to established *P. pratensis* in liquid and granular form. Therefore, it is possible for these herbicides to come into direct contact with conidia on leaf surfaces and influence germination and primary infection.

Pathogenesis by *D. sorokiniana* on leaves of *P. pratensis* is stimulated or inhibited on leaves of plants previously exposed to the herbicides by soil application and inoculated with conidia in water droplets. It is probable that the pathogen responds to some physiological change induced by the herbicide in the host. A common mode of action for these herbicides in grasses is inhibition of mitosis in root apices (3–5,20,23). Preemergence herbicides may or may not be readily translocated; the dinitroanilines (benefin) are not readily translocated to leaves (20), whereas the substituted ureas (siduron) are readily translocated to stems and leaves (7) and are metabolized by *P. pratensis* (13). The inhibition of root growth by the herbicides included in this study could significantly influence leaf spot development irrespective of their ability to be translocated. Root inhibition could result in hormonal imbalances and interfere with uptake and translocation of minerals (2); such disturbances could influence the physiology of the whole plant and pathogenesis by *D. sorokiniana* on the leaves.

The quantity of soil-applied preemergence herbicides absorbed by the roots of *P. pratensis* relative to the volume applied is unknown. Some portion of the herbicides is probably adsorbed on the clay and peat components of the soil mix employed (7). This effect, however, is expressed across all treatments. The significant increase in leaf spot development in response to bensulide and siduron was greatest at the most dilute concentrations (Fig. 1B and D); also, the greatest inhibition of leaf spot development by benefin and DCPA likewise occurred at the most dilute concentrations (Fig. 1A and C). Because the greatest stimulation and inhibition of leaf spot development occurs at the lowest concentrations of the various herbicides, the potential adsorption of the herbicides on the clay and peat components of the soil mix does not seem to be a major factor affecting the results.

It is important that the response of *D. sorokiniana* leaf spot on *P. pratensis* exposed to preemergence or postemergence herbicides (9) by soil application generally is different. The auxinlike postemergence herbicides (2,4-D, 2,4,5-D, 2,4,5,-TP, MCPP, and dicamba), with few exceptions, stimulate leaf spot by increasing the extent of chlorosis associated with lesions (9). In the present study, only bensulide is stimulatory at all concentrations (Fig. 1B). DCPA and siduron show some ability to stimulate leaf spot (Fig. 1C and D), but most concentrations of DCPA and siduron and all concentrations of benefin (Fig. 1A) either have no effect or inhibit leaf spot.

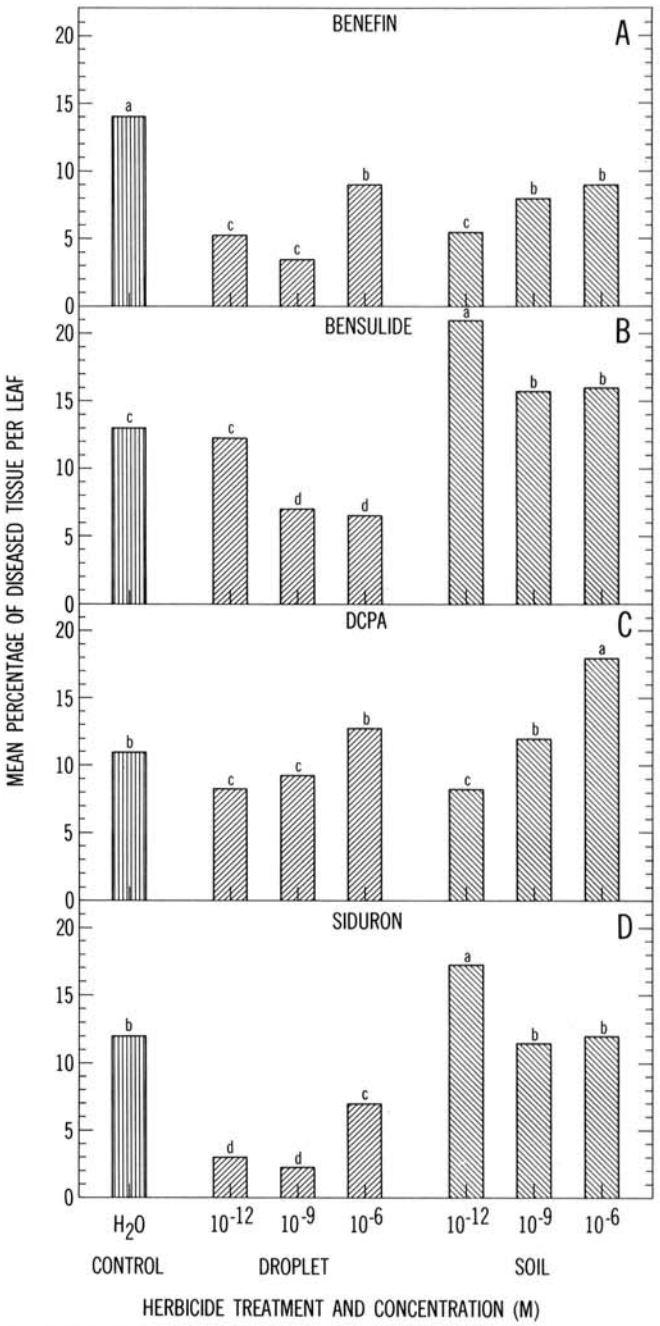

Fig. 1. The influence of preemergence herbicides on the severity of *Drechslera sorokiniana* leaf spot on *Poa pratensis*. Mean percentages of disease per living leaf within droplet and soil-drench treatments followed by the same letter are not significantly different according to Duncan's multiple-range test (P=0.05).

LITERATURE CITED

1. Altman, J., and Campbell, C. L. 1977. Effect of herbicides on plant disease. Annu. Rev. Phytopathol. 15:361-385.
2. Ashton, F. M., and Bayer, D. E. 1976. Effects on solute transport and

plant constituents. Pages 219–253 in: L. J. Audus, ed. Herbicides: Physiology, Biochemistry, Ecology. Vol. 1, 2nd ed. Academic Press, New York. 608 pp.
3. Bingham, S. W. 1968. Effect of DCPA on anatomy and cytology of roots. Weed Sci. 16:449-452.
4. Chang, C. T., and D. Smith. 1972. Effect of DCPA on ultrastructure of foxtail millet cells. Weed Sci. 20:220-225.
5. Cutter, E. G., Ashton, F. M., and Huffstutter, D. 1968. The effects of bensulide on the growth, morphology and anatomy of oat roots. Weed Res. 8:346-352.
6. Crafts, A. S. 1975. Modern weed control. University of California Press, Berkeley. 440 pp.
7. Geissbuhler, H., Martin, H., and Voss, G. 1975. The substituted ureas. Pages 209–291 in: P. C. Kearney, and D. D. Kaufman, eds. Herbicides: Chemistry, Degradation, and Mode of Action. Vol. 1, 2nd ed. Marcel Dekker, Amsterdam, The Netherlands. 500 pp.
8. Hodges, C. F. 1972. Influence of culture age and temperature on germination of *Helminthosporium sorokinianum* conidia and on pathogenicity of *Poa pratensis*. Phytopathology 62:1133-1137.
9. Hodges, C. F. 1978. Postemergent herbicides and the biology of *Drechslera sorokiniana*: Influence on severity of leaf spot on *Poa pratensis*. Phytopathology 68:1359-1363.
10. Hodges, C. F. 1980. Interaction of sequential leaf senescence of *Poa pratensis* and pathogenesis by *Drechslera sorokiniana* as influenced by postemergent herbicides. Phytopathology 70:628-630.
11. Hodges, C. F. 1981. Growth and reproduction of *Drechslera sorokiniana* as influenced by preemergence herbicides. Mycologia 73:244-251.
12. Hsia, Y. T., and Christensen, J. J. 1951. Effect of 2,4-D on seedling blight of wheat caused by *Helminthosporium sativum*. Phytopathology 41:1011-1020.
13. Jordan, L. S., Zurqiyah, A. A., De Mur, A. R., and Clerx, W. A. 1975. Metabolism of siduron in Kentucky bluegrass (*Poa pratensis* L.). J. Agric. Food Chem. 23:286-290.
14. Karr, G. W., Jr., Gudauskas, R. T., and Dickens, R. 1979. Effects of three herbicides on selected pathogens and diseases of turfgrasses. Phytopathology 69:279-282.
15. Katan, J., and Eshel, Y. 1973. Interactions between herbicides and plant pathogens. Residue Rev. 45:145-177.
16. Kavanagh, T. 1969. The influence of herbicides on plant disease. I. Temperate fruit and hops. Sci. Proc. R. Dublin Soc., Ser. B. 2:179-189.
17. Kavanagh, T. 1974. The influence of herbicides on plant disease. II. Vegetables, root crops, and potatoes. Sci. Proc. R. Dublin Soc., Ser. B. 3:251-256.
18. Madison, J. H. 1971. Practical Turfgrass Management. Van Nostrand Reinhold Co., New York. 466 pp.
19. Oka, I. N., and Pimentel, D. 1976. Herbicide (2,4-D) increases insect and pathogen pests on corn. Science 193:239-240.
20. Probst, G. W., Golab, T., and Wright, W. L. 1975. Dinitroanilines. Pages 453–500 in: P. C. Kearney, and D. D. Kaufman, eds. Herbicides: Chemistry, Degradation, and Mode of Action. Vol. 1, 2nd ed. Marcel Dekker, Amsterdam, The Netherlands. 500 pp.
21. Richardson, L. T. 1957. Effect of insecticides and herbicides applied to soil on the development of plant disease. I. The seedling disease of barley caused by *Helminthosporium sativum* P.K. & B. Can. J. Plant Sci. 37:196-204.
22. Robinson, P. W., and Hodges, C. F. 1976. An inoculation apparatus for evaluation of *Bipolaris sorokiniana* lesion development on progressively older leaves of *Poa pratensis*. Phytopathology 66:360-362.
23. Splittstoesser, W. E., and Hopen, H. J. 1970. Root growth inhibition by siduron and its relief by kinetin. Physiol. Plant. 23:964-970.