Postemergent Herbicides and the Biology of Drechslera sorokiniana: Influence on Severity of Leaf Spot on Poa pratensis

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ABSTRACT

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The influence of four chlorophenoxy (2,4-D, 2,4,5-T, 2,4,5-TP, MCPP) and one benzoic acid (dicamba) postemergent herbicides were evaluated for their influence on the development of leaf spot caused by *Drechslera sorokiniana* on *Poa pratensis*. Leaf spot development was severely inhibited on plants not previously exposed to any of the herbicides and leaf-inoculated with conidia in droplets of each herbicide solution prepared at a concentration of 10^{-3} M (2,4,5-TP at 10^{-4} M). Leaf spot development was uneffected or inhibited on plants not previously exposed to the various herbicides and leaf-inoculated with conidia in droplets of 2,4-D, 2,4,5-T, and 2,4,5-TP solutions at concentrations of 10^{-12}, 10^{-9}, and 10^{-6} M. In contrast, MCPP and dicamba at concentrations of 10^{-12}, 10^{-9}, and 10^{-6} M in droplets containing conidia and applied to leaves of plants not previously exposed to the herbicides, resulted in increased leaf spot severity. There was a substantial increase in leaf spot severity on the foliage of plants spray-treated with 2,4,5-T, MCPP, and dicamba, at all concentrations and then inoculated with conidia in droplets of distilled water; plants sprayed with 2,4-D and 2,4,5-TP showed increased disease at the 10^{-12} M concentration, but at higher concentrations, 2,4-D had no effect, and 2,4,5-TP remained inhibitory to disease development. All plants grown in soil treated with various concentrations of each herbicide and inoculated with conidia in droplets of distilled water showed an increase in disease, except for 2,4,5-TP at concentrations of 10^{-6} and 10^{-4} M, which continued to inhibit disease development. Four lesion types produced by *D. sorokiniana* were observed on the herbicide-treated plants. These ranged from small purple-brown necrotic areas with or without halos to enlarged necrotic areas with or without severe chlorotic to straw-colored streaking of the uninfected tissue of the leaf. The results suggest that herbicide-increased disease is, for the most part, independent of any stimulatory effect of the herbicides on in vitro growth of *D. sorokiniana*. Instead, herbicide-induced increases in disease may involve imbalances in the carbohydrate-nitrogen metabolism of *P. pratensis*, and the severe chlorotic to straw-colored streaking of infected leaves is suggestive of ethylene evolution in diseased tissue and (or) toxin production.

Additional key words: *Bipolaris*, *Helminthosporium*, mecoprop, silvex.

Drechslera sorokiniana (Sacc.) Subram. and Jain *(Helminthosporium sativum)* causes leaf spot, leaf blight, and root rot of grasses and cereals in the north-central United States (40). The diseases caused by this pathogen are chronic on *Poa pratensis* L. (4, 5, 21, 42) throughout the growing season and various cultural practices have been implicated in contributing to disease development (9, 17, 21, 37). The potential influence of postemergent herbicides on diseases incited by *D. sorokiniana*, however, has received little attention. The use of postemergent herbicides on *P. pratensis* grown in monocultures is intense and includes the synthetic auxin-like herbicides 2,4-D, 2,4,5-TP, dicamba, and MCPP; 2,4,5-T also was used at one time.

Several reviews have established that herbicides can stimulate or inhibit diseases incited by fungal pathogens (3, 23, 24, 25). Most studies on *Drechslera* sp. have shown 2,4-D to have no effect on, or to inhibit, mycelium growth (6, 8, 16, 34, 41). Stimulation and inhibition of *D. sorokiniana* mycelium growth by 2,4-D also has been reported and is formulation and concentration dependent (20, 22). The herbicides 2,4-D, 2,4,5-TP, 2,4,5-T, MCPP, and dicamba have been shown to influence germ-tube growth from conidia, mycelium growth, and production of conidia of *D. sorokiniana* (20). None of these herbicides influence conidium germination, except at 10^{-4} and (or) 10^{-3} M concentrations at which they are inhibitory. Most concentrations of 2,4,5-T, 2,4,5-TP, MCPP, and dicamba stimulate conidia germ-tube growth and mycelium growth (20). MCPP and 2,4,5-T generally inhibit sporulation of *D. sorokiniana*, and 2,4-D, 2,4,5-TP, and dicamba are inhibitory or stimulatory to sporulation depending on concentration (20).

Research on the influence of postemergent herbicides on *Drechslera*-incited diseases is limited to cereals. Predisposition of wheat to infection by *D. sorokiniana* (*H. sativum*) (22) and of corn to *D. heterostrophus* (*H. maydis*) (32) occurs in response to 2,4-D; conversely, 2,4-D reduces root rot of barley incited by *D. sorokiniana* (*H. sativum*) (34). The research presented here was initiated

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to determine the influence of 2,4-D, 2,4,5-T, 2,4,5-TP, MCPP, and dicamba acids in molar concentrations of 10^{-12}, 10^{-9}, 10^{-6}, and 10^{-3} on leaf lesion development incited by *D. sorokiniana* on *P. pratensis*.

MATERIALS AND METHODS

Poa pratensis 'Newport' was used for all studies. Plants were vegetatively propagated in a steamed 2:1 loam-peat soil mix (2:1, v/v) in 7.6-cm (3-in.) plastic pots. All plants were grown in the greenhouse for 60 days under a 16-hr daylength supplemented by incandescent lights. Cultures of *Drechslera sorokiniana* (Sacc.) Subram. and Jain were maintained on 20 ml of 1.0% Czapek Dox Broth (10 g/liter) in 3% (w/v). Bacto-agar in 15 × 150 mm sterile, plastic petri dishes. To maintain a constant level of virulence only conidia from 20-day-old cultures were used for inoculations and the pathogen was continuously cycled on *P. pratensis* with individual cultures prepared from a hyphal-tip isolated from diseased tissue (19).

Postemergent herbicides evaluated for their influence on disease development included 2,4-dichlorophenoxyacetic acid (2,4-D), 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), 2,4,5-trichlorophenoxypropionic acid (2,4,5-TP, silvex), 2(2-methyl-4-chlorophenoxy)propionic acid (MCPP, mecoprop), and 2-methoxy-3,6-dichlorobenzoic acid (dicamba). All herbicides, except 2,4,5-TP, were utilized in molar concentrations of 10^{-12}, 10^{-9}, 10^{-6}, and 10^{-3}; 2,4,5-TP was not soluble at 10^{-3} M and, therefore, concentrations used were 10^{-12}, 10^{-9}, 10^{-6}, and 10^{-4} M.

Herbicides were applied to plants in droplet, spray, 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. All inoculations were conducted on the four youngest, visible leaf blades of one shoot of each plant. Suspensions of 10 conidia in 0.02 ml (500 per ml) of distilled water (or herbicide solution) were prepared with an automatic particle counter (High Accuracy Products Corp., Montclair, CA 91763) and used for all leaf blade 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 in a specially designed inoculation apparatus (36). The foliage of plants in the spray treatments received four alternate-day applications of 10 ml (40 ml total) of the appropriate herbicide solution atomized over the entire shoot surface and inoculated 2 days after the last application of herbicide. The soil in the soil-drench treatments received two alternate-day applications of 20 ml (40 ml total) of the appropriate herbicide solution and were inoculated 2 days after the last herbicide application. Spray- and soil-drench-treated plants were inoculated as previously described, except that conidia were suspended in distilled water. Each treatment consisted of 10 shoots (one per plant) with four leaves each and was replicated four times (40 plants and 120 leaves per each concentration of each herbicide). Inoculated plants were incubated 6 days at 22 C under low-intensity (about 75 μE) continuous fluorescent (daylight) lamps and then evaluated for disease severity.

Disease severity was determined on 10-cm lengths of inoculated leaf blades harvested after a 6-day-incubation period (36). Total leaf area of the specimen and the amount of diseased area on the specimen were estimated. The estimated diseased area was expressed as a percentage of the estimated total area of the leaf specimen. Total leaf blade area was estimated to the nearest whole number by multiplying the 10-cm length of the leaf by its width (determined with an ocular micrometer) at the midpoint of its length. The area of diseased tissue on leaf specimens was estimated by multiplying the estimated length and width (longest chords of the lesions) of each lesion. Lesion measurements included necrotic and chlorotic areas associated with lesions. Lesions with an area of less than 0.4 mm^2 were not included in the area estimation of diseased tissue. The summation of the estimated lesion areas was expressed as a percentage of the leaf blade area of the specimen and reflected disease severity.

RESULTS

Droplet treatments with 2,4,5-T and 2,4,5-TP in which conidia were applied in droplets of the respective herbicide solutions to leaf blades of plants not previously exposed to herbicides inhibited disease development (Table 1). Inoculations in droplets of 2,4-D, MCPP, and dicamba also inhibited disease development at a concentration of 10^{-3} M (Table 1); inoculations in other concentrations of 2,4-D had no significant influence on disease development. Inoculations in droplets of MCPP and dicamba at concentrations of 10^{-12}, 10^{-9}, and 10^{-6} M increased disease severity (Table 1).

Plants spray-treated with the various concentrations of each herbicide and then inoculated with conidia in distilled-water droplets showed significant inhibition of disease development only in response to 2,4,5-TP at concentrations of 10^{-6} and 10^{-4} M (Table 1). Plants spray-treated with 2,4,5-T, MCPP, and dicamba at all concentrations showed significant increases in leaf disease (Table 1). Lesion development was stimulated by 2,4-D at a concentration of 10^{-12} M; other concentrations had no significant effect (Table 1).

Plants soil-treated with the various herbicides and then leaf-inoculated with conidia in droplets of distilled water showed less disease development where 2,4,5-TP was applied at concentrations of 10^{-6} and 10^{-4} M (Table 1); other concentrations of 2,4,5-TP stimulated lesion development. Plants soil-treated with 2,4,-D, 2,4,5-T, MCPP, and dicamba at all concentrations showed significantly more disease (Table 1).

The greatest increases in disease severity occurred in response to 2,4,5-T, dicamba, and MCPP applied as either a spray- or soil-treatment (Table 1); MCPP droplet-treated plants also showed an increase in disease. The greatest disease-promoting activity of 2,4,5-T, dicamba, and MCPP on spray-treated (also droplet-treated for MCPP) plants occurred at the 10^{-6} M concentration (Table 1); among those plants in treated soil, maximum disease severity occurred in response to 10^{-6} or 10^{-3} M concentrations. Disease severity in response to 2,4-D reached the magnitude of that of 2,4,5-T, dicamba, and MCPP only on plants in soil treated with 2,4-D at 10^{-6} M (Table 1).

Four types of lesions induced by *D. sorokiniana* were observed on *P. pratensis* leaves. The least severe lesions were small purple-brown necrotic areas without chlorotic

halos; lesions of the same or slightly larger size also occurred with distinct halos 1-2 mm wide. These two types of lesions were common on control plants and on herbicide-droplet-inoculated plants on which disease inhibition occurred; i.e., for all droplet inoculations at the 10^{-3} M concentration and all droplet-inoculation concentrations for 2,4-D, 2,4,5-T, and 2,4,5-TP (Table 1). These lesion types are typical of those produced by *D. sorokiniana* on *P. pratensis* in spring and summer of the growing season. The third lesion type was characterized by enlarged purple-brown necrotic areas, often extending to the margins of the leaf, with halos measuring 1-4 mm wide. This lesion type also occassionally displayed chlorotic streaking interconnecting lesions. The fourth lesion type was characterized by enlarged necrotic areas like the third lesion type, but necrotic areas were elongated and accompanied by chlorotic to straw-colored streaking that often involved the entire leaf sample. These last two lesion types occurred most commonly on plants spray- or soil-treated with herbicides. The more severe lesion types occurred most frequently in response to those herbicides associated with the greatest increase in disease severity; i.e., plants spray-treated with 2,4,5-T, dicamba, and MCPP at 10^{-6} M concentrations (Table 1). Lesion development of the last two types are commonly observed on *P. pratensis* infected by *D. sorokiniana* in late autumn.

DISCUSSION

Herbicides are believed to modify plant disease by (i) increasing or decreasing the structural defense of the host; (ii) by inhibiting or stimulating microflora that compete with a pathogen; (iii) by increasing or decreasing pathogen growth or virulence; or (iv) by inducing physiological changes in the host that increase or decrease disease expression (3, 23). The data presented in this study cannot be interpreted to support the first two hypotheses (i.e., structural defense mechanisms or competing microflora); however, some plausible implications are suggested relative to the last two hypotheses.

Stimulation and inhibition of *Drechslera* spp. in vitro by chlorophenoxy herbicides (especially 2,4-D) is well established (6, 8, 16, 20, 22, 34, 41) and is of special interest relative to the results of studies with droplet inoculations. Conidia in droplets of herbicides applied to plants not previously exposed to herbicides represent a transition treatment for comparing in vitro reactions of *D. sorokiniana* with subsequent disease development in vivo on a susceptible host. Droplet treatments with all herbicides at the 10^{-3} M (10^{-4} M for 2,4,5-TP) severely

TABLE 1. Mean percentage of necrotic tissue on leaf blades of *Poa pratensis* exposed to various postemergent herbicides and inoculated with *Drechslera sorokiniana*

Treatment and concentration (M)	Mean percentage of necrotic tissue per leaf blade[a]				
	Herbicides				
	2,4-D	2,4,5-T	2,4,5-TP	MCPP	Dicamba
Nontreated controls	11.8 ad	10.4 a	12.7 a	13.5 a	11.2 a
Droplet treatment[b]					
10^{-12}	14.4 a	3.8 b	3.8 bde	22.7 ge	27.9 b
10^{-9}	14.5 a	3.0 b	3.0 bc	33.8 c	20.2 c
10^{-6}	14.4 a	4.1 b	5.1 bde	34.3 d	15.4 d
10^{-3} or 10^{-4}[c]	0.4 b	1.8 b	1.8 be	2.7 e	6.7 e
Treatment mean	10.9	3.2	3.4	23.4	17.6
Spray treatment[d]					
10^{-12}	22.9 c	22.3 c	21.0 c	29.0 f	24.2 bg
10^{-9}	14.5 a	21.1 c	9.3 ad	33.7 c	18.9 c
10^{-6}	15.1 a	37.0 d	7.4 d	35.4 cd	31.1 f
10^{-3} or 10^{-4}[c]	10.9 d	24.9 cd	3.4 e	20.1 b	25.8 bg
Treatment mean	15.9	26.3	10.3	29.6	25.0
Soil treatment[e]					
10^{-12}	21.1 cf	19.9 c	22.3 c	23.1 g	14.6 d
10^{-9}	19.6 cf	22.2 c	19.9 c	25.2 g	19.8 c
10^{-6}	31.3 e	28.1 d	7.8 d	32.7 c	25.9 bg
10^{-3} or 10^{-4}[c]	18.4 f	33.3 e	6.9 d	35.2 c	23.1 g
Treatment mean	22.6	25.9	14.2	29.1	20.9

[a]Necrotic tissue expressed as the percentage of the mean total area of the four youngest visible leaf blades of 80 individual shoots (four replicates each of 20 shoots). Data analyzed between treatments and treatment concentrations. Mean percentages followed by the same letter not significantly different according to Duncan's multiple range test ($P = 0.05$).

[b]Plants not previously exposed to herbicide. Leaves inoculated with 10 conidia in 0.02-ml droplet of each herbicide at each concentration. Five droplets applied to each leaf at 1-cm intervals.

[c]The herbicide 2,4,5-TP was not soluble at 10^{-3} M and was used at 10^{-4} M.

[d]Foliage of each treated plant was sprayed with 40 ml (four alternate-day applications of 10 ml) of each herbicide at each concentration, and conidia inoculated in 0.02-ml distilled water droplets.

[e]Soil in which plants were growing was drenched with 40 ml (two alternate-day applications of 20 ml) of each herbicide at each concentration, and conidia inoculated in 0.02-ml distilled water droplets.

inhibited disease development (Table 1) and is consistent with in vitro inhibition of conidium germination at that concentration (20). Droplet treatments with 2,4-D, 2,4,5-T, and 2,4,5-TP at molar concentrations of 10^{-12} to 10^{-6} had no effect or inhibited disease development on *P. pratensis* (Table 1), but these concentrations stimulated *D. sorokiniana* in vitro (20). Conversely, droplet treatments with MCPP and dicamba at 10^{-12} to 10^{-6} M concentrations markedly increased disease severity (Table 1), and both herbicides also stimulate in vitro growth of *D. sorokiniana* germ tubes and mycelium (20). No clear explanation exists for the seemingly inconsistent in vitro and in vivo reactions of *D. sorokiniana* in response to 2,4-D, 2,4,5-T, and 2,4,5-TP. The in vitro stimulation of the pathogen by 2,4-D, 2,4,5-T, and 2,4,5-TP (20); the inhibition of *D. sorokiniana* disease in droplet inoculations with these herbicides; and the increase in disease on plants spray- and soil-treated with these herbicides suggests that disease severity is linked more directly to the physiology of the herbicide-treated plants than to stimulation of germ tubes. Other observations on germ-tube stimulation in *Drechslera* sp. without any subsequent increase in numbers of lesions also are supportive of this concept (2, 20, 36).

The results suggest that physiological changes in *P. pratensis* that influence *D. sorokiniana* disease development are associated with very dilute concentrations of some herbicides and perhaps with the organ of the plant absorbing the herbicide. This is illustrated by 2,4,5-TP (and to a lesser extent by 2,4-D), which had an inhibitory effect on disease severity when applied in the droplet treatments, but which progressively increased disease severity at the most dilute concentration (10^{-12} and 10^{-9} M) in the spray and soil treatments (Table 1), respectively, and, hence, from leaves to roots as the absorbing organs. The importance of herbicide absorption by *P. pratensis* for disease development is further illustrated by 2,4,5-T, which severely inhibited disease in droplet treatments, but increased disease in spray and soil treatments in which the herbicide was absorbed by the plants (Table 1). Even MCPP and dicamba, both of which stimulated disease severity in droplet treatments, induced even greater disease severity among spray- and soil-treated plants (Table 1). These results show that, when auxin-like herbicides are absorbed by the tissues of *P. pratensis,* physiological disturbances occur that accelerate disease development. The final concentration of herbicide in the host tissue resulting from spray and soil treatments is not known, however, it seems reasonable to conclude that as the treatment concentration is increased the concentration in the plant is increased. It also is apparent that the increase in disease associated with 2,4-D and to a lesser extent 2,4,5-TP treatments is greatest when these herbicides are absorbed by the roots (Table 1); disease severity on plants spray or soil treated with 2,4,5-T, MCPP, and dicamba is about equal suggesting that absorption of these herbicides by leaves or roots is equally efficient in producing physiological change in the host conducive to increased disease (Table 1).

Many of the biochemical and metabolic changes known to occur in response to chlorophenoxy herbicides (33) are within the realm of changes known to influence disease in plants. Chlorophenoxy herbicides increase respiration, repress photosynthesis, and increase amino acids and protein in plants at the probable expense of soluble carbohydrate levels (30, 33, 35). Decreased soluble sugar levels and increased levels of free amino acids in *P. pratensis* have been associated with larger lesions caused by *D. sorokiniana* (37) and also with increased severity of melting-out caused by *D. poae* (*H. vagans*) (29). Other studies show no significant relationship between sugar content of *P. pratensis* leaves and disease severity resulting from infection by *D. dictyoides* (*H. dictyoides*) (14, 15) and *D. sorokiniana* (10). Also, glutamine accelerates *D. sorokiniana* germ-tube growth and penetration of *Agrostis palustris* leaves (12, 13, 17). The evidence is sufficient to conclude that potential carbohydrate-amino acid imbalances induced by auxin-like herbicides in *P. pratensis* could be, in part, responsible for increased disease when infected by *D. sorokiniana* and that research pertaining to this hypothesis should be pursued.

The chlorotic to straw-colored streaking of severely diseased leaves on plants spray- or soil-treated with 2,4,5-T, MCPP, or dicamba is suggestive of premature senescence. Heiminthosporal is produced by isolates of this pathogen and has been characterized as a nonspecific toxin believed to predispose susceptible hosts to infection and to induce chlorosis (11, 27, 28). Potential interactions between chlorophenoxy herbicides, *P. pratensis,* and *D. sorokiniana* resulting in increased levels of toxin production and subsequent chlorotic streaking is conjecture, but the chlorotic streaks suggest that future research should examine potential toxin involvement.

Ethylene evolution from diseased tissue is another plausible hypothesis for the chlorotic streaking observed. The ability of ethylene to induce yellowing and to destroy chlorophyll is well established (1, 31, 43). It also is well documented that auxin and auxin-like chlorohenoxy herbicides induce ethylene production in plant tissue (26, 31, 33). These characteristics of ethylene symptoms and production in plants, combined with reports of ethylene evolution by bacterial and fungal pathogens in vitro and increased ethylene evolution from plant tissue damaged by pathogens (18, 38) suggests that the chlorotic and straw-colored streaking of severely diseased leaves may be induced by ethylene in a synergistic host-herbicide-pathogen interaction. This hypothesis is supported further by the evolution of ethylene from carrot disks induced by *D. carbonus* (*H. carbonum*) (7).

Ethylene-induced fungistasis of germinating conidia of *D. sorokiniana* has been reported (39). This observation may be of special interest in the inhibition of disease following conidia inoculation in droplets of 2,4-D, 2,4,5-T, and 2,4,5-TP (Table 1). The ability of chlorophenoxy herbicides to induce ethylene production in plants may provide some potential for ethylene evolution into the herbicide droplets on the leaf surface and prevent conidia germination and subsequent disease development.

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