Suppression of Sclerotinia Stem Rot of Soybean by Lactofen Herbicide Treatment

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ABSTRACT

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Severity of Sclerotinia stem rot of soybean after treatment with lactofen (Cobra) and other herbicides was assessed in field experiments conducted in Michigan from 1995 to 1997. At sites where disease pressure was high, disease severity was reduced 40 to 60% compared with controls when lactofen was applied at the V3 (1995 and 1996) or R1 (1997) growth stages. Corresponding seed yields were unchanged or up to 20% greater when lactofen was applied at the R1 stage in 1997. Disease se-

verity was not reduced by lactofen treatments in years and at sites where disease pressure was low to medium, and corresponding yields often were reduced by $\approx 10\%$. High levels of glyceollin accumulated in lactofen-injured leaves collected from field plots in 1996 and 1997. High glyceollin content in lactofen-treated leaves was associated with significant reductions in lesion size when leaves were challenge-inoculated with *Sclerotinia sclerotiorum*.

Additional keywords: diphenyl ether, protoporphyrinogen IX oxidase, Protox, white mold.

There are many agronomic and horticultural crops that exhibit altered disease responses after applications of registered herbicides (2,15,17). Suppression or enhancement of disease incidence and severity by herbicide treatment can occur directly through single or combined effects on the pathogen, host, or surrounding microorganisms (15). Additionally, herbicides may indirectly affect disease levels by controlling weeds, which may eliminate alternative disease hosts or alter the microclimate (15).

The incidence and severity of Sclerotinia stem rot (white mold) disease of soybean has increased in the Midwestern production zones of the United States during the last decade (9). A contributing factor could be the decline in the number and potency of triazine (most commonly atrazine) herbicide applications used in corn production prior to soybean rotations. Triazine herbicides such as atrazine have a direct effect on *Sclerotinia sclerotiorum*, the cause of Sclerotinia stem rot of soybean and many other dicot crop plants. Laboratory studies have shown that carpogenic germination of *S. sclerotiorum* sclerotia is enhanced if sclerotia are incubated in soil containing atrazine. However, stipes and apothecia were malformed, and ascospores were not produced (6,18). The reduced use of atrazine in combination with changes in agronomic practices, such as the trend toward no-till and narrower row spacing (9) and increased use of cultivars related to the Sclerotinia stem rot-susceptible cv. Williams 82, provides more favorable conditions for infection and Sclerotinia stem rot development.

Another example of a possible direct effect of a herbicide on a soybean pathogen and resulting disease is provided by Black et al. (5). Postemergent application of the diphenyl ether herbicide acifluorfen resulted in reduced disease intensity of *Rhizoctonia foliar*

blight, caused by *Rhizoctonia solani* AG-1, and increased yields under low disease pressure. However, there was no such effect under high disease pressure. Mycelial growth and sclerotial production of two isolates were not significantly inhibited by field-strength concentrations when the fungus was grown on acifluorfen-amended potato dextrose agar (PDA).

An example of herbicide treatment affecting soybean defense responses is provided by studies of glyphosate (14,16). Treatment of soybean with nonphytotoxic concentrations of glyphosate inhibited accumulation of glyceollin, the soybean phytoalexin, when inoculated with incompatible *Pseudomonas syringae* pv. *glycinea* or *Phytophthora megasperma* f. sp. *glycinea*. Resistance of hypocotyls to incompatible *Phytophthora megasperma* f. sp. *glycinea* was suppressed in glyphosate-treated seedlings. Hypersensitive cell death was observed in glyphosate-treated plants inoculated with incompatible *Pseudomonas syringae* pv. *glycinea*, despite an increase in bacterial numbers compared with untreated plants, which suggests resistance was suppressed only partially.

Our paper describes the effects of postemergent applications of diphenyl ether and other classes of herbicides on Sclerotinia stem rot in soybean. The study was initiated after growers reported reduced disease severity in soybean fields sprayed with lactofen (Cobra), a diphenyl ether herbicide. Field trials were conducted in Michigan from 1995 to 1997 to assess the effects of herbicide treatments on four soybean cultivars that differ in their reactions to Sclerotinia stem rot. Additional field studies were done to investigate herbicide-induced alterations to host biochemical defenses and how alterations may contribute to observed disease suppression.

MATERIALS AND METHODS

Treatments. Field trials were conducted during the 1995 to 1997 growing seasons at the Michigan State University research farms in Ingham County and at a producer's field in Bay County,

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MI. Sclerotinia stem rot-resistant cv. Northrup King (NK)S 19-90, moderately resistant cv. Corsoy 79, and susceptible cvs. Elgin 87 and Williams 82 were planted in seven-row plots, 6.1 m long with 17.8 cm between rows. The experiments were based on a split-plot randomized complete block design, with cultivars as main plots and treatments as subplots. The herbicide treatments, summarized in Table 1, were applied with appropriate adjuvants at the V3 or V4 growth stage (10) with a CO₂-pressurized hand-held sprayer delivering 190 liters ha⁻¹. Lactofen also was applied at the R1 growth stage in 1997. Water alone was applied each year as one control, and crop oil adjuvant at 1.17 liters ha⁻¹ was applied in 1997 as an additional control.

The Ingham County site was amended with *S. sclerotiorum* inoculum during the fall prior to sowing by incorporating crop trash containing sclerotia into the top 3 cm of soil and was irrigated daily during flowering to maintain humid canopy conditions. The Bay County site has a history of severe Sclerotinia stem rot and is in an environment favorable for natural infection and disease development. The trials were assessed for Sclerotinia stem rot severity at the R7 growth stage (10). Thirty plants per plot were rated on a scale of 0 to 3, according to Grau et al. (12), in which 0 = no symptoms, 1 = lesions on lateral branches only, 2 = lesions on main stem but little or no effect on pod-fill, and 3 = lesions on main stem resulting in plant death and poor pod-fill. A disease severity index (DSI) for each plot was calculated. Plots were harvested for seed yield determination ≈2 weeks after the R8 stage (10). Analyses of variance were performed on the DSI and yield data separately for each year and site, using the Statistical Analysis System program (SAS Institute, Cary, NC).

Physiological effects. Duplicate samples of leaves were collected from NKS 19-90 and Williams 82 field plots at the Ingham County site in 1996, 3 days after V3 lactofen treatment. Leaves with only moderate herbicide injury were selected. Healthy upper leaves were collected in 1996 6 weeks after treatment, when plants had outgrown herbicide injury. Duplicate leaf samples were collected in 1997 4 days after 0.04, 0.07, and 0.11 kg a.i. ha⁻¹ lactofen treatment at the V4 stage. One set of leaves from each collection time was stored at -20°C and later extracted for measurement of the soybean phytoalexin glyceollin, as an indicator of induced defense. Each leaf in the second set of field leaves was inoculated on the day of collection with a 2.5-mm-diameter mycelial plug of *S. sclerotiorum* taken from the margin of a colony grown on PDA at 21°C. Resulting lesion diameters were measured after 2 days of incubation at room temperature.

Frozen leaf tissue was cut into 0.5- to 1.0-cm pieces, transferred to an Erlenmeyer flask, and extracted by covering overnight with 80% methanol (20 to 25 ml/g fresh weight), followed by boiling until 20% of the original volume remained. The resulting leaf extract was collected, and the flask was rinsed with 1 to 2 ml of water. Extract and aqueous rinsate were combined and extracted three times with equal volumes of chloroform. Combined chloroform extracts were dried with anhydrous sodium sulfate. Chloroform was removed by rotary evaporation under reduced pressure. Resi-

dues were resuspended in small volumes (≈200 µl) of ethyl acetate, applied to silica gel (250 µm) thin-layer chromatography plates containing fluorescent indicator (Redi/Plate, Silica Gel GF, Fisher Scientific, Itasca, IL), and developed in chloroform/methanol (100:10, vol/vol) solution. Bands migrating to the same R_f as the glyceollin standard (provided by M. Gijzen, Agriculture and Agri-Food Canada) were identified by fluorescence quenching under short-wave UV light. Bands were scraped from the plates, glyceollin was eluted from the silica gel with ethyl acetate, and ethyl acetate was removed under a flow of N₂. Glyceollin isomers were separated and quantified by high-performance liquid chromatography with a 250 × 4.6-mm silica column (Econosphere Silica 5U, Alltech Associates, Deerfield, IL) with 5% isopropanol in hexane as the mobile phase and detected by absorption at 215 nm. Elution times for isomers I, II, and III at a flow rate of 1 ml min⁻¹ were 11.2, 12.2, and 13.3 min, respectively. Each isomer was quantified by interpolating from curves prepared from published extinction coefficients (4) and absorbances obtained after injecting known amounts of isomers. Log-transformed data were subjected to a regression containing linear and quadratic trend analyses. Means of untransformed data are presented in Tables 2 and 3.

RESULTS

Field studies. Lactofen treatment caused severe foliar injury or burn within 1 day of application. Defoliation and malformation of developing trifoliate leaves also occurred; however, plants outgrew the injuries and appeared healthy within 3 weeks. There was no cultivar by treatment interaction for DSI and yield, so data for each site and year were combined across cultivars for each treatment for presentation (Table 4).

Disease severity and seed yields of water control plots varied across the two locations and 3 years studied (Table 4). Disease severity was greater at the irrigated Ingham County site than at the Bay County site in 1996 and 1997. The near absence of disease at Ingham in 1995 was attributed to unusually hot, drying winds during the growing season.

Disease severity at the Ingham County site was low in 1995, and there were no differences between lactofen- and water-treated plots (Table 4). However, there was a significant yield reduction after lactofen treatment compared with water controls. Conversely, there was a significant reduction in DSI after lactofen treatment at the Bay County site, but no differences in seed yields.

There were significantly lower DSI for lactofen and acifluorfen treatments compared with the water control at the Ingham County site in 1996; however, corresponding yields were not greater. Interestingly, yields were reduced by imazethapyr and chlorimuron ethyl plus thifensulfuron methyl treatments at the Ingham County site, despite an absence of macroscopic injury symptoms after application (Table 4). There were no differences in DSI between any herbicide treatment and water control at the Bay County site in 1996, but there was a significant reduction in yield after lactofen treatment (Table 4).

TABLE 1. Summary of herbicides applied to field plots in 1995 to 1997

Common name	Brand name and manufacturer	Year tested	Concentration (kg a.i. ha ⁻¹)
Lactofen	Cobra, Valent USA Corp.	1995	0.11
		1996	0.11
		1997	0.04, 0.07, 0.11, 0.23
Acifluorfen	Blazer, BASF Corp.	1996	0.42
		1997	0.42
Fomesafen	Reflex, Zeneca Ag Products	1996	0.28
Fomesafen	Flexstar, Zeneca Ag Products	1997	0.25
Imazethapyr	Pursuit, American Cyanamid Co.	1996	0.07
		1997	0.07
Bentazon	Basagran, BASF Corp.	1996	0.84
Chlorimuron ethyl + thifensulfuron methyl	Classic, Du Pont; Pinnacle, Du Pont	1996	0.004 each

There were significant reductions in disease severity at Ingham County in 1997 after treatment with each concentration of lactofen at the R1 growth stage, and yields were greater after 0.07 and 0.11 kg a.i. ha⁻¹ lactofen treatments compared with the water control (Table 4). There were no differences in DSI between any herbicide treatment and the water control at Bay County in 1997; however, the yields for all R1 lactofen treatments were significantly lower than the water control.

Physiological effects. Three days after 0.11 kg a.i. ha⁻¹ lactofen treatment at the V3 growth stage in 1996 leaves contained large areas of brown necrosis that were not present on water-treated leaves. *S. sclerotiorum* lesion diameters on lactofen-treated leaves were significantly smaller than on the respective water controls for each cultivar (Table 2). Glyceollin was not detected in water control treatments; however, it accumulated in lactofen-treated leaves. Glyceollin accumulation was five times greater in NK S19-90 than in Williams 82 (Table 2). Six weeks after V3 treatment, there were no macroscopic injury symptoms on upper leaves collected from any treatment. There were no differences in *S. sclerotiorum* lesion diameters among treatments for either cultivar. Lesions on leaves from older plants were smaller compared with those on control leaves collected 3 days after treatment, possibly suggesting age-related resistance. There were no differences in glyceollin content of leaves 6 weeks after lactofen treatment (Table 2).

Four days after V4 treatment in 1997 herbicide injury after 0.11 kg a.i. ha⁻¹ lactofen treatment was worse than after 0.04 or 0.07 kg a.i. ha⁻¹ lactofen treatment, although injury was not quantified. *Sclerotinia* sp. lesions on leaves treated with any rate of lactofen were significantly smaller than water controls for each cultivar but were not significantly different among the three rates (Table 3). Glyceollin levels for each cultivar were significantly greater than water controls for each lactofen rate; however, levels were not significantly different among lactofen rates. Although glyceollin response appeared to be linear with increasing lactofen rates, it was not demonstrated statistically. Glyceollin accumulation was approximately twice as high for NK S19-90 as for Williams 82 after lactofen treatment, illustrating a significant cultivar by treatment interaction (Table 3).

DISCUSSION

Three years of field studies in Michigan demonstrated suppression of *Sclerotinia* stem rot disease in plots treated with the herbicide lactofen (Cobra). However, disease was not suppressed by lactofen in every situation. The best suppression was observed

in locations and years in which disease incidence was high. Seed yield reductions after application of lactofen occurred when disease pressure was low to medium, most likely as a result of severe injury caused by treatments. Yield reductions also would have occurred in cases of high disease pressure but were counteracted by the increased protection against *Sclerotinia* stem rot. Previous studies acknowledged that soybean exhibits foliar injury after diphenyl ether herbicide sprays and that injury increases with increasing lactofen and acifluorfen rates (13,19). In the same studies, yield reductions of 2.5 to 9.8% occurred in lactofen-treated soybean compared with controls but were apparently not statistically lower. These studies support the current results on the yield-reducing effect of lactofen and demonstrate that a high likelihood of severe *Sclerotinia* stem rot is required before lactofen should be considered as a disease-suppressing treatment.

Interestingly, lactofen applied at the V4 growth stage in 1997 did not alter disease response, but lactofen applied slightly earlier, at V3 in 1995 and 1996 or at R1 in 1997, resulted in less severe *Sclerotinia* stem rot. Although herbicide burn was not quantified, injury after the V4 spray in 1997 was not noticeably different than after the V3 spray in 1995 and 1996. It is conceivable that differences between years in environmental conditions (e.g., temperature and humidity) at the time of spraying, onset of infection, and during disease development could have caused the host to respond differently. Further field trials may aid understanding of these complex interactions.

Possible mechanisms involved in lactofen suppression of *Sclerotinia* stem rot can be proposed based on the current results and mode of action of lactofen. It is unlikely that reduced disease severity occurred because lactofen inhibited *S. sclerotiorum* directly. Lactofen degrades rapidly under aerobic conditions; the average field half-life is 3 days (1). Thus, it is unlikely that carpogenic germination and production of ascospores would be affected by the small volumes of herbicide that might reach the soil surface. In the plant, lactofen has very limited translocation and is metabolized to biologically inactive residues within 24 days (1), which, if applied at the V3 growth stage, would be prior to ascospore production and infection. Additionally, field-strength preparations of lactofen inhibited growth of *S. sclerotiorum* on PDA by <20% compared with water in preliminary studies (results not shown), and the related diphenyl ether herbicide acifluorfen did not significantly inhibit *R. solani* AG-1 in petri dish bioassays (5), suggesting these compounds are not strongly fungicidal.

It is likely that lactofen initiates responses similar to those associated with the hypersensitive response. Lactofen and other diphenyl ether compounds inhibit protoporphyrinogen IX oxidase, (Protox), an enzyme involved in the biosynthesis of heme and

TABLE 2. Resistance response of field soybean leaves 3 days or 6 weeks after V3 lactofen treatment in 1996

Cultivar	Treatment	Glyceollin (µg/g FW) ^w	Lesion diam. (mm) ^x
3 days			
Williams 82	Water control	0.0	10.9
	Lactofen (0.11 kg ha ⁻¹)	7.4 ^y	6.5 ^z
NKS 19-90	Water control	0.0	10.2
	Lactofen (0.11 kg ha ⁻¹)	38.2 ^y	4.6 ^z
6 weeks			
Williams 82	Water control	1.7	5.8
	Lactofen (0.11 kg ha ⁻¹)	1.0	6.9
NKS 19-90	Water control	4.3	5.5
	Lactofen (0.11 kg ha ⁻¹)	2.9	5.3

^w Mean total glyceollin extracted from five leaves per plot harvested from each of four blocks per treatment. FW = fresh weight.

^x Lesion diameters measured 2 days after *Sclerotinia sclerotiorum* inoculation; means of five leaves per plot harvested from each of four blocks per treatment.

^y Means significantly ($P < 0.05$) higher than the corresponding water control.

^z Means significantly ($P < 0.05$) lower than the corresponding water control.

TABLE 3. Resistance response of field soybean leaves 4 days after V4 lactofen treatment in 1997

Cultivar	Treatment	Glyceollin (µg/g FW) ^x	Lesion diam. (mm) ^y
Williams 82	Water control	1.5 a ^z	10.5 a
	Lactofen, 0.04 kg ha ⁻¹	13.4 b	5.7 b
	Lactofen, 0.07 kg ha ⁻¹	20.9 b	6.1 b
	Lactofen, 0.11 kg ha ⁻¹	23.8 b	4.9 b
NKS19-90	Water control	1.8 a	8.9 a
	Lactofen, 0.04 kg ha ⁻¹	36.5 c	4.7 b
	Lactofen, 0.07 kg ha ⁻¹	41.3 c	4.8 b
	Lactofen, 0.11 kg ha ⁻¹	44.8 c	4.8 b

^x Mean total glyceollin extracted from five leaves per plot harvested from each of four blocks per treatment. FW = fresh weight.

^y Lesion diameters measured 2 days after *Sclerotinia sclerotiorum* inoculation; means of five leaves per plot harvested from each of four blocks per treatment.

^z Means followed by the same letter are not significantly different ($P < 0.05$).

chlorophyll in the photosynthetic pathway (1). Inhibition of Protox causes protoporphyrinogen IX to accumulate and leak out of plastids and mitochondria and is autoxidized to protoporphyrin. Protoporphyrin is activated by light, resulting in generation of active oxygen species and lipid peroxidation.

This process may explain the accumulation of glyceollin in lactofen-damaged leaves and is strongly supported by a recent study of the involvement of oxidative processes in the initiation of glyceollin synthesis (8). Based on their results, Degoussé et al (8) proposed a causal link between lipid peroxidation and synthesis of glyceollin. There are other reports of a close association between production of active oxygen species and phytoalexin accumulation in several plants (3). Glyceollin levels in field leaves of each cultivar increased with increasing lactofen rates in 1997, possibly reflecting the level of injury caused by the treatments, although injury was not assessed quantitatively. Colony growth of *S. sclerotiorum* on PDA was inhibited by glyceollin concentrations of 10 µg ml⁻¹ (results not shown); however, its role in suppressing Sclerotinia stem rot in lactofen-treated soybean has yet to be determined. Because lactofen induces a response that is similar in appearance to the hypersensitive disease resistance response, the induction of other localized defenses and the possibility that lactofen may induce a systemic resistance response is under investigation.

Lactofen also may affect disease severity by altering the canopy environment or reducing sites on the plant that are natural infection courts. Assuming that the disease is initiated primarily through petal infection (11) within the canopy, it is conceivable that any injury resulting in greater air movement and lower humidity in the canopy could reduce the incidence and severity of infection. Additionally, the reduction in disease also could be partially explained by observations of delayed flowering, senescence, and maturity in lactofen-treated plots compared with controls. Further careful ex-

perimentation and analyses are necessary to establish any association between canopy environmental conditions and plant development and disease escape.

Interestingly, NK S19-90 had significantly greater levels of glyceollin after lactofen treatment than Williams 82, suggesting NK S19-90 has the capacity for a greater defensive response when stressed or challenged by a pathogen. This conclusion is partially supported by recent studies in which lesions were significantly smaller on NK S19-90 than on Williams 82 leaves excised from plants grown under controlled conditions and inoculated with *S. sclerotiorum* (7). Investigations of other physiological defenses combined with data on plant architecture, maturity, etc., will help elucidate why some cultivars are more resistant than others when grown under field conditions.

Future research will attempt to investigate more closely the mechanism(s) of lactofen-induced Sclerotinia stem rot resistance in soybean. The timing and rate of application for best disease control and higher yields will be optimized. The possible association between the levels of injury and disease suppression will be investigated for lactofen and other injurious diphenyl ether herbicides. Studies of the effect of lactofen on other microorganisms, pathogenic or saprophytic, would be of interest, particularly given the potential widespread use of lactofen as a dual-purpose compound, i.e., as a weed control and as a strategy in integrated control of Sclerotinia stem rot.

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TABLE 4. Sclerotinia stem rot severity and seed yields of field soybeans treated with various herbicides from 1995 to 1997 at two locations in Michigan

Treatment and stage applied	DSI ^w		Yield (t ha ⁻¹) ^x	
	Ingham	Bay	Ingham	Bay
1995				
Water control, V3	3.1	15.8	3.5	3.5
Lactofen 0.11 kg ha ⁻¹ , V3	3.3	7.6 ^y	3.1 ^y	3.3
1996				
Water control, V3	48.5	7.2	2.9	3.6
Lactofen 0.11 kg ha ⁻¹ , V3	21.0 ^y	4.0	3.0	3.2 ^y
Acifluorfen 0.42 kg ha ⁻¹ , V3	32.8 ^y	5.2	2.9	3.4
Fomesafen 0.28 kg ha ⁻¹ , V3	46.7	8.8	2.8	3.5
Imazethapyr 0.07 kg ha ⁻¹ , V3	42.6	7.1	2.5 ^y	3.2
Bentazon 0.84 kg ha ⁻¹ , V3	42.3	4.8	2.8	3.5
Chlorimuron ethyl + thifensulfuron methyl 0.004 kg ha ⁻¹ each, V3	45.4	7.0	2.5 ^y	3.5
1997				
Water control, V4	48.0	19.0	2.0	3.4
Crop oil control, V4	46.3	19.9	2.0	3.5
Crop oil control, R1	45.0	23.4	2.2	3.5
Lactofen 0.04 kg ha ⁻¹ , V4	47.6	23.3	2.0	3.4
Lactofen 0.07 kg ha ⁻¹ , V4	50.8	15.2	1.9	3.4
Lactofen 0.11 kg ha ⁻¹ , V4	52.2	24.5	1.9	3.4
Lactofen 0.04 kg ha ⁻¹ , R1	37.6 ^y	19.3	2.1	3.2 ^y
Lactofen 0.07 kg ha ⁻¹ , R1	28.7 ^y	21.1	2.3 ^z	3.1 ^y
Lactofen 0.11 kg ha ⁻¹ , R1	28.8 ^y	18.3	2.4 ^z	3.2 ^y
Lactofen 0.23 kg ha ⁻¹ , R1	32.3 ^y	14.1	2.2	3.2 ^y
Acifluorfen 0.42 kg ha ⁻¹ , V4	49.0	23.4	2.1	3.3
Fomesafen 0.25 kg ha ⁻¹ , V4	46.3	17.7	2.1	3.5
Imazethapyr 0.07 kg ha ⁻¹ , V4	47.8	22.6	2.0	3.3

^w Disease severity index per plot averaged across test cultivars.

^x Yield per plot, expressed at 13% moisture, averaged across test cultivars.

^y Means significantly ($P < 0.05$) lower than the corresponding water control.

^z Means significantly ($P < 0.05$) higher than the corresponding water control.

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