Field Survival of *Phoma proboscis* and Synergism with Herbicides for Control of Field Bindweed

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ABSTRACT

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Small plot field tests were conducted in Washington County, AR, and Phillips County, CO, to determine the efficacy of *Phoma proboscis* conidia in control of field bindweed (*Convolvulus arvensis*) under various temperature and moisture conditions during 1990–1993. Treatments included combinations of conidia with a formulation of 2,4-dichlorophenoxyacetic acid (2,4-D) and 2-(2-methyl-4-chlorophenoxy)propionic acid (MCPP) used at a sublethal dose of 18 g a.i./ha, the surfactants Activate 9-0 or Activate Plus, corn oil, or sodium alginate. Dew periods of less than 6 hr or temperatures less than 10 C or approaching 32 C were inhibitory to the infection process required for field bindweed control. A rate of 10^7 conidia per milliliter in combination with a sublethal dose of 2,4-D and MCPP increased disease to the level achieved with 10^8 conidia per milliliter and controlled field bindweed. The 10^8 conidia per milliliter rate in combination with a sublethal dose of 2,4-D and MCPP killed mature field bindweed tissue during the later part of the growing season (31 July and 4 September 1992) when dew periods were adequate (8–10 hr). Air-dried conidia were as effective as fresh conidia in causing disease. Nylon membranes and field bindweed stems infested with *P. proboscis* were buried at 1-, 10-, and 20-cm depths at the Arkansas field site and recovered monthly during the winters of 1991–1992 and 1992–1993. No significant difference was found in fungal survival due to depth of burial. Higher winter survival frequencies in the second year were associated with 2–4 C lower average monthly soil temperatures relative to the first year. Survival frequency was higher on membranes than on stems.

Additional keywords: biocontrol, biological control, disease enhancement, pathogen, weed control, pesticide, mycoherbicide, epidemiology

Field bindweed (*Convolvulus arvensis* L.) is one of the most widespread, aggressive, and persistent crop weeds in the world (15). The herbaceous vines spread horizontally on the ground or vertically over crop plants, interfering with crop growth and harvesting operations (31). An extensive root system allows field bindweed to compete effectively for nutrients and water, resulting in severe reductions in crop yields and profits (3,20,30).

Tillage and chemical control of field bindweed are difficult, costly, and inconsistent, and may require up to 5 yr for complete success (2,3,8,20,31–33). Additionally, the environmental impact of repeated chemical herbicide applications, including unwanted damage to crops (9,26) and groundwater contamination (18), warrants consideration of other methods for weed control. Most efforts to control field bindweed with biological agents have emphasized insects (5,21–24,27,30). In 1989, a gall mite, *Aceria malherbae* Nuzzaci, imported from Greece (25) was established in field bindweed plots near Bushland, TX; it is now being evaluated in three other states (6).

In recent years, fungi with potential for controlling field bindweed have been studied (10,14,16,17,19,28). *Phoma proboscis* Heiny, first isolated and identified from Colorado samples (10), infects leaves and stems of field bindweed. *P. proboscis* pathogenicity is specific to members of the family Convolvulaceae, with the exception of pathogenicity on *Omphalodes linifolia* Moench in the Boraginaceae (11). Laboratory experiments demonstrated the efficacy of applications of *P. proboscis* conidia in killing field bindweed seedlings when temperatures ranged from 16–28 C during dew periods of more than 9 hr (14). Disease development was drastically inhibited at 32 C (14).

Because *P. proboscis* was not known to occur outside of Colorado, the proposed field tests of *P. proboscis* on small plots of field bindweed at a site in Fayetteville, AR, were subject to an Environmental Assessment by the USDA/APHIS/PPQ Office, Hyattsville, MD, and a permit was granted in the fall of 1991. However, a 1993 survey of Sherman, Russell, McPherson, and Sumner counties in Kansas and Dundy County in Nebraska revealed the presence of *P. proboscis* in each of these areas, suggesting that distribution of *P. proboscis* may be widespread (D. Heiny, *unpublished*).

This paper presents results of a series of field tests designed to determine the effectiveness of formulations of *P. proboscis* conidia for control of field bindweed under various natural environmental conditions. Additional experiments were conducted to determine winter survival ability of the fungus in soil. An abbreviated report of part of this work has been published (12).

MATERIALS AND METHODS

Inoculum production. *P. proboscis* was originally isolated from infected field bindweed as described previously (10). Specimens were deposited in the U.S. National Fungus Collections (BPI #1103137) and the Kew Herbarium (K #H288/90), and living cultures were placed in the American Type Culture Collection (ATCC #74032, Patent Collection). *P. proboscis* stock cultures were maintained on potato-dextrose agar (PDA) acidified with 0.3 ml of 25% lactic acid per 100 ml of media. Conidia from stock cultures were suspended in sterile water and spread on Difco oatmeal agar plates, prepared one-half strength with agar supplemented. Plates were incubated for 7 days at 23 C with a 12-hr photoperiod (General Electric cool-white fluorescent tubes, lighted incubator Model 1-35VL, Percival Mfg. Co., Boone, IA). After the addition of a few milliliters of distilled water to cultures, the surfaces of pycnidia were scraped with a glass slide to promote streaming of conidia into suspension. Following 10–30 min of streaming, the conidia were filtered through eight layers of cheesecloth. On average, each plate yielded from 3×10^8 to 1.25×10^9 conidia. Concentrations of conidia were adjusted based on hemacytometer counts. Dried conidia used in some treatments (see below) were concentrated conidial suspensions mixed with 0.5–1 g of kaolin (Sigma Chemical Co., St. Louis, MO) per 10^9 conidia, air-dried at room temperature (22–24 C), broken into small pieces, and stored over desiccant for 1 wk at 4 C prior to resuspension for inoculations.

Monitoring viability and pathogenicity. Water agar plates spread with conidia from inoculum treatments were

Field tests were conducted at the University of Arkansas Agricultural Experiment Station with the approval of the Director of the Experiment Station.

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Table 1. Outline of experiments for evaluation of formulations of *Phoma proboscis* conidia on field bindweed in Colorado and Arkansas during 1990–1993

Location	Year	Planting date	Application date	Treatments[y]	Evaluations		
					Counts	% Necrosis	Dry wt.
Colorado	1990	21 May	12 June	1, 2, 3, 4, 5	4 wk		
		21 May	7 July	2, 5, 6, 7	6 wk		6 wk
Arkansas	1991	10 September	5 October	A, B, C, D, E, F, G	4 wk		
	1992	14 April	5 May	A, B, C, D, E, F, G	4 wk		
		14 April	20 May	A, B, C, D, E, F, G	4 wk	4 wk	6 wk
		29 April	3 June	A, B, C, D, E, F, G	4 wk	4 wk	
		13 July	20 August	A, B, C, D, E, F, G	4 wk	4 wk	6 wk
		24 August	30 September	A, B, C, D, E, F, G		4 wk	
	1992	22 May	9 June	I, J, K, L	4 wk		
		22 May	17 June	I, J, K, L	4 wk		
		29 April	17 July	M, N, O, P, Q		4 wk	
		22 June	4 September	F, G, H, R		4 wk	
Colorado	1993	None[z]	11 July	A2, C2, E, C3, F, G2	5 wk		

[y]Treatments described in Table 2. All experiments included four replicate plots for each treatment, except application dates of 17 July and 4 September in which only three replicate plots were used.
[z]Natural infestation of field bindweed in a smooth brome (*Bromus inermis* Leyss.) field.

placed at the field site following plot inoculations and at room temperature in the laboratory overnight to confirm germination. After 16–20 hr, germination was stopped by coating the agar surface with lactophenol cotton blue. For field tests in 1991 and 1992, virulence of conidia preparations and effectiveness of natural dew deposition were evaluated on field bindweed in pots that were placed in the plots during inoculations. One pot from each plot was incubated the same evening in a dew chamber for 12 hr. A second pot from each plot was recovered the following morning after exposure to natural dew. Both groups of pots finally were placed in a growth chamber for 2 wk at 24 C with a 15-hr per day photoperiod. The growth chamber was a Conviron E-7 model (Asheville, NC) with cool-white fluorescent plus incandescent light at 300 $\mu E \cdot m^{-2} \cdot s^{-1}$.

Plot preparation. In 1990–1992, plots measuring 1 × 2 m, separated by 1.5 m, were planted with approximately 100 presoaked field bindweed seeds. Plots were weeded and watered as needed to promote seed germination, and alleys were cultivated or mowed. Treatments were completely randomized and each treatment had four replications unless stated otherwise. In 1993, a field of smooth brome (*Bromus inermis* Leyss.) in Phillips County, CO, was determined to be naturally infested with a population of field bindweed. Plots measuring 1 × 2 m were marked with wooden stakes, and mapped for later detection. The number of field bindweed shoots in each plot was recorded.

Details of planting and inoculation dates are presented (Table 1). Treatments were applied 3–4 wk after planting, except in a few tests as noted (Table 1), when most seedlings had four or more true leaves. Applications of treatments were made with 100 ml/plot (500 L/ha) using the fine mist adjustment of a compressed air sprayer (Chapin Model 2501, Batavia, NY), when winds were calm and just before sundown so that conidia were not immediately exposed to direct sunlight.

Table 2. Formulations evaluated in field plot inoculations of field bindweed with *Phoma proboscis* conidia

Treatment	Description[z]
1	No treatment
2	2% Activate 9-0
3	10^7 conidia/ml
4	10^7 conidia/ml + 2% Activate 9-0
5	10^8 dried conidia/ml + 2% Activate 9-0
6	10^8 dried conidia/ml + 0.125% sodium alginate + 2% Activate 9-0
7	0.125% sodium alginate + 2% Activate 9-0
A	No treatment
B	10^7 dried conidia/ml in surfactant
C	10^7 fresh conidia/ml in surfactant
D	10^8 fresh conidia/ml in surfactant
E	Surfactant alone
F	Surfactant + WBG
G	10^7 conidia/ml in surfactant + WBG
I	0.1% Activate Plus
J	10^7 dried conidia/ml + 0.1% Activate Plus
K	2×10^7 fresh conidia/ml + 0.1% Activate Plus
L	2×10^7 fresh conidia/ml + 0.1% Activate Plus + 10% corn oil
M	10^6 conidia/ml in modified surfactant
N	10^7 conidia/ml in modified surfactant
O	10^6 conidia/ml in modified surfactant + WBG
P	10^7 conidia/ml in modified surfactant + WBG
Q	Modified surfactant + WBG
H	10^6 conidia/ml in surfactant + WBG
R	10^8 conidia/ml in surfactant + WBG
A2	0.1% Activate Plus
C2	5×10^7 dried conidia/ml
C3	5×10^7 dried conidia/ml in surfactant
G2	5×10^7 dried conidia/ml in surfactant + WBG

[z]Activate 9-0 = a complex blend of alkylpolyethoxyethanol, *N*-butanol, isopropanol, and dimethylpolysiloxane (Uniroyal Chemical, Raleigh, NC); Activate Plus = an ionic spreader/activator consisting of a mixture of alkylarylpolyoxyethylene glycols, free fatty acids, and isopropanol (Riverside/Terra Corp., Sioux City, IA); sodium alginate (Kelgin MV, Kelco Division of Merck & Co., Inc., San Diego, CA); WBG = Weed-B-Gon (Chevron Chemical Company, San Francisco, CA) used at 36 mg a.i./L, which is 0.03125 of the recommended rate for control of lawn weeds; surfactant = 0.1% Activate Plus + 10% corn oil in water; modified surfactant = 0.1% Activate Plus + 5% corn oil in water. Fresh conidia were harvested directly from oatmeal agar cultures. Dried conidia consisted of concentrated conidia suspensions mixed with small amounts of kaolin (Sigma Chemical Co., St. Louis, MO) air-dried and stored over desiccant at 4 C for 1 wk prior to rehydration for inoculations.

Formulations. Selection of formulations for field treatments was based on results of laboratory experiments. Treatment compositions and their designations for field tests are presented (Table 2). The adjuvants Activate 9-0 and Activate Plus in mixtures with conidia increased necrosis of seedlings relative to treatments with conidia alone (e.g., 70 vs. 40% of tissues necrosing, respectively, within 2 wk after inoculation), with only

0–2% of tissues necrosing due to the adjuvant alone (D. Heiny, *unpublished*). Dilution experiments established that a chemical herbicide formulation called Weed-B-Gon (WBG, Chevron Chemical Company, Ortho Consumer Products Division, San Francisco, CA) at a dose of 36 mg a.i./L (0.03125 of the recommended rate for control of lawn weeds) in combination with conidia of *P. proboscis* resulted in a synergistic effect, namely 2–3 times as much necrosis relative to treatment with conidia alone, that was particularly useful for killing mature field bindweed vines. Weed-B-Gon contains the following ingredients by weight: 10.8% dimethylamine salt of 2,4-dichlorophenoxyacetic acid (2,4-D), 11.6% dimethylamine salt of 2-(2-methyl-4-chlorophenoxy) propionic acid (MCPP), and 77.6% inert. The 36 mg a.i./L rate of WBG alone caused only minor epinasty on some field bindweed plants. This rate is equivalent to 18 g a.i./ha when applied at 100 ml per 2 m^2 plot and was used in each treatment containing WBG (Table 2).

1990 field experiments. Plots for 1990 tests were planted with field bindweed in Phillips County, CO, and evaluated by plant counts recorded before inoculation and 4 or 6 wk later as indicated, and estimates of percent tissue necrosis, including leaves and stems (Tables 1 and 2). Total shoots harvested from each plot were oven-dried and weight/plot was recorded. These values were divided by the number of plants alive in each plot 6 wk after inoculation. Percentage killed was calculated as number of plants dead/plot after 4 or 6 wk divided by total number of plants inoculated/plot. Temperature and relative humidity at the site were monitored with a hygrothermograph.

1991 and 1992 field experiments. Seven treatments were applied on six dates to separate sets of plots in Washington County (Fayetteville, AR) to evaluate response to differing conditions of temperature and dew period (Tables 1 and 2).

Additional tests were conducted to examine as many environmental variations as possible in one season. Treatments *I, J, K,* and *L* (Table 2) were intended to assess the effect of corn oil on disease development. Treatments *M, N, O, P,* and *Q* (Table 2) were designed to compare the effectiveness of a lower concentration of conidia (10^6 conidia per milliliter) with the 10^7 conidia per milliliter rate, with and without WBG. The usefulness of a higher concentration of conidia (10^8 conidia per milliliter) was evaluated with Treatments *F, G, H,* and *R* applied on 4 September (Tables 1 and 2).

Treatments were evaluated by plant counts recorded before inoculation and 4 wk later, estimates of percent necrosis of total stem and leaf tissue per plot, and dry weight measurements of all shoots collected from each plot 6 wk after inoculation (5–20 and 8–20 treatments; Table 1). Oven-dried weights per plot were divided by the number of plants alive in the respective plots 4 wk after inoculation. Percentage killed was calculated as number of plants dead/plot after 4 wk divided by total number of plants inoculated/plot. Late-season plants were significantly larger and difficult to distinguish as individuals, so vine counts were not practical and percent ground cover based on single plot area was used to estimate plant development in each plot. Percent necrosis of total vine leaves and stems per plot 4 wk after inoculation was estimated with reference to percent ground cover at the time of inoculation. Environmental conditions at the plot site were monitored with the aid of a Campbell 21X micrologger weather station (Campbell Scientific, Inc., Logan, UT).

1993 field experiment. The 1993 tests in Phillips County, CO, included treatments with conidial suspensions one-half as concentrated as the most successful treatments from 1992 experiments, applied midsummer (Tables 1 and 2).

Survival in soil. The rate of *P. proboscis* survival in soil (Nixa cherty silt loam) was evaluated over two winters at the Fayetteville, AR, field plot site. Nylon membranes (0.45 μm, MAGNA Nylon 66, Micron Separations, Inc., Westborough, MA) were cut to approximately 2 cm^2, sterilized by autoclaving, placed on dialysis membrane over PDA, and spread with a suspension of *P. proboscis* conidia. Field bindweed stems 2.5 cm in length were autoclaved for 20

Table 3. Percentage of field bindweed plants killed within 4 wk after small plots in Arkansas were inoculated with formulations of *Phoma proboscis* conidia[x]

Treatment	Description[y]	Inoculation date 10/5/91 (%)[z]	5/5/92 (%)	5/20/92 (%)	6/3/92 (%)	8/20/92 (%)
A	No treatment	3.4 a	1.8 a	4.8 c	5.0 c	0.0 b
B	10^7 dried conidia/ml	5.4 a	2.0 a	63.8 b	32.8 b	8.8 b
C	10^7 fresh conidia/ml	10.3 a	2.5 a	52.8 b	29.3 b	12.5 b
D	10^8 fresh conidia/ml	6.8 a	5.3 a	83.0 a	52.5 a	50.5 a
E	Surfactant only	4.6 a	4.3 a	7.5 c	7.0 c	0.0 b
F	WBG	0.9 a	0.0 a	0.0 c	4.3 c	0.0 b
G	10^7 fresh conidia/ml + WBG	3.6 a	4.8 a	52.0 b	40.3 ab	43.3 a

[x]Each value is a mean of four replicates of plots. Each 1 × 2 m plot received 100 ml of the specified treatment. Values within a column not followed by the same letter are significantly different ($P = 0.05$) according to an LSD test. Plots located at Fayetteville, AR. LSD for comparing application times within a formulation treatment ($P < 0.05$) = 6.8. Analysis of residuals confirmed independence of observations and equality of variances.

[y]All treatments except *A* were in surfactant = Treatment *E*: 0.1% Activate Plus (Riverside/Terra, Sioux City, IA) and 10% corn oil in distilled water. *B*, Conidia air-dried with kaolin and stored for 1 wk at 4 C prior to resuspension. *C* and *D*, Conidia harvested directly from oatmeal agar cultures. *F*, WBG = 36 mg a.i./L of Weed-B-Gon (Chevron Chemical Company, San Francisco, CA), which is 0.03125 of the recommended rate for control of lawn weeds.

[z]Percentage of plants killed based on the number of plants killed within 4 wk after inoculation divided by the number of plants alive before inoculation.

Table 4. Percent necrosis of field bindweed leaves and stems 4 wk after small plots in Arkansas were inoculated with formulations of *Phoma proboscis* conidia[y]

Treatment	Description[z]	Inoculation date 5/20/92 (%)	6/3/92 (%)	8/20/92 (%)	9/30/92 (%)
A	No treatment	0.0 d	0.0 c	0.0 c	4.3 b
B	10^7 dried conidia/ml	86.3 bc	50.0 b	40.0 b	30.0 a
C	10^7 fresh conidia/ml	81.3 c	50.0 b	47.5 b	30.0 a
D	10^8 fresh conidia/ml	95.8 a	75.0 a	73.8 a	45.0 a
E	Surfactant only	0.0 d	0.0 c	2.0 c	2.0 b
F	WBG	0.0 d	0.0 c	3.0 c	10.0 b
G	10^7 fresh conidia/ml + WBG	92.5 ab	40.0 b	72.5 a	45.0 a

[y]Each value is a mean of four replicates of plots. Each 1 × 2 m plot received 100 ml of the specified treatment. Percent ground cover by bindweed shoots in each plot estimated prior to inoculations. Percent necrosis of total shoot tissue 4 wk after inoculation estimated with reference to 100% of tissue available for infection at time of inoculation based on ground cover estimates. Values within a column not followed by the same letter are significantly different ($P = 0.05$) according to an LSD test. Plots located at Fayetteville, AR. LSD for comparing application times within a formulation treatment ($P < 0.05$) = 7.6. Analysis of residuals confirmed independence of observations and equality of variances.

[z]All treatments except *A* were in surfactant = Treatment *E*: 0.1% Activate Plus and 10% corn oil in distilled water. *B*, Conidia air-dried with kaolin and stored for 1 wk at 4 C prior to resuspension. *C* and *D*, Conidia harvested directly from oatmeal agar cultures. *F*, WBG = 36 mg a.i./L of Weed-B-Gon, which is 0.03125 of the recommended rate for control of lawn weeds.

min at 120 C, dipped in a suspension of *P. proboscis* conidia and placed on 1.5% water agar. During incubation for 18 days at 23 C with a 12-hr photoperiod, the membranes became covered with a dense, dark mycelial mat bearing pycnidia, and stems became impregnated with hyphae and embedded pycnidia. For each of the 2 yr of the experiment, five infested stems and two colonized membranes were placed in approximately 50 ml of sifted soil in each of 54 pouches made of 1-mm mesh nylon net fabric. During November of 1991 and 1992, three pouch replicates were buried separately in a marked row at each of three depths (1, 10, and 20 cm) for each month to be sampled. At monthly intervals for 6 mo, infested stems and colonized membranes were removed from the recovered pouches, washed in sterile distilled water, surface-sterilized in 0.26% NaOCl for 2 min, rinsed in sterile distilled water, and placed on PDA for detection of *Phoma* colonies. After 7 days of culture incubation as described above, the identities of suspected *P. proboscis* isolates were confirmed by pathogenicity tests. Field bindweed shoot tips were tagged and inoculated with single drops of hyphal and conidial suspensions from the isolates and incubated for at least 12 hr in dew at 24 C. Those shoot tips inoculated with *P. proboscis* suspensions became necrotic within 3 days.

Statistical analyses. Data were analyzed by analysis of variance and the LSD test using SAS for Personal Computers (SAS Institute Inc., Cary, NC).

RESULTS

1990 field experiments. Temperature and dew period affect disease development in the *P. proboscis*-field bindweed interaction. Temperatures between 16 and 28 C and dew periods of 9 hr or more favor infection and disease (14). At the time of the 21 May inoculation in Colorado, the temperature was 15.5 C and the subsequent low temperature was 9 C with only 2 hr of dew. The high temperature the next day was 26.5 C. Only 48% of conidia germinated on water agar after 16 hr at the field site, compared with 97% at a constant 22 C. The next substantial dew period (12 hr at 20 C) was 2 days later. Following the 7 July inoculation, 91% of conidia on water agar had germinated after 16 hr at the field site. Temperatures were cool at the time of inoculation, but wind prevented dew deposition. The following evening, 33 mm (1.3 in) of rain fell. These conditions were not favorable for infection and the treatments had little effect in controlling field bindweed. No significant differences ($P = 0.05$) were found between treatment means for plant counts or size of plants 4 wk after the 21 May inoculations and for plant counts 6 wk after the 7 July inoculations. In the 7 July tests, no difference was found between mean dry weights harvested from plots given Treatment *5* (10^8 dried conidia per milliliter in Activate 9-0) and Treatment *6* (10^8 dried conidia per milliliter in Activate 9-0 and sodium alginate). Harvests from the Treatment *2* plots sprayed with surfactant alone were found to have mean dry weights (5.8 g/plant) significantly higher ($P = 0.05$) than harvests from the plots treated with conidia (Treatment *5* = 2.3 g/plant; Treatment *6* = 1.8 g/plant). The mean dry weight from Treatment *7* plots (2% Activate 9-0 + 0.125% sodium alginate; 3.8 g/plant) was not significantly different from those of Treatment *5* and *6* plots.

These results indicated a need for the development of formulations to enhance infection in unfavorable environments, particularly when dew periods were not adequate. Thus, further laboratory experiments were conducted and different adjuvants with additional advantages were selected for field tests in the subsequent years.

1991 and 1992 field experiments. The summer of 1992 was unusually cool and wet for northwest Arkansas, with the year's total rainfall of 190.5 cm (75 in) 88.9 cm (35 in) above normal. By the end of June, high temperatures rose above 32 C. Plants inoculated before July were small, having up to eight leaves and being no more than 10 cm in length. Beginning in July, growth rates increased, plants had vines 20–30 cm long, and leaves ranged to 5 cm in length at inoculation. For the 4 September experiment, with a June planting date, vines were 30–61 cm long when inoculated.

Germination of fresh or dried conidia after 14–20 hr on water agar plates at the field site ranged from 78–97%, compared with 80–98% in the laboratory. Inoculations of field bindweed in pots at the field site confirmed pathogenicity of *P. proboscis* for each test. However, the dew period (6 hr) the first morning after the 20 May inoculation was not conducive to infection, resulting in only 1–5% of leaf and stem tissues necrosing on plants in pots left overnight in plots and further maintained in growth chambers. This is in contrast to a high level of disease that developed in the plots following the 20 May inoculation (Tables 3 and 4). Sequential dew periods of 14.5 and 10.5 hr (Fig. 1) when low temperatures were 16 and 13 C, respectively, probably contributed to increasing the level of infections in plots relative to infections in pots. Inoculated pots exposed to 12–19 hr dew in controlled temperature dew chambers sometimes were not as severely diseased as the corresponding plots (Treatments *B, C, D, G*). This suggested that exposure to sequential, intermittent dew periods favored disease compared with a single, continuous dew period.

Low temperatures during the first dew period did not rise above 10 C following the 5 October 1991, and 5 May and 30 September 1992 inoculations (Figs. 1 and 2). Little disease developed in plots used in the 5 October 1991 and 5 May 1992

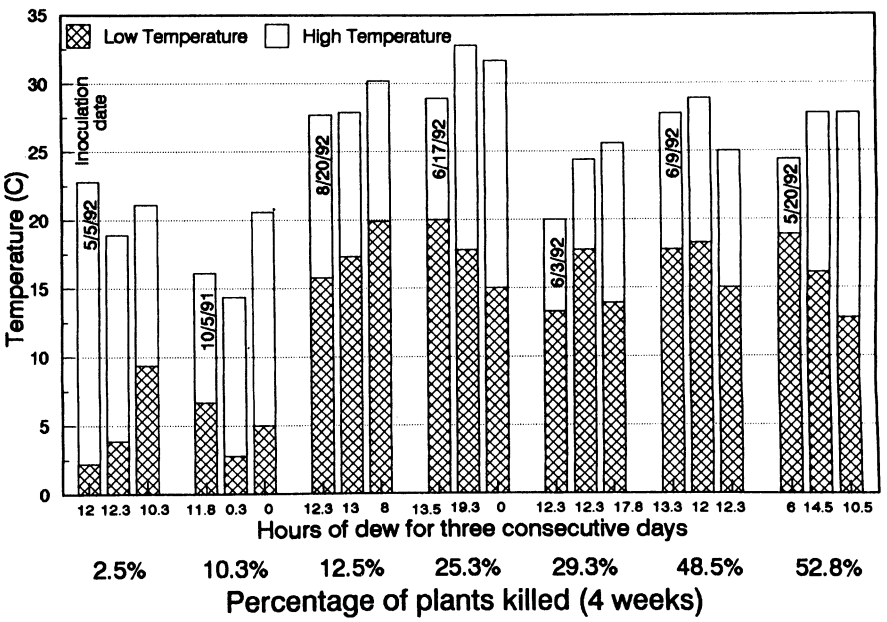

Fig. 1. The influence of high and low temperatures on percentage of plants killed during 3 consecutive days immediately following inoculation of field bindweed with *Phoma proboscis*. Data are presented from left to right in order of increasing percentage killed measured 4 wk after inoculation to emphasize temperature-disease relationships, not chronologically by date of inoculation. Only results from treatments with 10^7 conidia per milliliter are shown. The dew periods for each day also are indicated. Each hour-of-dew value, measured from noon to noon, is the average from two leaf wetness sensors. The tests were conducted in small plots in Arkansas.

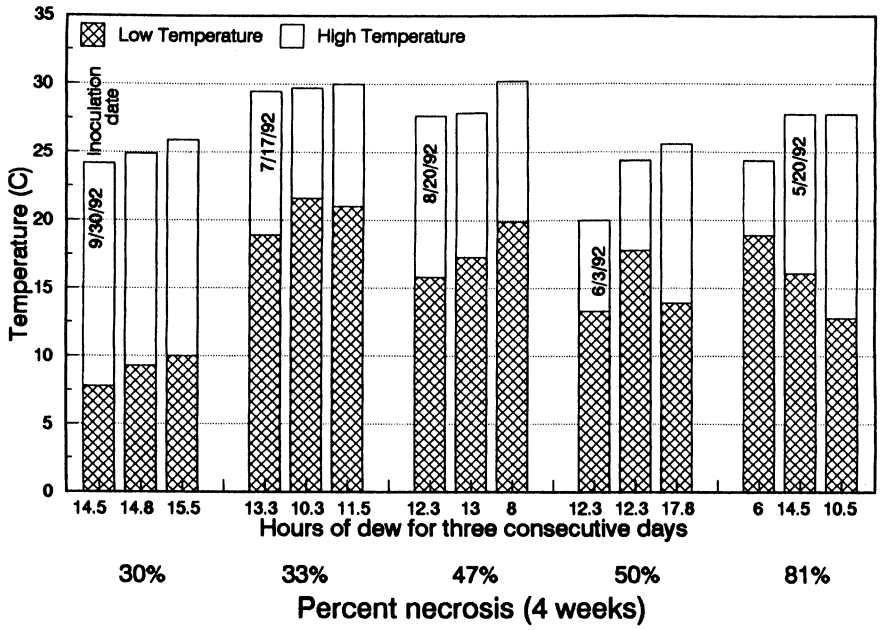

Fig. 2. The influence of high and low temperatures on necrosis of total shoots during 3 consecutive days immediately following inoculation of field bindweed with *Phoma proboscis*. Data are presented from left to right in order of increasing percent necrosis measured 4 wk after inoculation to emphasize temperature-disease relationships, not chronologically by date of inoculation. Only results from treatments with 10^7 conidia per milliliter are shown. The dew periods for each day also are indicated. Each hour-of-dew value, measured from noon to noon, is the average from two leaf wetness sensors. The tests were conducted in small plots in Arkansas.

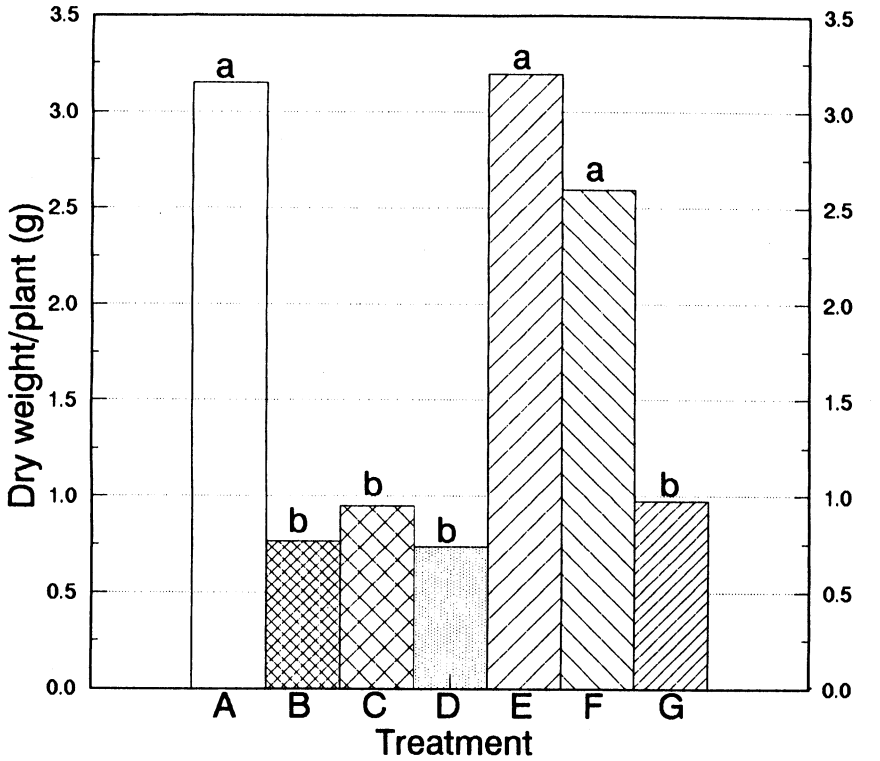

Fig. 3. Effect of *Phoma proboscis* conidial formulations on mean shoot dry weight 6 wk postinoculation of field bindweed plots on 20 May or 20 August 1992. The two experiments were not significantly different ($P = 0.4$), so results were combined. Each value is the mean of eight replicate plots. Values not assigned the same letter are significantly different ($P = 0.05$) by an LSD test. All treatments except *A* (not treated) were mixed in surfactant (Treatment *E*, 0.1% Activate Plus and 10% corn oil in distilled water). Treatment *B*, 10^7 dried conidia per milliliter; *C*, 10^7 fresh conidia per milliliter; *D*, 10^8 fresh conidia per milliliter; *E*, surfactant only; *F*, 36 mg a.i./L of Weed-B-Gon; *G*, 10^7 fresh conidia per milliliter and 36 mg a.i./L of Weed-B-Gon.

inoculations (Table 3), despite 12-hr dew periods the first morning in each case. For the 30 September inoculations, extensive vine development made individual plants difficult to distinguish at the time of ratings, so leaves and stems in each plot were rated for percent necrosis only (Table 4). Ratings for the 30 September test were the lowest of those experiments rated for percent necrosis (Table 4), likely resulting from low temperatures during the 13- to 15-hr dew periods succeeding the inoculations.

Disease increased during the period between inoculation and 2 wk, and between 2 and 4 wk after inoculations in each test. On older, flowering vines, *P. proboscis* infected and killed flower pedicels, and thus prevented seed production. *P. proboscis* was isolated from scattered lesions on field bindweed leaves and stems in control plots not treated with conidia during inoculations, indicating fungal inoculum spread between plots, aided by wind or splashing rain. Severity of the resulting disease was minimal, with only 2–10% of shoot tissue necrosing (Table 4).

The highest percentage of seedlings killed in each experiment resulted from treatment with 10^8 conidia per milliliter (Table 3). Treatment with rehydrated conidia essentially was as effective in killing seedlings as treatment with fresh conidia. When environmental conditions immediately after inoculation were conducive to infection (20 May), the addition of WBG did not increase the percentage of seedlings killed relative to conidia alone. When conditions were suboptimal (3 June, 20 August), because of warmer temperatures and a consequent faster rate of field bindweed growth, the addition of WBG improved kill relative to conidia only. Although the percentage killed appears low for some experiments (Table 3), those plants that did not die were injured severely and remained stunted.

The trends in data patterns for percent necrotic tissue were similar to data patterns for percentage killed (Table 4). Little difference resulted from treatments with either dried or fresh conidia. The highest amount of disease occurred after treatment with 10^8 conidia per milliliter. In general, WBG at 36 mg a.i./L in combination with 10^7 conidia per milliliter (Treatment *G*) achieved the same result as the 10^8 conidia per milliliter treatment alone (Treatment *D*), with the exception of the 3 June inoculation, in which disease was slightly lower in Treatment *G*. A rainfall of 0.25 mm (0.01 in) occurred within a few hours after inoculation on 3 June, which may have diluted the WBG on plant surfaces. The lowest percent tissue necrosis in the 10^7 conidia per milliliter treatments (Fig. 2) resulted when daily low temperatures were less than 10 C (30%) or when daily high temperatures approached 30 C (33%). Percent necrosis

increased as temperatures approached 20 C as long as the corresponding high temperature was not above 28 C (Fig. 2).

Average dry weights for the 20 May and 20 August inoculations indicated a significantly lower biomass in those plots that received conidia treatments versus those that did not (Fig. 3). The values reported may be slightly high because only living plants were counted, but all field bindweed shoots, whether living or dead, were harvested from plots. However, dead shoots usually had no leaves.

Results of the 9 June (Group 1) and 17 June (Group 2) inoculations (Fig. 4) indicated higher overall disease for Group 1 than Group 2. The use of rehydrated dried conidia (Treatment *J*) at one-half the concentration of fresh spores resulted in approximately one-half the rate of seedling kill in Group 1, indicating equivalent infectivities between dried and fresh conidia. The presence (Treatment *L*) or absence (Treatment *K*) of 10% corn oil made little difference in percentage killed for Group 1. However, 10% corn oil may have contributed to increased percentage killed in Group 2, but the differences were not statistically significant (Fig. 4). Plots for the Group 1 and 2 experiments were planted at the same time, but no measurable difference was detected in plant size due to the 1-wk difference in inoculation dates. One difference between the Group 1 and 2 inoculations was the temperature within 2 days of inoculation. The maximum temperature on the second day for Group 2 was 32.8 C, in contrast to temperatures below 29 C for 1 wk following inoculation of Group 1. Both groups were exposed to at least 13 hr of dew the morning after inoculation. No dew occurred on the third morning following inoculation of Group 2, whereas Group 1 was consistently exposed to morning dew of 11–20 hr for 6 days following inoculation, including two rainfalls totaling 4.3 cm (1.68 in). Two rainfalls within 1 wk after Group 1 was inoculated totaled 0.9 cm (0.37 in). The results for the June Group 1 and 2 tests suggested that when temperature and moisture conditions are not entirely favorable for infection, the addition of corn oil can promote a slightly higher level of disease development, expressed as percentage of seedlings killed.

Daily high temperatures for 1 wk following the 17 July inoculation were above 28 C, with dew periods of 6–14 hr during low temperatures of 19–23 C. Within 4 wk, Treatment *P* containing conidia mixed with WBG nearly doubled the amount of necrosis developing after treatment with conidia alone (Treatment *N*), while necrosis was minimal in plots treated with the same rate of WBG alone in surfactant (Treatment *Q;* Table 5). The addition of WBG to 10^6 conidia per milliliter (Treatment *O*) also increased necrosis to the level caused by 10^7 conidia per milliliter (Treatment *N*).

The minimum temperature following the 4 September inoculation was 18.4 C, with 8 hr of dew the next morning and a high of 28.9 C the next day. Mean necrosis following the 4 September inoculation was similar to necrosis following the 17 July treatments. Increasing the concentration of inoculum to 10^8 conidia per milliliter in combination with WBG (Treatment R; Table 5) resulted in the percent of necrotic tissue approaching that on smaller plants in the 20 May inoculation when temperatures were cooler (Table 4).

1993 field experiment. The temperature was 15.6 C during inoculation on 11 July in Colorado, but no dew occurred for the next 20 hr. A brief rainfall late the following afternoon when temperatures were below 24 C was followed by several days of cool weather. Conditions in August were dry, and field bindweed and bromegrass both suffered from drought at the time of final counts. Many shoots probably were killed by lack of water rather than by the treatments applied. The only significant difference in shoot death occurred between the surfactant controls (Treatment *A2* or *F*) and conidia mixed with surfactant and WBG (Treatment *G2;* Table 6). Surfactant control *E* had a higher mean percentage killed rating than one of the conidial treatments, *C3*. Leaf spots symptomatic of *P. proboscis* infection,

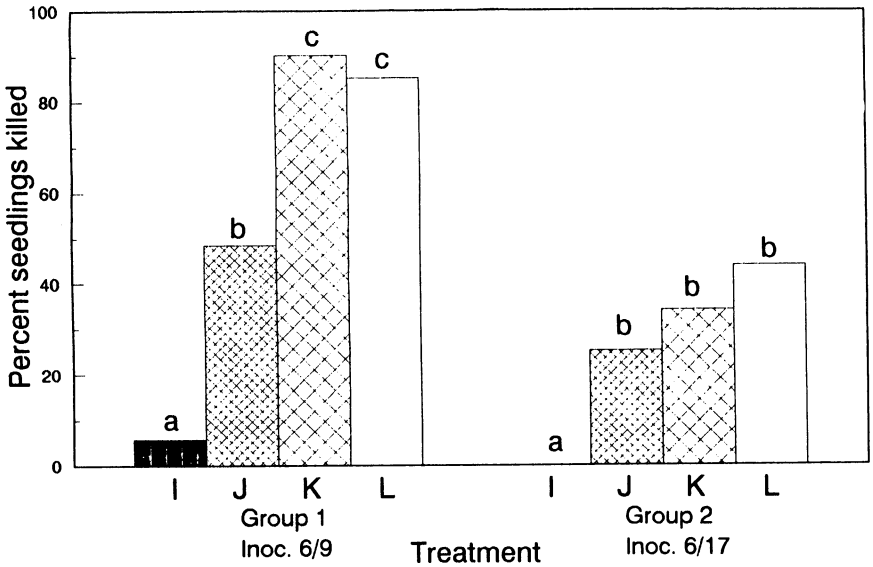

Fig. 4. Percentage of seedlings killed within 4 wk following inoculation of field bindweed plots with conidia of *Phoma proboscis* on 9 June or 17 June at the Fayetteville, AR, test site. Each value is the mean of four replicate plots. Values not assigned the same letter are significantly different ($P = 0.05$) by an LSD test or a *t* test. Treatments: *I,* 0.1% Activate Plus; *J,* 0.1% Activate Plus and 10^7 dried conidia per milliliter; *K,* 0.1% Activate Plus and 2×10^7 fresh conidia per milliliter; *L,* 0.1% Activate Plus and 2×10^7 fresh conidia per milliliter and 10% corn oil.

Table 5. Percent necrosis of field bindweed leaves and stems in small plots in Arkansas 4 wk after application of formulations of *Phoma proboscis* conidia[x]

		Inoculation date		
Description	Treatment[y]	7/17/92 (%)	Treatment[y]	9/4/92 (%)
10^6 conidia/ml	*M*	22.5 b		...[z]
10^7 conidia/ml	*N*	33.3 b		...[z]
10^6 conidia/ml + WBG	*O*	33.3 b	*H*	33.3 bc
10^7 conidia/ml + WBG	*P*	60.0 a	*G*	50.0 b
10^8 conidia/ml + WBG		...[z]	*R*	85.0 a
WBG	*Q*	0.3 c	*F*	5.3 c

[x]Each value is a mean of three replicates of plots. Each 1 × 2 m plot received 100 ml of the specified treatment. Percent ground cover by bindweed shoots in each plot estimated prior to inoculations. Percent necrosis of total shoot tissue 4 wk after inoculation estimated with reference to 100% of tissue available for infection at time of inoculation based on ground cover estimates. Values within a column not followed by the same letter are significantly different ($P = 0.05$) according to an LSD test. Plots located at Fayetteville, AR.

[y]Treatments *M, N, O, P, Q* in modified surfactant consisting of 0.1% Activate Plus and 5% corn oil in water. Treatments *H, G, R, F* in surfactant consisting of 0.1% Activate Plus and 10% corn oil in water. Conidia harvested directly from oatmeal agar cultures. WBG = 36 mg a.i./L Weed-B-Gon, which is 0.03125 of the recommended rate for control of lawn weeds.

[z]Not tested.

grasshoppers, leaf miner damage, and yellowing were prevalent on field bindweed throughout the field.

Survival in soil. No significant effect on survival of *P. proboscis* was observed for depth of burial. However, survival was significantly higher ($P = 0.0001$) on membranes than on stems (Fig. 5). The December 1991 low percentage of survival on membranes was attributed to accidental excessive surface sterilization of the membranes. In addition, survival generally was higher in the second than in the first year ($P = 0.0001$), in direct correlation with higher average soil temperatures in the first year (Fig. 6). As soil temperatures increased between April and May, survival of *P. proboscis* declined, except on membranes in 1992–1993 (Fig. 5). Seedlings emerging in the plot area in April of 1993 became naturally infected with *P. proboscis* and some seedlings died, indicating that the fungus had survived in the soil at that site. Other fungi frequently isolated from stems and membranes included species of *Fusarium, Mucor, Penicillium, Rhizopus, Trichoderma,* and yeasts.

DISCUSSION

Although disease levels sufficient to control field bindweed did not always develop, field tests were consistent in revealing environmental conditions that favored or hindered infection of field bindweed by *P. proboscis*. Laboratory experiments (14) were accurate predictors of the influence of temperature and dew periods on disease development, since disease was limited when adequate dew periods coincided with low temperatures, and dew periods of less than 9 hr usually interfered with disease development. Encouraging, however, was the observation that successive favorable dew periods could enhance disease development if at least 6 hr of dew occurred the first night after inoculation (20 May 1992 experiment). Since pycnidia of *P. proboscis* can develop within 5 days after inoculation of field bindweed tissues (13), part of the increase in disease probably was due to secondary infection by germinating conidia dispersed from new pycnidia. However, these experiments were not designed to quantify secondary inoculum effects separately.

The best time for application of *P. proboscis* for biocontrol of field bindweed would be in the spring when temperatures are cool (16–24 C), and moisture is adequate. The best control occurred following the 20 May 1992 inoculation in Arkansas. The relative immaturity of plants at the time of inoculation as well as cool temperatures (highs of 17–28 C for 1 wk, followed by 11–22 C the next week and lows above 10 C for 5 days) and a sustained rainy period contributed to this result. Several days of cool temperatures following the 1993 inoculation in bromegrass apparently could not compensate for a lack of dew the first evening. The optimum time for application of spore treatments in Colorado is likely May through early June, because moisture and temperature are more conducive to disease during that period.

The ability of dried conidia of *P. proboscis* to retain high levels of viability following storage represented a distinct advantage of *P. proboscis* over *Phomopsis convolvulus* Ormeno, another pathogen with potential for use in control of field bindweed. Various protocols were tested to dry, store, and rehydrate conidia of *P. convolvulus,* but resulting germination ranged from 0–20% (28). Dried conidia of *P. proboscis* retained germination rates ranging from 78–98%.

Control of field bindweed in small plots was achieved following application of conidia of *P. proboscis* by a combination of kill, necrosis, and suppression of growth. Others (30) have advocated the combined use of multiple stress factors to achieve effective control of field bindweed. It may be advantageous to apply *P. proboscis* initially for complete shoot kill, then, as new shoots begin to emerge from surviving roots, follow with a mass release of an arthropod that

Table 6. Percentage of field bindweed shoots killed within 5 wk after small plots in Colorado were inoculated with formulations of *Phoma proboscis* conidia[x]

Treatment	Description[y]	Inoculation date (7/11/93) %[z]
A2	Activate Plus	16.3 b
C2	Conidia + Activate Plus	36.5 ab
C3	Conidia + Activate Plus + corn oil	20.3 ab
E	Activate Plus + corn oil	23.1 ab
F	Activate Plus + corn oil + WBG	11.7 b
G2	Conidia + Activate Plus + corn oil + WBG	44.2 a

[x]Each value is a mean of four replicates of plots. Each 1 × 2 m plot of field bindweed and smooth brome received 100 ml of the specified treatment. Values within a column not followed by the same letter are significantly different ($P = 0.05$) according to an LSD test. Plots located in Phillips County, CO.

[y]Treatments: *A2*, 0.1% Activate Plus. *C2*, 5×10^7 dried conidia/ml in Treatment *A2*. Conidia were air-dried in kaolin for storage prior to resuspension for inoculations. *C3*, 5×10^7 dried conidia/ml in Treatment *E*. *E*, 0.1% Activate Plus and 10% corn oil in water. *F*, 0.1% Activate Plus, 10% corn oil, and 36 mg a.i./L of Weed-B-Gon, which is 0.03125 of the recommended rate for control of lawn weeds. *G2*, 5×10^7 dried conidia/ml in Treatment *F*.

[z]Percentage of entire shoots (vines) killed based on the number of shoots killed within 5 wk after inoculation divided by the number of shoots alive before inoculation.

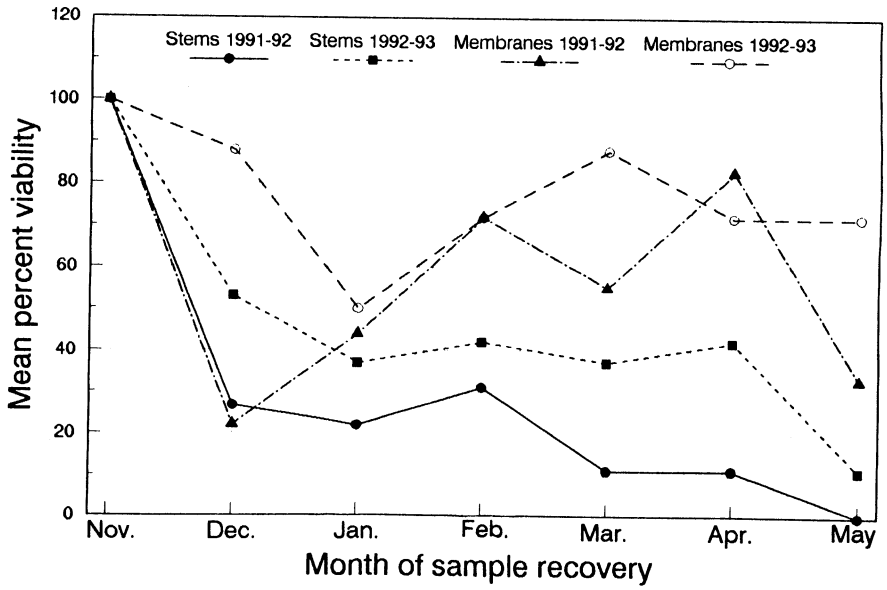

Fig. 5. Recovery of viable *Phoma proboscis* propagules following burial of infested field bindweed stems or nylon membranes (0.45 m) for 1–6 mo at the Fayetteville, AR, field site, during 1991–1992 or 1992–1993. Each value is the mean of three replicates, each composed of five stems and two membranes, at each of three depths (1, 10, and 20 cm). No significant difference was found due to depth of burial according to an LSD test ($P = 0.05$). Survival was significantly higher on membranes than on stems ($P = 0.0001$). Overall, survival was lower in the first year than in the second year ($P = 0.0001$). Analysis of residuals confirmed the independence of observations and equality of variances.

can become established for sustained suppression of field bindweed growth. In addition to the mite *A. malherbae* (6), one possible candidate is the plume moth, *Emmelina monodactyla* (L.), the larvae of which frequently were observed damaging field bindweed shoots at the plot site (D. Heiny, *unpublished*).

Inoculation of mature field bindweed in plots with 10^8 conidia per milliliter in combination with WBG at 36 mg a.i./L on 31 July 1992, following rainfall, resulted in little green tissue remaining after 4 wk (D. Heiny, *unpublished*). A similar result was quantified in the plot set inoculated 4 September. The effectiveness of this treatment during the warmest part of the growing season is encouraging from a biocontrol standpoint. However, from a commercial viewpoint, the higher rate of conidia may add to the cost of production for large-scale applications. Further work is needed to evaluate the feasibility of this treatment.

Chlorophenoxy herbicides, such as 2,4-D and MCPP, are auxins that have multiple effects on the growth and structure of plants, including dedifferentiation and initiation of cell division in mature cells, and inhibition of cell division in primary meristems (4). Immature cytoplasm is prevented from maturing, mature cytoplasm reverts to the immature stage, and the number of ribosomes increases. Cell enlargement is induced by low levels of 2,4-D as a result of increased activity of autolytic and synthetic enzymes responsible for cell wall loosening and synthesis of new cell wall material (4).

The use of sublethal doses of 2,4-D in combination with the pathogen *Cercospora rodmanii* Conway controlled water hyacinth (*Eichhornia crassipes* (Mart.) Solms) (7). Herbicides and growth regulators can also alter disease susceptibility of crop plants (1). The effect of low rates of WBG in enhancing disease following inoculation with *P. proboscis* may be due to restoration of field bindweed tissues to a more juvenile condition. Whitworth and Muzik (32) found that field bindweed stems of a clone susceptible to 2,4-D were increasingly affected by 2,4-D with increasing age. In laboratory experiments, *P. proboscis* was less successful in causing disease on older mature tissues than on young tissues, such as meristems, young stems, and petioles (10,14). Thus, WBG may effectively increase the target area for infection by *P. proboscis*.

P. proboscis survived over the winter in soil. Before we obtained the permit for field testing of *P. proboscis*, the fungus had not been found in Arkansas, and specifically not on any bindweed plants growing in the region surrounding the plot area. Thus, infections of seedlings occurring in the spring following the 1992 tests were due to *P. proboscis* surviving at the site, rather than to inoculum being brought in by winds or other means from outside the plot area. Higher rates of survival in the soil occurred on the nylon membrane than on dead stems, possibly because the membrane did not provide a food base for competing organisms. The decline in survival as temperatures increased may have been due to increased activity of other microorganisms native to the soil, resulting in competition and antagonism (29). However, by the time *P. proboscis* populations began to decline in the soil, new shoots of field bindweed were emerging, providing an alternate and more hospitable environment for the pathogen's survival and reproduction.

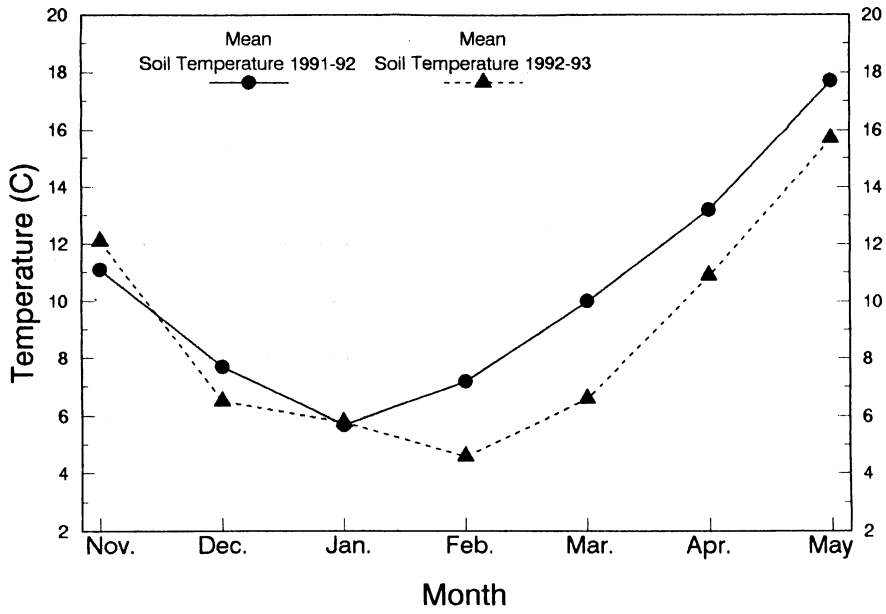

Fig. 6. Monthly averages of soil temperatures recorded daily at a depth of 12 cm at the Fayetteville, AR, field site for November through May of 1991–1993.

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LITERATURE CITED

1. Altman, J., and Rovira, A. D. 1989. Herbicide-pathogen interactions in soil-borne root diseases. Can. J. Plant Pathol. 11:166-172.
2. Anonymous. 1991. How to give bindweed the business. Agrichem. Age 13:1216-17.
3. Anonymous. 1991. AG Review advertising insert by DowElanco. Special Bindweed Issue. Colo. Rancher Farmer 45(6):37A-D.
4. Ashton, F. M., and Crafts, A. S. 1981. Mode of Action of Herbicides. 2nd ed. John Wiley & Sons, Inc., New York.
5. Baloch, G. M. 1977. Insects as biological control agents of field bindweed, *Convolvulus arvensis*. PANS 23:58-64.
6. Boldt, P. E., and Sobhian, R. 1993. Release and establishment of *Aceria malherbae* (Acari:Eriophyidae) for control of field bindweed in Texas. Environ. Entomol. 22:234-237.
7. Charudattan, R. 1986. Integrated control of waterhyacinth (*Eichhornia crassipes*) with a pathogen, insects, and herbicides. Weed Sci. 34 (Suppl. 1):26-30.
8. DeGennaro, F. P., and Weller, S. C. 1984. Differential susceptibility of field bindweed (*Convolvulus arvensis*) biotypes to glyphosate. Weed Sci. 32:472-476.
9. Heering, D. C., and Peeper, T. F. 1991. Field bindweed (*Convolvulus arvensis*) control in winter wheat (*Triticum aestivum*) with herbicides. Weed Technol. 5:411-415.
10. Heiny, D. K. 1990. *Phoma proboscis* sp. nov. pathogenic on *Convolvulus arvensis*. Mycotaxon 36:457-471.
11. Heiny, D. K. 1992. Host-specificity of *Phoma proboscis* in the Convolvulaceae. (Abstr.) Phytopathology 82:498.
12. Heiny, D. K. 1993. Field efficacy and survival of *Phoma proboscis* pathogenic on field bindweed. (Abstr.) Phytopathology 83:1348.
13. Heiny, D. K. 1993. Histology of infection of field bindweed by *Phoma proboscis*. (Abstr.) Phytopathology 83:1415.
14. Heiny, D. K., and Templeton, G. E. 1991. Effects of spore concentration, temperature, and dew period on disease of field bindweed caused by *Phoma proboscis*. Phytopathology 81:905-909.
15. Holm, L. G., Plucknett, D. L., Pancho, J. V., and Herberger, J. P. 1977. The World's Worst Weeds: Distribution and Biology. University Press of Hawaii, Honolulu.
16. Morin, L., Watson, A. K., and Reeleder, R. D. 1989. Efficacy of *Phomopsis convolvulus* for control of field bindweed (*Convolvulus arvensis*). Weed Sci. 37:830-835.
17. Morin, L., Watson, A. K., and Reeleder, R. D. 1990. Effect of dew, inoculum density, and spray additives on infection of field bindweed by *Phomopsis convolvulus*. Can. J. Plant Pathol. 12:48-56.
18. Office of Technology Assessment, Congress of the United States. 1990. Beneath the Bottom Line: Agricultural Approaches to Reduce Agrichemical Contamination of Ground water. Summary. Superintendent of Documents, U.S. Government Printing Office, Washington, DC.
19. Ormeno-Nunez, J., Reeleder, R. D., and Watson, A. K. 1988. A new species of *Phomopsis* recovered from field bindweed (*Convolvulus arvensis*). Can. J. Bot. 66:2228-2233.
20. Rosenthal, S. S. 1983. Field bindweed in California—Extent and cost of infestation. Calif. Agric. 37:16-17.
21. Rosenthal, S. S. 1985. Potential for biological control of field bindweed in California's coastal vineyards. Agric. Ecosyst. Environ. 13:43-58.
22. Rosenthal, S. S., and Buckingham, G. R. 1982. Natural enemies of *Convolvulus arvensis* in Western Mediterranean Europe. Hilgardia 50:1-19.
23. Rosenthal, S. S., and Carter, J. 1977. Host

specificity and biology of *Galernica rufa*—a potential biological control agent for field bindweed. Environ. Entomol. 6:155-158.
24. Rosenthal, S. S., Clement, S. L., Hostettler, N., and Mimmocchi, T. 1988. Biology of *Tyta luctuosa* (Lep:Noctuidae) and its potential value as a biological control agent for the weed *Convolvulus arvensis*. Entomophaga 33:185-192.
25. Rosenthal, S. S., and Platts, B. E. 1990. Host specificity of *Aceria (Eriophyes) malherbe*, [Acari: Eriophyidae], a biological control agent for the weed, *Convolvulus arvensis* [Convolvulaceae]. Entomophaga 35:439-463.
26. Schoenhals, M. G., Wiese, A. F., and Wood, M. L. 1990. Field bindweed (*Convolvulus arvensis*) control with imazapyr. Weed Technol. 4:771-775.
27. Smith, R. C. 1938. A preliminary report on the insects attacking bindweed, with special reference to Kansas. Trans. Kansas Acad. Sci. 41:183-191.
28. Sparace, S. A., Wymore, L. A., Menassa, R., and Watson, A. K. 1991. Effects of the *Phomopsis convolvulus* conidial matrix on conidia germination and the leaf anthracnose disease of field bindweed (*Convolvulus arvensis*). Plant Dis. 75:1175-1179.
29. Waksman, S. A. 1952. Soil Microbiology. John Wiley & Sons, Inc., New York.
30. Wang, R., and Kok, L. T. 1985. Bindweeds and their biological control. Biocontrol News Inf. 6:303-310.
31. Weaver, S. E., and Riley, W. R. 1982. The biology of Canadian weeds. 53. *Convolvulus arvensis* L. Can. J. Plant Sci. 62:461-472.
32. Whitworth, J. W., and Muzik, T. J. 1967. Differential response of selected clones of bindweed to 2,4-D. Weeds 15:275-280.
33. Wiese, A F., and Rea, H. E. 1959. Bindweed (*Convolvulus arvensis*) control and seedling emergence as affected by tillage, 2,4-D, and competitive crops. Agron. J. 51:672-675.