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Integration of biological control of common groundsel (*Senecio vulgaris*) and chemical control

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Control of the annual weed, common groundsel, may be troublesome because of insufficient control by herbicides. Biological control based on stimulating epidemics of the rust fungus *Puccinia lagenophorae* on common groundsel populations may be an alternative to herbicides if the rust fungus can be integrated with the general use of herbicides against other weeds. Formulations of monolinuron, metoxuron, and pendimethalin were selected for the study. The integration of the rust fungus with each of the three herbicides was evaluated. A three-step procedure was followed to evaluate the integration of the rust fungus and these formulated herbicides. The effect of the selected herbicides on common groundsel was tested in the first step. Only formulated monolinuron completely controlled common groundsel under controlled conditions, indicating that biological control is not required in situations where monolinuron is employed for weed control. The effect of metoxuron and pendimethalin on the rust fungus was tested under controlled conditions in the second step. Formulated metoxuron was not compatible with the rust fungus, indicating that biological control cannot be employed in situations where metoxuron is applied. The effect of pendimethalin on *P. lagenophorae* epidemics was evaluated using an epidemiological model in the third step. Formulated pendimethalin had no detectable effects on *P. lagenophorae* epidemics. We concluded that use of *P. lagenophorae* epidemics for common groundsel control is complementary to application of formulated pendimethalin against other weeds.

Nomenclature: Monolinuron; metoxuron; pendimethalin; common groundsel, *Senecio vulgaris* L. SENVU; *Puccinia lagenophorae* Cooke.

Key words: Epidemics, herbicides, *Puccinia lagenophorae*, system management approach.

The system management approach of biological weed control has been proposed as an alternative to herbicides (Müller-Schärer and Frantzen 1996). It aims at inducing and stimulating disease epidemics on weeds to reduce their competitiveness at the population level. A theoretical framework was proposed for this approach (Frantzen and Müller-Schärer 1998). Apart from the introduction of a relatively small amount of pathogen to a weed population to induce an epidemic, the environment will have to be favorable for the development of disease epidemics in weeds to use the system management approach of biological weed control successfully. Many biotic and abiotic factors may stimulate, or inhibit, the progress of epidemics; the abundance of herbicides in the environment is one of these (see reviews of Altman and Campbell 1977; Katan and Eshel 1972). Herbicide use may be considered in a crop environment even when biological weed control is used because the effect of a control agent is generally limited to a single weed species (Charudattan 2000).

The weed, common groundsel, and one of its antagonists, the rust fungus *P. lagenophorae* Cooke, were chosen for a model system to develop this approach (Frantzen and Hatcher 1997). Field experiments were designed to demonstrate control of common groundsel in carrots (*Daucus carota* L.) by epidemics of *P. lagenophorae*. The annual plant, common groundsel, is not a major weed, but it has the potential to cause problems in specific crops because of an increasing incidence of herbicide resistance (Frantzen and Hatcher 1997). Especially, control of common groundsel in carrot is insufficient (D. Baumann, personal communication), and biological control might be an alternative to herbicides. The biotrophic fungus *P. lagenophorae* reduces the competitiveness of its host (Paul et al. 1993 and references therein) and may control common groundsel populations if epidemics develop rapidly enough (Frantzen and Müller-Schärer 1998).

The Swiss horticultural guidelines provide a list of herbicides that may or may not control common groundsel in vegetables (Anonymous 2000). For the present study three herbicides were selected from this list according to the following criteria: (1) they should be applicable in vegetables in which control of common groundsel is known to be difficult, e.g., carrots (D. Baumann, personal communication), (2) they should represent a relatively broad range of potential control of common groundsel, and (3) they should represent various modes of action, i.e., soil vs. foliar herbicides. The three herbicides in the commercial formulations selected were monolinuron, metoxuron, and pendimethalin. Although we will refer to the active ingredients later, we would like to emphasize here that effects of active ingredients and those of their formulations were not separated in the present study. The Swiss horticultural guidelines are the basis for determining complete control of common groundsel for the soil herbicide monolinuron, sufficient control for the foliar herbicide metoxuron, and no control for the soil herbicide pendimethalin (Anonymous 2000).

We sought to develop a relatively rapid and inexpensive procedure to evaluate the integration of the system management approach with *P. lagenophorae* epidemics and chemical weed control with each of the three selected herbicides. A three-step procedure was employed to quantify the effect under controlled conditions of a herbicide on (1) common groundsel, (2) *P. lagenophorae*, and (3) the velocity of epidemic spread using an epidemiological model. The first step is included to select herbicides that, when applied alone, sufficiently control common groundsel, i.e., no biological control is required. The second step is included to select herbicides that do not inhibit the development of *P. lagenophorae* and thus permit quantification of some basic parameters to be used in the epidemiological model of the third step. The third step elucidates the potential effects of the selected herbicides on the velocity of epidemic progress of *P. lagenophorae* using a relatively simple epidemiological model.

Materials and Methods

General

In the present study, the maternal Swiss common groundsel line ELS and the single sorus Swiss *P. lagenophorae* line ELS were used (Wyss 1997). We assumed that the plants used were genetically similar because common groundsel is a strong inbreeder, and plants came from one mother plant. Also, we assumed that spores of the fungus were genetically similar because the fungus reproduced asexually from a single sorus.

Commercial formulations of herbicides were used, i.e., Aresin containing 50% monolinuron, Dosanex containing 80% metoxuron, and Stomp SC containing 36% pendimethalin. The recommended rates of application in the field were 0.75, 1.6, and 0.54 kg ai ha^{-1} for monolinuron, metoxuron, and pendimethalin, respectively.

Impact of Herbicides on Common Groundsel

Seed Germination

Each of the five petri dishes was filled with 1% agar (wt/wt), 1% agar amended with 2.5 mg commercially formulated monolinuron per milliliter of agar, 2.5 mg commercially formulated metoxuron per milliliter of agar, or 3.75 μl commercially formulated pendimethalin per milliliter of agar. Petri dishes filled with agar served as controls. Quantities of the commercially formulated herbicides that were used were adjusted to the recommended application rates in the field, i.e., 2.5 kg ha^{-1} for formulated monolinuron and metoxuron, and 3.75 L ha^{-1} for formulated pendimethalin. One hundred seeds of common groundsel were placed evenly on the agar of each dish. Closed dishes were placed in an incubator with day–night cycles of 16 h of light (ca. 150 μmol m^{-2} s^{-1}) at 23 C and 8 h of darkness at 15 C. The relative humidity was kept constant at about 70%. Petri dishes were assigned randomly to a place in the incubator. Germination of common groundsel was recorded daily for 15 d for each petri dish, and the fraction of germinated seeds was calculated. A seed was considered to be germinated if a radicle was visible. The average fraction of germinated seeds of the five petri dishes per treatment was calculated and used in the subsequent analysis. The experiment was conducted three times.

First, differences between treatments with respect to the fraction of seeds germinated at 15 d after sowing were analyzed using Scheffé's test (Sokal and Rohlf 1981). Subsequently, curves were fitted to the data of one trial and one treatment using a log-logistic model (Frantzen 1994)

$$y = \frac{1}{1 + e^{-b \cdot \ln(t/\tau)}} \quad [1]$$

where y is the fraction of seeds germinated, b a shape parameter, t the time in days after placing the seeds on agar, and τ the midgermination time at which half the number of seeds has germinated, i.e., $y = 0.5$. The rate of germination at the midgermination time was computed (Frantzen 1994)

$$v = b/4^{*}\tau \quad [2]$$

The parameter estimates obtained from the data of the three trials were used in a subsequent test of Scheffé (Sokal and Rohlf 1981) to detect significant differences between herbicide and control treatments with respect to seed germination. Data were checked for homogeneity of variance and normality before analysis.

Plant Fitness

Agar was prepared and amended with either monolinuron or pendimethalin (see Seed Germination). Disks of 1-cm diameter and 0.4-cm thickness were cut from the agar, and each disk was placed at the center of a 270-ml pot filled with nutrient-amended peat.[1] Three seeds of common groundsel were placed on each disk, and 10 replicated pots were used for each herbicide and control. Pots were assigned randomly to a place in a climate room with day–night cycles of 16 h of light (ca. 150 μmol m^{-2} s^{-1}) at 23 C and ca. 60% RH and 8 h of darkness at 15 C and ca. 80% RH. If necessary, emerging plants were randomly thinned to one per pot, and roots on the piece of agar were covered with soil. The survival of plants until the seed set, the number of days between sowing and appearance of the first seed head, the number of seeds in the first seed head, and the shoot dry weight at the time of the first seed head were recorded. The experiment was conducted twice.

A similar procedure was followed to quantify the effect of metoxuron on plant fitness, except that seeds were sown at the center of a pot without using agar. Plants were treated with metoxuron at the time when they had an average of three true leaves. The 10 pots were arranged on a floor area of 0.25 m^2, and 25 ml of a solution of 2.5 mg commercially formulated metoxuron per milliliter was sprayed on the plants in a spray volume of 500 L ha^{-1} using a hand sprayer.[2] Control plants were sprayed with water. Pots were assigned randomly to a place in an incubator before treatment with metoxuron, or water, and assigned randomly to a place in a climate chamber afterward. The incubator and climate chamber had day–night cycles as above. The experiment was conducted twice.

All pots in the climate chamber were treated twice with 5% Teknar[3] to control sciarid flies (*Sciaridae*).

The differences between plants treated with herbicide and the respective controls in days until the first seed head, the

number of seeds in the first seed head, and the dry weight at the time of seed set were tested for significance using one-factor analysis of variance (Sokal and Rohlf 1981). Analysis was done for each trial separately. Data were checked for homogeneity of variance and normality before analysis.

Impact of Herbicides on *P. lagenophorae*

Spore Germination

Agar was prepared and amended with either metoxuron or pendimethalin (see Seed Germination). Microscope slides were covered with agar, and three slides were used for each herbicide and control. Aecidiospores of *P. lagenophorae* were settled onto the slides using a settling tower. Each slide was placed in a petri dish with wetted blotting paper at the bottom to keep relative humidity close to 100%. Petri dishes were placed in an incubator in the dark at 18 C and ca. 80% RH. After 24 h the fraction of spores germinated was determined for each slide. Two hundred spores per slide were examined to determine the fraction of spores germinated. A spore was considered to be germinated if the germination tube was equal to or longer than the diameter of the spore. The average fraction of spores germinated on three replicate slides was used in the analysis. The experiment was conducted three times.

Spore Production

Agar was prepared and amended with commercially formulated pendimethalin (see Seed Germination). Disks of 1-cm diameter, which were about 0.4 cm thick, were cut from the agar, and each disk was placed at the center of a 270-ml pot filled with nutrient-amended peat.[1] Three seeds of common groundsel were placed on each disk, and 10 replicate pots were used for the herbicide and control treatments. The subsequent culturing of plants was similar to the procedure followed in the experiment described in the plant fitness section.

Plants were inoculated with *P. lagenophorae* 21 d after sowing, when plants had an average of three to four true leaves. Five millileters of a suspension of 0.5 mg *P. lagenophorae* spores per milliliter was sprayed onto the plants. After inoculation, plants were covered with a plastic bag for about 16 h to assure a relative humidity close to 100%. The fungus started to sporulate 10 d after inoculation, i.e., the latent period, and spores were collected every third day in Trial 1 and every second day in Trial 2. Spores were collected from groups of five plants, and the number of spores collected per group was determined by adding 5 ml of deionized water and two droplets of the surfactant Tween 20 to a snap cup with the spores, taking five samples from the suspension, and counting the number of spores in each sample using a hemocytometer. The average of the samples was used as a parameter for the number of spores collected per group of five plants, and the average of two groups of either pendimethalin-treated plants or control plants was calculated. These averages were subsequently analyzed.

A gamma distribution was fitted to the data (van den Bosch et al. 1988a)

$$\ln I_t = \alpha + (\delta_1 * \ln t) + (\delta_2 * t) \qquad [3]$$

where I is the spore production determined at time t, and α, δ_1, and δ_2 are the parameters of the gamma distribution estimated by nonlinear regression.

The experiment was repeated once, i.e., the experiment embodied two trials.

Spread of Epidemic

The velocity of epidemic spread was calculated assuming the annular expansion of *P. lagenophorae* epidemics. The model of van den Bosch et al. (1988b, 1988c) was used, which is based on estimates of the net reproductive number R_0, the contact distribution D, and the time kernel $i(t)$. The net reproductive number is the number of offspring produced by one mother individual. Here, offspring is the number of spores produced by one lesion (i.e., one mother spore). The offspring settles down at a place that is distant from the mother, and the probability density function describing the dispersal distances of all offspring is called the contact distribution. Offspring is not produced at the same time, and the mathematical function describing the production of offspring in time is called the time kernel. In its most simple form the time kernel is given by the generation time of an organism.

Here, we explored the effects of the herbicide pendimethalin on the time kernel $i(t)$ and assumed no major, or detectable, effects of herbicide on the net reproductive number and the contact distribution D (see also Discussion). The parameter R_0 was set at 383, and the parameter D was set at an exponential distribution with $\sigma = 28$ cm. These values of the parameters were estimated in a field experiment as reported elsewhere (Frantzen and van den Bosch 2000). The time kernel $i(t)$ is characterized by the latent period p, the average time to produce a spore during the infectious period μ, and the standard deviation ν of μ. The parameters μ and ν can be derived from the parameters of the gamma distribution (van den Bosch et al. 1988a, 1988c),

$$\mu = \frac{\delta_1 + 1}{-\delta_2} \quad \text{and} \qquad [4]$$

$$\nu = \sqrt{\frac{\delta_1 + 1}{(-\delta_2)^2}} \qquad [5]$$

where the parameters are as explained for Equation 3.

Results

Impact of Herbicides on Common Groundsel

The soil herbicide pendimethalin stimulated the germination of common groundsel seeds, i.e., a mean fraction of 0.92 (standard error of mean of 0.02) of seeds germinated with treatment as compared with a mean fraction of 0.71 (standard error of mean of 0.06) of seeds that germinated in the control, and this difference was significant (Scheffé's test, $P < 0.05$). The soil herbicide monolinuron nearly completely inhibited germination, i.e., a mean fraction of less than 0.01 (standard error of mean less than 0.01) of seeds germinated, and this was significantly lower than that in the control (Scheffé's test, $P < 0.05$). The dynamics of germination differed between seeds germinating on agar amended with and without pendimethalin (Table 1; Figure 1). Seeds on agar amended with pendimethalin germinated faster than

TABLE 1. Effects of formulated pendimethalin on seed germination dynamics of *Senecio vulgaris*.

Treatment	Trial	Parameters[a]			
		b	τ(days)	*v*[b]	R^2
Control	1	1.82 (0.12)	8.87 (0.23)	0.05	0.98
	2	1.95 (0.04)	11.61 (0.09)	0.04	0.99
	3	1.27 (0.07)	4.35 (0.15)	0.07	0.98
Pendimethalin	1	2.01 (0.15)	4.33 (0.18)	0.12	0.98
	2	2.37 (0.15)	4.36 (0.14)	0.14	0.98
	3	2.52 (0.11)	2.92 (0.06)	0.22	0.99

[a] Parameter estimates of the log-logistic model $y = 1/1 + e^{-b \ln(t/\tau)}$ where y is the fraction of seeds germinated, b a shape parameter, t the time in days after placing the seeds on agar, and τ is the midgermination time (in days after placing seeds on agar) at which half the number of seeds were germinated, i.e. $y = 0.5$.

[b] The rate of germination (dy/dt) at the midgermination time was computed as: $v = b/4\tau$.

those on agar without pendimethalin. This faster germination is indicated by a combination of a higher, but not significant ($P > 0.05$), value of parameter *b,* a shorter, but not significant ($P > 0.05$), midgermination time *τ,* and a significant ($P < 0.05$) higher velocity *v* at the midgermination time.

No plants emerged on agar amended with monolinuron in the plant fitness test. The number of plants surviving until seed set was relatively low for plants treated with the foliar herbicide metoxuron when compared with control plants (Table 2A). Plants surviving the metoxuron treatment produced the first seed head significantly later than did control plants. No major effect on fitness parameters could be detected for plants emerging on agar amended with pendimethalin (Table 2B).

Monolinuron was excluded from subsequent tests because of the relatively large impact on common groundsel. No common groundsel plants could be expected to emerge and survive in fields treated with monolinuron.

Impact of Herbicides on *P. lagenophorae*

Both metoxuron and pendimethalin completely inhibited the germination of *P. lagenophorae* spores (Table 3), and no *P. lagenophorae* epidemics can be expected to develop if the spores come into contact with these herbicides. Contact between the foliar herbicide metoxuron and the airborne *P. lagenophorae* spores is inevitable in the field, and therefore this herbicide was excluded from further tests. Direct contact between the soil herbicide pendimethalin and the *P. lagenophorae* spores is less likely. The only contact between pendimethalin and *P. lagenophorae* might occur indirectly by way of secondary systemic effects of pendimethalin on aboveground parts of the plants. If so, pendimethalin may interfere with the process of penetration and colonization of stems and leaves of the plants by *P. lagenophorae*. This possibility was tested in the subsequent spore production experiment, in which no significant differences could be detected between plants emerging and growing on water agar amended with pendimethalin and control plants (Table 4; Figure 2).

Spread of Epidemic

The velocity of epidemic spread, i.e., the increase of the radius of a disease focus, was estimated to be about 10 cm per day (Table 5). No effect of pendimethalin on the velocity of epidemic spread was detected.

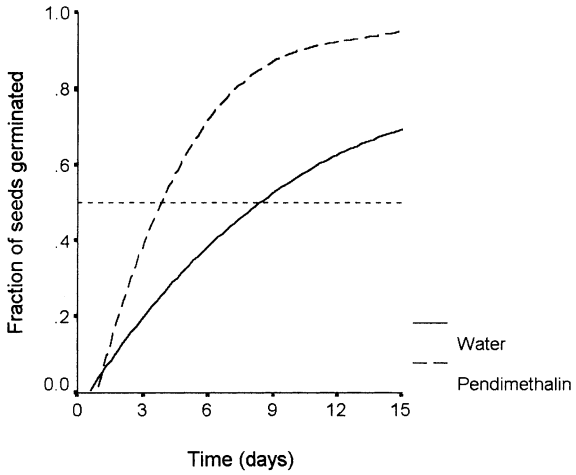

FIGURE 1. Dynamics of *Senecio vulgaris* seed germination on water agar and on water agar amended with pendimethalin. Curves are based on averages of parameters of a log-logistic model (Equation 1) estimated in three trials. The dotted, horizontal line indicates the 0.5 level of the fraction of seeds germinated.

Discussion

The results presented support the Swiss horticultural guidelines (Anonymous 2000) with respect to complete control of common groundsel by monolinuron. Treatment of common groundsel seeds with commercially formulated monolinuron almost completely inhibited germination, and the very few emerging seedlings did not survive. Clearly, control of common groundsel by *P. lagenophorae* epidemics is not required in crops where weed management includes the use of monolinuron at the recommended field rate against other weeds. The inhibition of seed germination was not detected using dilutions of 1:10, 1:20, and 1:40 of formulated monolinuron (Rossi 1999).

The results with formulated metoxuron were ambivalent with respect to control of common groundsel. Neither did metoxuron kill all the treated plants nor was a significant effect of metoxuron on dry weight of the surviving plants detected. The results do not indicate whether the observed effects of metoxuron on common groundsel under controlled conditions correspond with sufficient control in the field. The Swiss horticultural guidelines can, therefore, neither be supported nor rejected with respect to the use of metoxuron against common groundsel (Anonymous 2000). If control by metoxuron is insufficient in the field, control by *P. lagenophorae* epidemics might be complementary to the use of metoxuron against other weeds. This option, however, does not seem to be feasible because germination of *P. lagenophorae* spores is inhibited by metoxuron, as demonstrated in the present study. Because a *P. lagenophorae* epidemic needs some time to spread all over a common groundsel population, and knowing that the time window of weed control, in general, is relatively short, an application of metoxuron subsequent to epidemic spread does not seem to be a vital option. Katan and Eshel (1972) emphasize that effects of herbicides on fungi may be concentration dependent. To check this possibility, the effects of metoxuron at

TABLE 2. Effects of the herbicides metoxuron (A) and pendimethalin (B) on plant fitness parameters of *Senecio vulgaris*, the number of plants surviving until seed set, the average time of appearance of the first seed head in days after sowing, the average dry weight of plants in grams at time of first seed head, and the average number of seeds in the first seed head. Significance level of differences between herbicide treatment and the appropriate control is indicated, where *** indicates $P < 0.001$, ** indicates $P < 0.01$, and ns indicates $P > 0.05$.

			Parameters		
Trial	Treatment	Number of surviving plants	Time of seed set (days)	Dry weight (g)	Number of seeds (per flower head)
A					
1	Control	10	45.0 (0.9)	0.99 (0.04)	32.0 (2.3)
	Metoxuron	6	54.0 (1.8)***	0.83 (0.08)^{ns}	16.8 (4.0)**
2	Control	10	46.1 (0.8)	1.32 (0.09)	28.1 (3.1)
	Metoxuron	2	55.5 (0.5)***	1.07 (0.14)^{ns}	18.0 (14.0)^{ns}
B					
1	Control	10	41.9 (0.8)	1.21 (0.07)	23.9 (3.3)
	Pendimethalin	10	46.1 (1.2)**	1.07 (0.08)^{ns}	20.5 (2.9)^{ns}
2	Control	10	43.7 (0.5)	1.52 (0.11)	20.8 (2.7)
	Pendimethalin	10	43.2 (0.7)^{ns}	1.43 (0.08)^{ns}	29.7 (3.6)^{ns}

dilutions of 1:10, 1:20, and 1:40 of the recommended rate on *P. lagenophorae* spore germination were quantified (Rossi 1999). No germination of spores was observed at dilutions of 1:10 and 1:20, and the fraction of spores that germinated at a dilution of 1:40 was lower than that of the control, i.e., 0.29 vs. 0.54 of the control. We may conclude that *P. lagenophorae* epidemics, in general, are inhibited even if application of metoxuron in the field is at a lower concentration than the recommended rate. The results also suggest that an application of metoxuron before inducing a *P. lagenophorae* epidemic is not appropriate because residuals may be present on plants for several days, delaying the start of a *P. lagenophorae* epidemic to a very great extent.

The results presented were clear with respect to control of common groundsel by pendimethalin. In agreement with the Swiss horticultural guidelines (Anonymous 2000), no detrimental effect of pendimethalin on the plant could be detected, except for a delay in the time of seed set in one trial of the fitness experiment. We may conclude that control of common groundsel by *P. lagenophorae* epidemics may be required when pendimethalin is used to control other weeds.

Germination of *P. lagenophorae* spores was inhibited by pendimethalin, and this suggested incompatibility between *P. lagenophorae* epidemics and pendimethalin. But we do not expect contact between the spores and pendimethalin in the field because pendimethalin, in general, is applied as a soil herbicide before emergence of common groundsel, whereas *P. lagenophorae* is an airborne fungus that infects aboveground organs of common groundsel. Translocation of dinitroanilines like pendimethalin from the belowground parts of the plant to the aerial parts can be excluded (Zimdahl 1993). Secondary systemic effects of pendimethalin on the plant might mediate the only possible interaction between pendimethalin and the rust fungus (cf. Katan and Eshel 1972). The likelihood of such an interaction was tested in the experiment described in the spore production section presented here. We could not detect any effect of pendimethalin on the infection process and subsequent spore production of the fungus.

TABLE 3. Effects of the herbicides metoxuron and pendimethalin on germination of *Puccinia lagenophorae* aecidiospores. Entries are averages (SEM in parentheses) of the fraction of spores germinated on three replicate slides. Data is of three replicate trials.

Trial	Treatment	Fraction of spores germinated
1	Control	0.33 (0.16)
	Metoxuron	0.00 (0.00)
	Pendimethalin	0.00 (0.00)
2	Control	0.62 (0.01)
	Metoxuron	0.00 (0.00)
	Pendimethalin	0.00 (0.00)
3	Control	0.68 (0.02)
	Metoxuron	0.00 (0.00)
	Pendimethalin	0.00 (0.00)

The effects of pendimethalin on *P. lagenophorae* epidemics were analyzed using an epidemiological model. This model incorporates three essential characteristics of the spread of any organism: the net reproductive number R_0, the time kernel $i(t)$, and the contact distribution D (Shigesada and Kawasaki 1997). The effects of pendimethalin on R_0 were not quantified in the present study. A field experiment would be required to determine these effects, if any, and such an effort did not seem to be appropriate because (1) we could not detect the effects of pendimethalin on disease severity in the present experiment (data not presented), and therefore major effects on R_0 can be excluded; and (2) the velocity of epidemic spread is logarithmically related to R_0 (Zadoks and van den Bosch 1994), and the minor effects of pendimethalin on R_0 will have only little effect on the velocity. In contrast, the contact distribution D is linearly related to the velocity (Zadoks and van den Bosch 1994), and small effects of pendimethalin on D may have relatively large effects on the velocity. But quantification of the contact distribution, in general, is troublesome (Frantzen and van den Bosch 2000) and does preclude detection of relatively subtle effects of pendimethalin, e.g., by altering the morphology of common groundsel on D. Thus, determining the effects of pendimethalin on the time kernel $i(t)$, as was done in the present study, is the most appropriate method to predict the effects on epidemic spread. If so, the results for the epidemiological model indicate that the use of pen-

TABLE 4. Effects of pendimethalin on spore production of *Puccinia lagenophorae*.

Trial	Treatment	Parameters[a]			R^2
		α	δ_1	δ_2	
1	Control	0.84 (0.72)	3.55 (0.52)	−0.46 (0.04)	0.98
	Pendimethalin	0.40 (0.64)	3.79 (0.47)	−0.48 (0.04)	0.99
2	Control	3.64 (0.33)	2.54 (0.24)	−0.35 (0.02)	0.99
	Pendimethalin	2.53 (0.63)	3.41 (0.46)	−0.46 (0.04)	0.97

[a] Parameter estimates (SE in parentheses) of gamma density fitted as $\ln I_t = \alpha + (\delta_1 \ln t) + (\delta_2 t)$, where I is the spore production determined at time t, and α, δ_1, and δ_2 are parameters of the gamma distribution estimated by nonlinear regression.

dimethalin does not affect epidemic spread of *P. lagenophorae*, and a combined use of pendimethalin and *P. lagenophorae* epidemics seems feasible. Such a complementary use is called horizontal integration, i.e., broadening the spectrum of weeds controlled by combining control methods (Müller-Schärer et al. 2000).

The three-step procedure used in the present study seems to be suitable for evaluating the integration of biological weed control following the system management approach and chemical control. Clearly, the procedure has to be adapted for each herbicide and each biocontrol agent. Here, we may conclude that *P. lagenophorae* epidemics may be used to control common groundsel complementary to the use of pendimethalin.

Sources of Materials

[1] Floragard TKS2, Floragard, Gerhard-Stalling-Str. 7, 26135 Oldenburg, Germany.

[2] Aero-Spray 2000®, Birchmeier & Cie, CH-5444 Künten, Switzerland.

[3] Novartis Agro AG, CH-4332 Stein, Switzerland.

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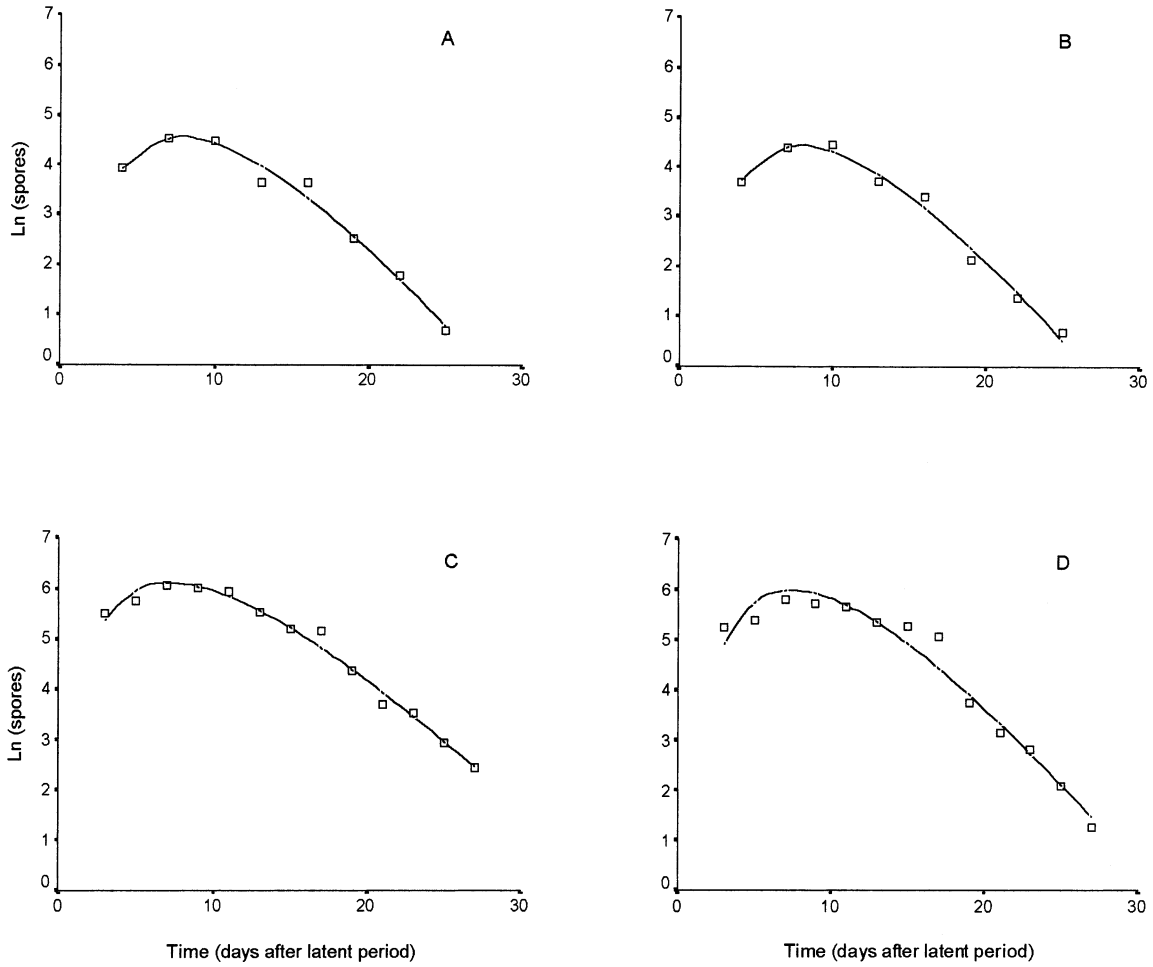

FIGURE 2. Effects of pendimethalin on *P. lagenophorae* spore production. Gamma distribution (Equation 3) fitted to data of *P. lagenophorae* aecidiospore production from Trials 1 (A, B) and 2 (C, D), and for *Senecio vulgaris* control plants (A, C) and plants treated with pendimethalin (B, D). Entries are averages of spores collected from two groups of five plants each.

TABLE 5. Effects of the herbicide pendimethalin on estimated velocity of epidemic spread of *Puccinia lagenophorae* on *Senecio vulgaris* based on various parameters.

		Parameters					
Trial	Treatment	R_0[a]	σ (cm)[b]	p (days)[c]	μ (days)[d]	ν (days)[e]	c (cm days^{-1})[f]
1	Control	383	28	10	9.9	4.6	9.8
	Pendimethalin	383	28	10	10.0	4.6	9.8
2	Control	383	28	10	10.1	5.4	10.2
	Pendimethalin	383	28	10	9.6	4.6	10.0

[a] The net reproductive number R_0 was determined by Frantzen and van den Bosch (2000) and is assumed not to be influenced by pendimethalin (see Discussion).

[b] The contact distribution D, as expressed by σ of an exponential distribution, was determined by Frantzen and van den Bosch (2000) and is assumed not to be influenced by pendimethalin (see Discussion).

[c] The latent period for all treatments and trials of the present study was 10 days.

[d] The average time to produce a spore during the infectious period is $\mu = (\delta_1 + 1)/(-\delta_2)$ (Equation 4) and estimates of δ_1 and δ_2 are presented in Table 4.

[e] The standard deviation of μ is $\nu = \sqrt{(\delta_1 + 1)/(\delta_2)^2}$ (Equation 5) and estimates of δ_1 and δ_2 are presented in Table 4.

[f] Velocity c was calculated using the model of van den Bosch *et al.* (1988b, 1988c).

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